

National Athletic Trainers' Association Position Statement: Environmental Cold Injuries

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Objective: To present recommendations for the prevention, recognition, and treatment of environmental cold injuries.

Background: Individuals engaged in sport-related or work-related physical activity in cold, wet, or windy conditions are at risk for environmental cold injuries. An understanding of the physiology and pathophysiology, risk management, recognition, and immediate care of environmental cold injuries is an essential skill for certified athletic trainers and other health care providers working with individuals at risk.

Recommendations: These recommendations are intended to provide certified athletic trainers and others participating in athletic health care with the specific knowledge and problem-solving skills needed to address environmental cold injuries. Each recommendation has been graded (A, B, or C) according to the Strength of Recommendation Taxonomy criterion scale.

Key Words: environmental physiology, hypothermia, frost-bite, frostnip, chilblain, pernio, immersion foot, trench foot

Cold injuries are a common result of exposure to cold environments during physical activity or occupational pursuits. Many individuals engage in fitness pursuits and physical activity year-round in environments with cold, wet, or windy conditions (or a combination of these), thereby placing themselves at risk of cold injuries. The occurrence of these injuries depends on the combination of 2 factors: low air or water temperatures (or both) and the influence of wind on the body's ability to maintain a normothermic core temperature, due to localized exposure of the extremities to cold air or surfaces. Cold injuries and illnesses occur in a wide range of physically active individuals, including military personnel, traditional winter-sport athletes, and outdoor-sport athletes, such as those involved in running, cycling, mountaineering, and swimming. Traditional team sports such as football, baseball, softball, soccer, lacrosse, and track and field have seasons that extend into late fall or early winter or begin in early spring, when weather conditions can increase susceptibility to cold injury. Reported rates of hypothermia and frostbite include 3% to 5% of all injuries in mountaineers and 20% of all injuries in Nordic skiers.¹ Cold injury frequency in military personnel is reported to range from 0.2 to 366 per 1000 exposures.¹⁻⁶

As the scope of physical activity participation broadens (eg, extreme sports, adventure racing) and environments with the potential for extreme weather conditions become more accessible, a review of cold injury physiology, prevention, recognition, treatment, and management is warranted. Clinicians practicing in settings or geographic regions that predispose individuals to cold injury must be aware of these risks and implement strategies to prevent cold injuries and to minimize them when they occur.

PURPOSES

This position statement includes a review of available literature, definitions of cold injuries, and a set of recommendations that will allow certified athletic trainers (ATs) and other allied health and medical providers to

1. Identify and employ prevention strategies to reduce cold-related injuries and illnesses in the physically active.
2. Describe factors associated with cold-related injuries and illnesses.
3. Provide on-site first aid and immediate care of cold-related injuries and illnesses.
4. Understand the thermoregulatory and physiologic responses to cold.
5. Identify groups with unique concerns related to cold exposure.

DEFINITIONS OF COLD INJURIES

Cold injuries are classified into 3 categories: decreased core temperature (hypothermia), freezing injuries of the extremities, and nonfreezing injuries of the extremities. Each scenario and its characteristic condition(s) will be described. A summary of the signs and symptoms of these injuries and illnesses is found in Table 1, with images of the skin conditions displayed in Figures 1 through 3.

Hypothermia

Traditionally, hypothermia is defined as a decrease in core body temperature below 95°F (35°C). Hypothermia is

Table 1. Signs and Symptoms of Cold-Related Injuries

Condition	Sign or Symptom ^a
Hypothermia ^{1,5,7-9} Mild	Core temperature 98.6°F to 95°F (37°C–35°C) Amnesia, lethargy Vigorous shivering Impaired fine motor control Cold extremities Polyuria Pallor Rhinorrhea Typically conscious Blood pressure within normal limits
Moderate	Core temperature 94°F to 90°F (34°C–32°C) Depressed respiration and pulse Cardiac arrhythmias Cyanosis Cessation of shivering Impaired mental function Slurred speech Impaired gross motor control Loss of consciousness Muscle rigidity Dilated pupils Blood pressure decreased or difficult to measure
Severe	Core temperature below 90°F (32°C) Rigidity Bradycardia Severely depressed respiration Hypotension, pulmonary edema Spontaneous ventricular fibrillation or cardiac arrest Usually comatose
Frostbite ^{1,5,8-13} Mild/superficial	Dry, waxy skin Erythema Edema Transient tingling or burning sensation Skin contains white or blue-gray colored patches Affected area feels cold and firm to the touch Limited movement of affected area
Deep	Skin is hard and cold Skin may be waxy and immobile Skin color is white, gray, black, or purple Vesicles present Burning aching, throbbing, or shooting pain Poor circulation in affected area Progressive tissue necrosis Neurapraxia Hemorrhagic blistering develops within 36 to 72 hours Muscle, peripheral nerve, and joint damage likely
Chilblain/pernio ^{5,13,14}	Red or cyanotic lesions Swelling Increased temperature Tenderness Itching, numbness, burning, or tingling Skin necrosis Skin sloughing
Immersion (trench) foot ^{5,8,14}	Burning, tingling, or itching Loss of sensation Cyanotic or blotchy skin Swelling Pain/sensitivity Blisters Skin fissures or maceration

^a Not all patients will display all signs and symptoms of the condition.



Figure 1. Frostbite.

classified as mild, moderate, or severe, depending upon measured core temperature. Information in the literature varies slightly as to which core temperatures are assigned to which degree of hypothermia, but in this paper, we will use the following definitions. Mild hypothermia is a core temperature of 95°F (35°C) to 98.6°F (37°C). Moderate hypothermia is a core temperature of 90°F (32°C) to 94°F (34°C). Severe hypothermia is a core temperature below 90°F (32°C).^{1,7-9} Each level of hypothermia has characteristic signs and symptoms, although individuals respond differently, and not every hypothermic person exhibits all signs and symptoms. Therefore, a detailed assessment is appropriate in all cases of potential cold injury. Hypothermia is most likely to occur with prolonged exposure to cold, wet, or windy conditions (or a combination of these) experienced during endurance events, outdoor team sports (eg, soccer, football), mountaineering, hiking, and military maneuvers and in occupations that require long periods outdoors or in unheated spaces (eg, public safety, building trades, transportation).



Figure 2. Chilblain. Photograph reprinted with permission: ©Christoph U. Lehmann, MD, Dermatlas; <http://www.dermatlas.org>.



Figure 3. Immersion (trench) foot.

Frostbite and Frostnip

Frostbite is actual freezing of body tissues. It is a localized response to a cold, dry environment, yet moisture from sweating may exacerbate frostbite due to increased tissue cooling. Similar to hypothermia, frostbite has stages, delineated by the depth of tissue freezing and resulting in frostnip, mild frostbite, or severe frostbite.^{1,8-13} Frostbite develops as a function of the body's protective mechanisms to maintain core temperature. Warm blood is shunted from cold peripheral tissues to the core by vasoconstriction of arterioles, which supply capillary beds and venules to the extremities and face, especially the nose and ears. Frostbite progresses from distal to proximal and from superficial to deep. As the temperature of these areas continues to decrease, cells begin to freeze. Damage to the frostbitten tissue is due to electrolyte concentration changes within the cells, resulting in water crystallization within the tissue. For cells to freeze, the tissue temperature must be below 28°F (-2°C).⁸⁻¹³

Frostnip, the mildest form of cold injury to the skin, is a precursor to frostbite. It can occur with exposure of the skin to very cold temperatures, often in combination with windy conditions. It can also occur from skin contact with cold surfaces (eg, metal, equipment, liquid). With frostnip, only the superficial skin is frozen; the tissues are not permanently damaged, although they may be more sensitive to cold and more likely, with repeated exposures, to develop frostnip or frostbite.⁸⁻¹³ Mild frostbite involves freezing of the skin and adjacent subcutaneous tissues; extracellular water freezes first, followed by cell freezing. Severe frostbite is freezing of the tissues below the skin and the adjacent tissues, which can include muscle, tendon, and bone.⁸⁻¹³

Chilblain (Pernio)

Chilblain, also known as pernio, is an injury associated with extended exposure (1-5 hours) to cold, wet conditions. Chilblain is an exaggerated or uncharacteristic inflammatory response to cold exposure. Prolonged constriction of the skin blood vessels results in hypoxemia and vessel wall inflammation; edema in the dermis may also be present. Chilblain can occur with or without

Table 2. Strength of Recommendation Taxonomy (SORT)^a

Strength of Recommendation	Definition
A	Recommendation based upon consistent and good quality patient-oriented evidence (morbidity, mortality, symptom improvement, cost reduction, and quality of life).
B	Recommendation based on inconsistent or limited-quality patient-oriented evidence.
C	Recommendation based on consensus, usual practice, opinion, disease-oriented evidence (measures of intermediate, physiologic, or surrogate end-points that may or may not reflect improvements in patient outcomes), or case series for studies of diagnosis, treatment, prevention, or screening.

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freezing of the tissue. The hands and feet are most commonly affected, but chilblain of the thighs has also been reported.¹⁴ Situations in which this can happen include alpine sports, mountaineering, hiking, endurance sports, and team sports in which footwear and clothing remain wet for prolonged periods due to water exposure or sweating.

Chilblain severity is time and temperature related. The higher the temperature of the water (generally ranging from 32°F [0°C] to 60°F [16°C]), the longer the duration of exposure required to develop chilblain. Time of exposure is usually measured in hours or even days, rather than the minutes or hours associated with frostbite. Chilblain and immersion foot (see below) occur in similar environments, but the former is a more superficial injury and can develop in a shorter time period than the latter.^{13,14}

Immersion (Trench) Foot. Immersion foot typically occurs with prolonged exposure (12 hours to 4 days) to cold, wet conditions, usually in temperatures ranging from 32°F to 65°F (0°C–18°C). This condition affects primarily the soft tissues, including nerves and blood vessels, due to an inflammatory response that results in high levels of extracellular fluid. The most common mechanism for developing immersion foot is the continued wearing of wet socks or footwear (or both).^{8,14}

EVIDENCE CLASSIFICATION

In this position statement, we present recommendations using an evidence-based review and the Strength of Recommendation Taxonomy (SORT) criterion scale (Table 2) proposed by the American Academy of Family Physicians.¹⁵ The recommendations are given a grade of A, B, or C based upon evidence using patient or disease-oriented outcomes (treatments or practices). Little outcomes-based research using randomized clinical trials on cold injury has been performed due to ethical constraints regarding standards of care and difficulties procuring large sample groups. These limitations should be weighed when assessing specific recommendations.

RECOMMENDATIONS

Recommendations are presented to help ATs and other health care providers minimize risks to the health and safety of physically active individuals exposed to cold environments and provide effective immediate care when needed. Individual responses to cold vary physiologically with combinations of cold, wet, and windy conditions as well as clothing insulation, exposure time, and other nonenvironmental factors. Therefore, these recommendations do not guarantee

complete elimination of cold-related injuries but may decrease risk. The National Athletic Trainers' Association (NATA) promotes the following approaches for prevention, recognition, and treatment of cold-related injuries.

Prevention

1. Perform a comprehensive, physician-supervised, pre-participation medical screening to identify athletes with a previous history of cold injury and athletes predisposed to cold injury based upon known risk factors (Table 3). This preparticipation examination should include questions pertaining to a history of cold injury and problems with cold exposure¹⁶ and should be performed before planned exposures to conditions that may lead to cold injury. *Evidence Category: C*
2. Identify participants who present with known risk factors (Table 3) for cold injury and provide increased monitoring of these individuals for signs and symptoms.⁵ *Evidence Category: C*
3. Ensure that appropriately trained personnel are available on-site at the event and are familiar with cold injury prevention, recognition, and treatment approaches.⁵ *Evidence Category: C*
4. Educate athletes and coaches concerning the prevention, recognition, and treatment of cold injury and the risks associated with activity in cold environments.⁵ *Evidence Category: C*
5. Educate and encourage athletes to maintain proper hydration and eat a well-balanced diet. These guidelines are especially imperative for activities exceeding 2 hours.^{17–19} Consistent fluid intake during low-intensity exercise is necessary to maintain hydration in the presence of typical cold-induced diuresis.^{20–22} Athletes should be encouraged to hydrate even if they are not thirsty, as evidence suggests the normal thirst mechanism is blunted with cold exposure.²³ *Evidence Category: C*
6. Develop event and practice guidelines that include recommendations for managing athletes participating in cold, windy, and wet conditions.^{24,25} The influence of air temperature and wind speed conditions should be taken into account by using wind-chill guidelines.^{26,27} Risk management guidelines (Table 3, Figure 4) can be used to make participation decisions based upon the prevailing conditions. Participation

decisions depend upon the length of anticipated exposure and availability of facilities and interventions for rewarming if needed. Modify activity in high-risk conditions to prevent cold injury. Monitor athletes for signs and symptoms and be prepared to intervene with basic treatment. Also monitor environmental conditions before and during the activity and adjust activities if weather conditions change or degenerate.^{28,29} *Evidence Category: C*

7. Clothing should provide an internal layer that allows evaporation of sweat with minimal absorption, a middle layer that provides insulation, and a removable external layer that is wind and water resistant and allows for evaporation of moisture. Examples of various clothing ensembles are found in Table 4. Toes, fingers, ears, and skin should be protected when wind-chill temperatures are in the range at which frostbite is possible in 30 minutes or less. Remove wet clothing as soon as practical and replace with dry, clean items.^{30–32} *Evidence Category: C*
8. Provide the opportunity for athletes to rewarm, as needed, during and after activity using external heaters, a warm indoor environment, or the addition of clothing. After water immersion, rewarming should begin quickly and the athlete should be monitored for afterdrop, in which the core temperature actually decreases during rewarming.^{33–35} *Evidence Category: C*
9. Include the following supplies on the field, in the locker room, or at convenient aid stations for rewarming purposes:
 - A supply of water or sports drinks for rehydration purposes as well as warm fluids for possible rewarming purposes. Fluids that may freeze during events in subfreezing temperatures may need to be placed in insulated containers or replaced intermittently.
 - Heat packs, blankets, additional clothing, and external heaters, if feasible, for active rewarming.
 - Flexible rectal thermometer probe to assess core body temperature. Rectal temperature has been identified as the best combination of practicality and accuracy for assessing core temperature in the field.³⁶ Other measurements (tympanic, aural, and esophageal) are problematic or difficult to obtain. The rectal thermometer used should be a low-reading thermometer (ie, capable of measuring temperatures below 95°F [35°C]).
 - Telephone or 2-way radio to communicate with additional medical personnel and to summon emergency medical transportation.
 - Tub, wading pool, or whirlpool for immersion warming treatments (including a thermometer and additional warm water to maintain required temperatures). *Evidence Category: C*
10. Notify area hospital and emergency personnel before large events to inform them of the potential for cold-related injuries. *Evidence Category: C*

Recognition and Treatment

Hypothermia (Mild)

11. Be aware of the signs and symptoms of hypothermia, which include vigorous shivering, increased blood pressure, rectal temperature less than 98.6°F (37°C) but greater than 95°F (35°C), fine motor skill impairment, lethargy, apathy, and mild amnesia (Table 1). *Evidence Category: A*
12. Rectal temperature obtained using a thermometer (digital or mercury) that can read below 94°F (34°C) is the preferred method for assessing core temperature in persons suspected of being hypothermic, even though procuring rectal temperature in the field can be a challenge. Using tympanic, axillary, or oral temperatures instead of rectal temperature is faulty due to environmental concerns, such as exposure to air temperatures; however, if either axillary or oral temperature is above 95°F (35°C), the person is not hypothermic.^{1,7–9} Figure 5 provides a treatment algorithm for hypothermia. *Evidence Category: B*
13. Begin by removing wet or damp clothing; insulating the athlete with warm, dry clothing or blankets (including covering the head); and moving the athlete to a warm environment with shelter from the wind and rain. *Evidence Category: C*
14. When rewarming, apply heat only to the trunk and other areas of heat transfer, including the axilla, chest wall, and groin.^{37–39} Rewarming the extremities can produce afterdrop, which is caused by dilation of peripheral vessels in the arms and legs when warmed. This dilation sends cold blood, often with a high level of acidity and metabolic byproducts, from the periphery to the core. This blood cools the core, leading to a drop in core temperature, and may result in cardiac arrhythmias and death.^{40,41} *Evidence Category: C*
15. Provide warm, nonalcoholic fluids and foods containing 6% to 8% carbohydrates to help sustain shivering and maintain metabolic heat production. *Evidence Category: C*
16. Avoid applying friction massage to tissues, as this may increase damage if frostbite is present.¹⁰ *Evidence Category: A*

Hypothermia (Moderate/Severe)

17. Be aware of the signs and symptoms of moderate and severe hypothermia, which may include cessation of shivering, very cold skin upon palpation, depressed vital signs, rectal temperature between 90°F (32°C) and 95°F (35°C) for moderate hypothermia or below 90°F (32°C) for severe hypothermia, impaired mental function, slurred speech, unconsciousness, and gross motor skill impairment (Table 1).^{1,7–9} *Evidence Category: A*
18. If an athlete with suspected hypothermia presents with signs of cardiac arrhythmia, he or she should be

Table 3. Prevention and Risk Management Process for the Certified Athletic Trainer

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1. Before event
 - Encourage proper hydration and nutrition, and discourage alcohol and drug use.
 - Ensure that athletes and coaches know the signs and symptoms of cold injury.
 - Identify participants at a high risk of cold injury. Risk factors include the following:
 - Lean body composition
 - Females
 - Older age
 - Black race
 - Lower fitness level
 - Presence of comorbidity (eg, cardiac disease, anorexia, Raynaud syndrome, exercise-induced bronchospasm)
 - Encourage proper conditioning and appropriate equipment and clothing choices.
 2. Environmental assessment
 - Evaluate immediate and projected weather information, including air temperature, wind, chance of precipitation or water immersion, and altitude.
 - Identify activity intensity requirements and clothing requirements for each individual.
 - Have alternate plans in place for deteriorating conditions and activities that must be adjusted or cancelled.
 - The following guidelines can be used in planning activity depending on the wind-chill temperature. Conditions should be constantly reevaluated for change in risk, including the presence of precipitation:
 - 30°F (−1.11°C) and below: Be aware of the potential for cold injury and notify appropriate personnel of the potential.
 - 25°F (−3.89°C) and below: Provide additional protective clothing, cover as much exposed skin as practical, and provide opportunities and facilities for rewarming.
 - 15°F (−9.44°C) and below: Consider modifying activity to limit exposure or to allow more frequent chances to rewarm.
 - 0°F (−17.78°C) and below: Consider terminating or rescheduling activity.
 3. Coaches' and athletes' roles
 - Coordinate a schedule of hydration and/or feeding.
 - Coordinate a schedule of rewarming or clothing changes as needed.
 - Identify possible activity modifications as conditions change (eg, change activity times, allow more frequent chances to rewarm, allow changes to clothing or equipment).
 - Become educated about the prevention and recognition of cold injuries.
 - Develop a schedule for monitoring athletes to allow early recognition of potential injury.
 4. Event management
 - Provide food and fluids.
 - Provide warming facilities.
 - Provide additional clothing and equipment for varying conditions.
 - Implement exposure control and rewarming schedules as needed.
 - Monitor environmental conditions and athletes regularly.
 5. Treatment preparations
 - Ensure medical staff is prepared to identify the signs and symptoms of cold injury.
 - Ensure medical staff has proper equipment and skills to assess cold injury, including assessment of low core temperatures.
 - Prepare an emergency action plan in the event that rapid transport is necessary.
 - Prepare active rewarming equipment (eg, whirlpool, hot packs, towels, blankets, dry clothing).
 - Identify warm, dry areas for athletes to passively rewarm, recover, or receive treatment.
 - Provide direct on-site (ie, sideline) means of passive rewarming (eg, additional clothing, space heaters).
-

moved very gently to avoid causing paroxysmal ventricular fibrillation.^{7–9} *Evidence Category: B*

19. Begin with a primary survey to determine the necessity of cardiopulmonary resuscitation (CPR) and activation of the emergency medical system. Remove wet or damp clothing; insulate the athlete with warm, dry clothing or blankets (including covering the head); and move the athlete to a warm environment with shelter from the wind and rain. *Evidence Category: C*
 20. When rewarming, apply heat only to the trunk and other areas of heat transfer, including the axilla, chest wall, and groin.^{37–39} *Evidence Category: C*
 21. If a physician is not present during the treatment phase, initiate rewarming strategies immediately and continue rewarming during transport and at the hospital. *Evidence Category: C*
 22. During the treatment and/or transport, continually monitor vital signs and be prepared for airway management. A physician may order more aggressive rewarming procedures, including inhalation rewarming, heated intravenous fluids, peritoneal lavage, blood rewarming, and use of antiarrhythmia drugs.^{41–46} *Evidence Category: C*
 23. When immediate management is complete, monitor for postrewarming complications, including infection and renal failure.⁴⁷ *Evidence Category: A*
- Frostbite (Superficial)**
24. Be aware of signs and symptoms of superficial frostbite, which include edema, redness or mottled gray skin appearance, stiffness, and transient tingling or burning (Table 1, Figure 1). *Evidence Category: A*
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25. Rule out the presence of hypothermia by evaluating observable signs and symptoms and measuring core temperature. *Evidence Category: C*
26. The decision to rewarm an athlete is contingent upon resources available and likelihood of refreezing. Rewarming can occur at room temperature or by placing the affected tissue against another person's warm skin. Rewarming should be performed slowly, and water temperatures greater than 98°F to 104°F (37°C–40°C) should be avoided. *Evidence Category: C*
27. If rewarming is not undertaken, protect the affected area from additional damage and further tissue temperature decreases and consult with a physician or transport to a medical facility.^{48–50} *Evidence Category: C*
28. Avoid applying friction massage to tissues and leave any vesicles (fluid-filled blisters) intact.^{48–50} *Evidence Category: C*
29. Once rewarming has begun, it is imperative that affected tissue not be allowed to refreeze, as tissue necrosis usually results.^{48–50} *Evidence Category: C*
30. Athletes should avoid consuming alcohol and using nicotine.⁵ *Evidence Category: B*

Frostbite (Deep)

31. Be aware of signs and symptoms of deep frostbite, which include edema, mottled or gray skin appearance, tissue that feels hard and does not rebound, vesicles, and numbness or anesthesia (Table 1).^{10–12} *Evidence Category: A*
32. Rule out the presence of hypothermia by assessing observable signs and symptoms and measuring core temperature.^{10–12,48–50} *Evidence Category: C*
33. To rewarm, the affected tissue should be immersed in a warm (98°F–104°F [37°C–40°C]) water bath. Water temperature should be monitored and maintained. Remove any constrictive clothing and submerge the entire affected area. The water will need to be gently circulated, and the area should be immersed for 15 to 30 minutes. Thawing is complete when the tissue is pliable and color and sensation have returned. Rewarming can result in significant pain, so a physician may prescribe appropriate analgesic medication. *Evidence Category: C*
34. If rewarming is not undertaken, the affected area should be protected from additional damage and further tissue temperature decreases. Consult with a physician or transport the athlete to a medical facility.^{48–50} *Evidence Category: C*
35. Tissue plasminogen activators (tPA) may be administered to improve tissue perfusion. These agents have been shown to limit the need for subsequent amputation due to tissue death.⁵¹ *Evidence Category: B*
36. Do not use dry heat or steam to rewarm affected tissue.^{48–50} *Evidence Category: C*

37. Avoid friction massage or vigorous rubbing to the affected tissues and leave any vesicles or fluid-filled blisters intact. If vesicles rupture, they should be treated to prevent infection.^{48–50} *Evidence Category: C*
38. Once rewarming has begun, it is imperative that the affected tissue not be allowed to refreeze, as tissue necrosis usually results. Also, weight bearing should be avoided when feet are affected. If the possibility of refreezing exists, rewarming should be delayed until advanced medical care can be obtained.^{48–50} *Evidence Category: C*
39. Athletes should avoid using alcohol and nicotine.^{48–50} *Evidence Category: B*
40. If tissue necrosis occurs and tissue sloughs off, debridement and infection control measures are appropriate.^{48–50} *Evidence Category: B*

Chilblain

41. Be aware of the signs and symptoms of chilblain, which include exposure to cold, wet conditions for more than 60 minutes at temperatures less than 50°F (16°C) and the presence of small erythematous papules, with edema, tenderness, itching, and pain (Table 1, Figure 2). Upon rewarming, the skin may exhibit inflammation, redness, swelling, itching, or burning and increased temperature.¹⁴ *Evidence Category: A*
42. Remove wet or constrictive clothing, wash and dry the area gently, elevate the area, and cover with warm, loose, dry clothing or blankets.¹⁴ *Evidence Category: C*
43. Do not disturb blisters, apply friction massage, apply creams or lotions, use high levels of heat, or allow weight bearing on the affected area.¹⁴ *Evidence Category: C*
44. During treatment, continually monitor the affected area for return of circulation and sensation.¹⁴ *Evidence Category: C*

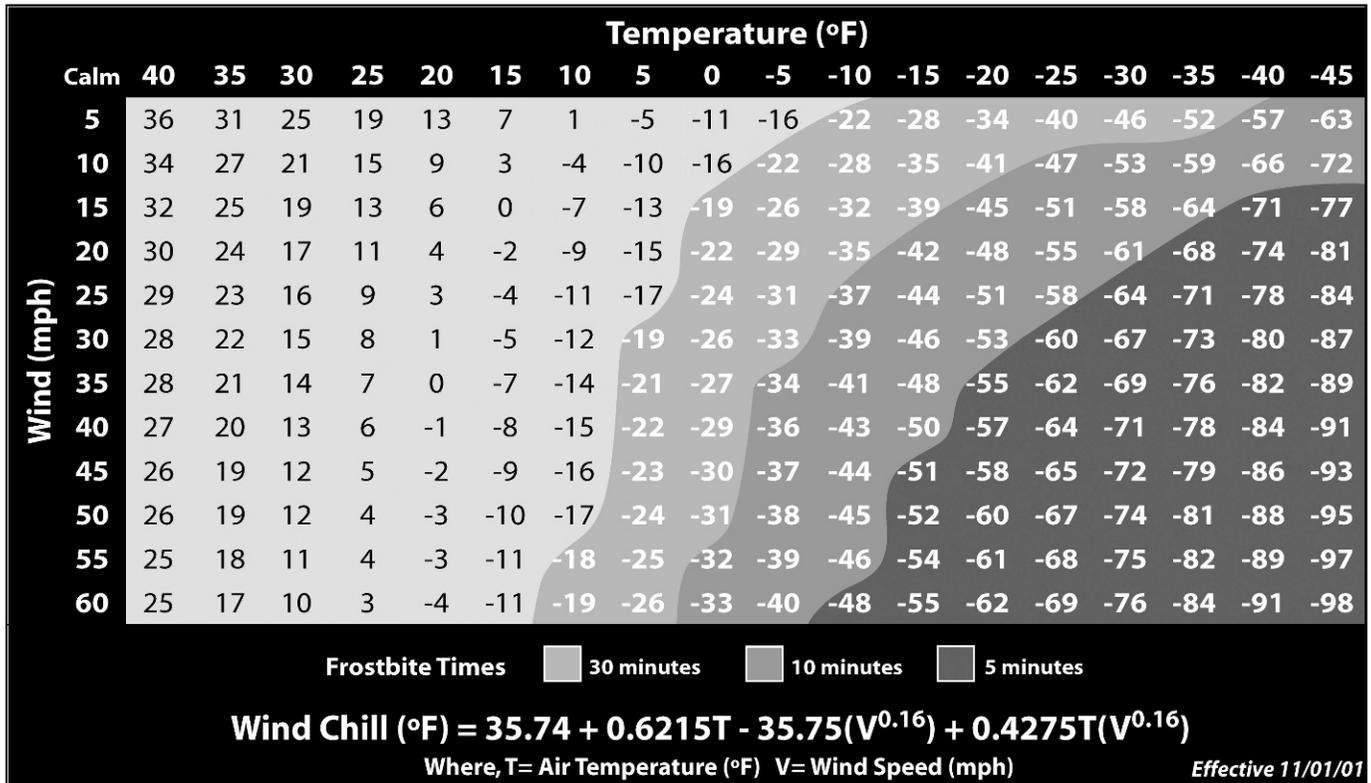
Immersion (Trench) Foot

45. Be aware of the signs and symptoms of immersion (trench) foot, which include exposure to cold, wet environments for 12 hours to 3 or 4 days, burning, tingling or itching, loss of sensation, cyanotic or blotchy skin, swelling, pain or sensitivity, blisters, and skin fissures or maceration (Table 1, Figure 3).^{8,14} *Evidence Category: A*
46. To prevent immersion foot, encourage athletes to maintain a dry environment within the footwear, which includes frequent changes of socks or footwear (or both), the use of moisture-wicking sock material, controlling excessive foot perspiration, and allowing the feet to dry if wearing footwear that does not allow moisture evaporation (eg, vinyl, rubber, vapor-barrier shoes or boots).^{5,8,14} *Evidence Category: C*
47. For treatment, thoroughly clean and dry the feet, and then treat the affected area by applying warm packs or soaking in warm water (102°F–110°F [39°C–43°C])

A



Wind Chill Chart



B

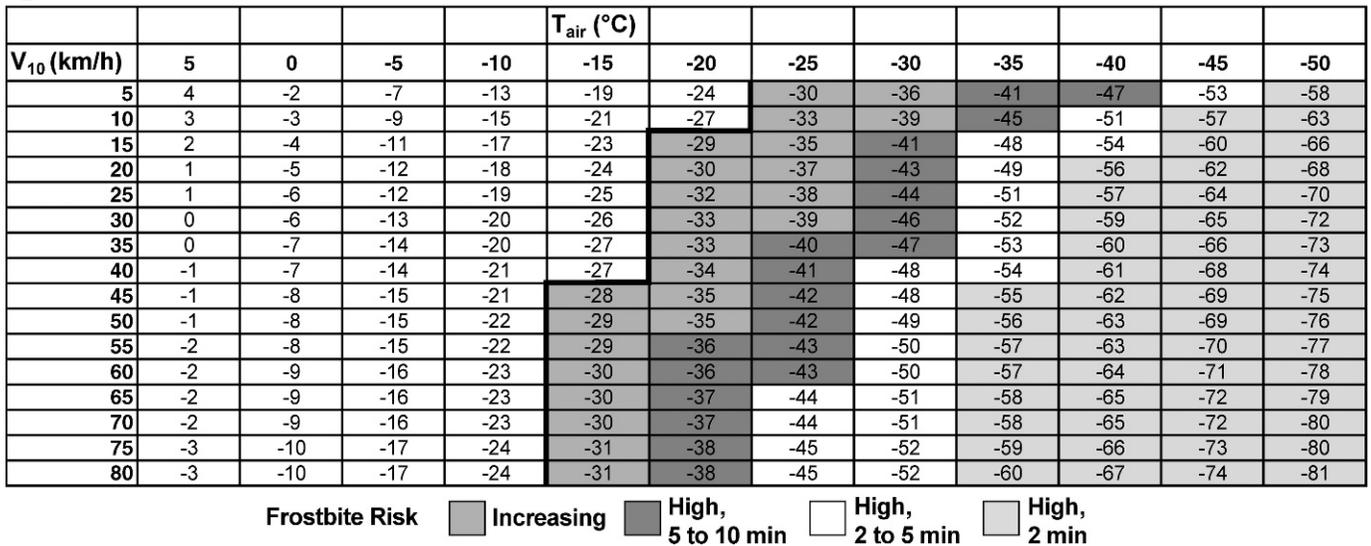


Figure 4. A, United States National Weather Service Wind Chill Chart (figure reproduced from <http://www.weather.gov/os/windchill/images/windchillchart3.pdf>). B, Meteorological Society of Canada/EnvironmentCanada Wind-Chill Calculation Chart (figure adapted from http://www.msc.ec.gc.ca/education/windchill/windchill_chart_e.cfm). T_{air} = Actual air temperature in °C; V₁₀ = wind speed at 10 m in km/h (as reported in weather observations). Temperatures to the left of the bold line are a low risk of frostbite for most people. Increasing indicates within 30 min, increasing risk of frostbite for most people within 30 minutes of exposure; High, 5 to 10 min, high risk for most people in 5 to 10 minutes of exposure; High, 2 to 5 min, high risk for most people in 2 to 5 minutes of exposure; and High, ≤2 min, high risk for most people in ≤2 minutes of exposure. Conversions: °F = (°C × 9/5) + 32; mile = km × 0.6214.

for approximately 5 minutes. Replace wet socks with a clean, dry pair, and rotate footwear or allow footwear to dry before reusing.^{8,14} Evidence Category: C

48. Use a risk management process that includes strategies for preventing, recognizing, and treating cold injuries during events. These strategies can then be used for

Table 4. Approximate Clothing Insulation of Various Clothing Combinations for Cold Weather Exposure⁹⁶

Clothing Combination	Clothing Insulation Factor (clo)
Shirt, lightweight trousers, socks, shoes, underwear briefs	0.6
Shirt, trousers, jacket, socks, shoes, underwear briefs	1
Wind-proof, waterproof jogging suit (jacket and pants), T-shirt, briefs, running shorts, athletic socks, athletic shoes	1.03
Fleece long-sleeved shirt, fleece pants, briefs, athletic socks, athletic shoes	1.19
Lightweight jacket, thermal long underwear top and bottoms, briefs, shell pants, athletic socks, athletic shoes	1.24
Lightweight jacket, long-sleeved fleece shirt, fleece pants, briefs, shell pants, athletic socks, athletic shoes	1.67
Ski jacket with detachable fiberfill liner, thermal long underwear bottoms, knit turtleneck, sweater, fiberfill ski pants, knit hat, goggles, mitten shell with fleece glove inserts, thin knee-length ski socks, insulated waterproof boots	2.3
Extreme cold-weather down-filled parka with hood, shell pants, fiberfill pants liner, thermal long underwear top and bottoms, sweat shirt, mitten shell with inner fleece gloves, thick socks, insulated waterproof boots	3.28
Extreme cold-weather expedition suit with hood (down-filled, 1 piece), thermal long underwear top and bottoms, sweat shirt, mitten with fleece liners, thick socks, insulated waterproof boots	3.67

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preparing and devising risk management protocols and plans when cold injuries may be a possibility. An example of a risk management process is found in Table 3. *Evidence Category: C*

BACKGROUND AND LITERATURE REVIEW

Thermoregulation

During cold stress, a normal body temperature is maintained through a complex regulatory system. A summary of the interactions among various physiologic influences and controls is found in Figure 6. Maintaining a normothermic body temperature depends on a dynamic balance between heat gained from metabolic heat production and heat lost to the environment.^{52–58} The body's basic responses to cold stress in order to maintain normothermia are an increase in metabolic heat production, or thermogenesis, and peripheral vasoconstriction at the skin surface to prevent heat loss to the environment.

An increase in metabolic heat production can be accomplished in 2 ways: nonshivering thermogenesis and shivering thermogenesis. Nonshivering thermogenesis is defined as an increase in metabolic heat production from sources other than muscle contraction and is not considered a major source of metabolic heat in adult humans.^{52–58} Shivering thermogenesis is defined as an increase in heat production due to involuntary muscle contraction.^{52–60} The onset and intensity of shivering are mitigated by the duration and intensity of the cold exposure. The large muscle groups of the trunk are typically the first muscles to begin contracting, and the contractions then spread to the extremities.⁶⁰ These alterations in body metabolism are mediated by the efferent output leaving the thermoregulatory center of the hypothalamus. This efferent output is determined by the integration of afferent input from the skin and the deep body receptors at the great vessels and by

the local temperature of the hypothalamic thermoregulatory center itself.^{58,61}

A decrease in peripheral blood flow due to blood vessel constriction prevents loss of heat to the environment by reducing the thermal gradient (ie, the temperature difference between the skin and the environment). The reduction in peripheral blood flow is highly specific and most pronounced in the extremities. Neural control of peripheral blood flow is under the influence of skin temperature, core temperature, and baroreflexes.^{53–57,61} The vasoconstriction response typically begins at a skin temperature of less than 93.2°F to 95.0°F (34°C–35°C)⁶² and is maximized at skin temperatures of 87.8°F (31°C) and below.⁶³ The magnitude and duration of the vasoconstriction is modulated by a cold-induced vasodilation (CIVD). The CIVD appears to limit the duration of the vasoconstriction and protect the affected area from local cold injury. Periodic fluctuations in blood flow create fluctuations in skin temperature after the initial drop in skin blood flow and temperature with cold exposure. This fluctuation between minimal and greater-than-normal flow continues throughout the cold exposure. The CIVD appears to be mediated by the cessation of norepinephrine release after initial release. It may also occur due to decreases in tissue temperature, which diminish sympathetic nerve conduction and stop the release of norepinephrine. The resulting blood flow increase rewarms the tissue, nerve activity is reestablished, norepinephrine is released, and vasoconstriction is reinitiated. When the CIVD is absent (which can result from certain drugs; see “Nonenvironmental Risk Factors”) and fails to provide protection, the risk of nonfreezing cold injury may increase.^{14,64}

Heat Loss Mechanisms

The human body loses heat to the environment through 4 mechanisms: radiation, convection, conduction, and evaporation. By manipulating environment or clothing (microenvironment) or both, humans can minimize heat

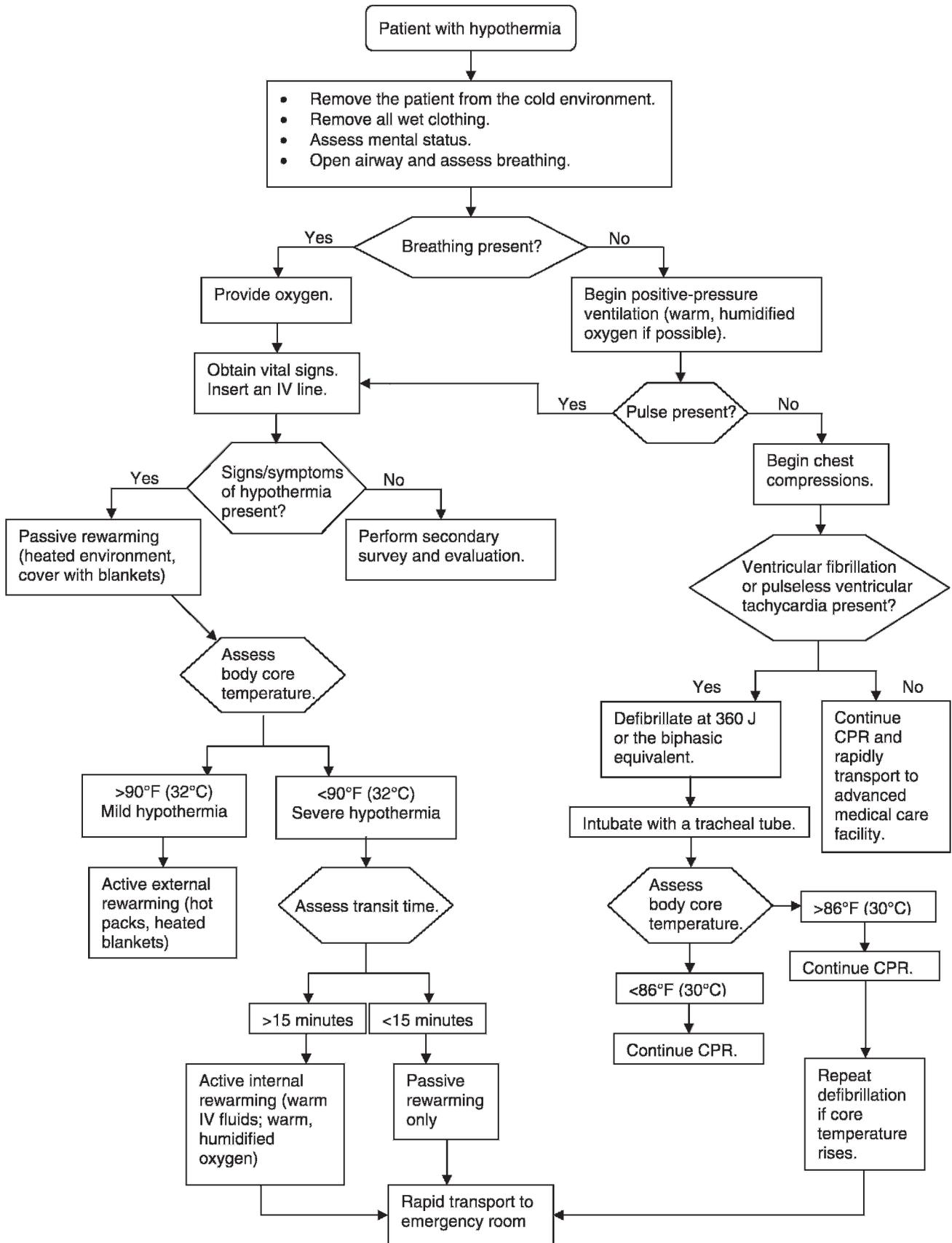


Figure 5. Algorithm for patient with hypothermia.

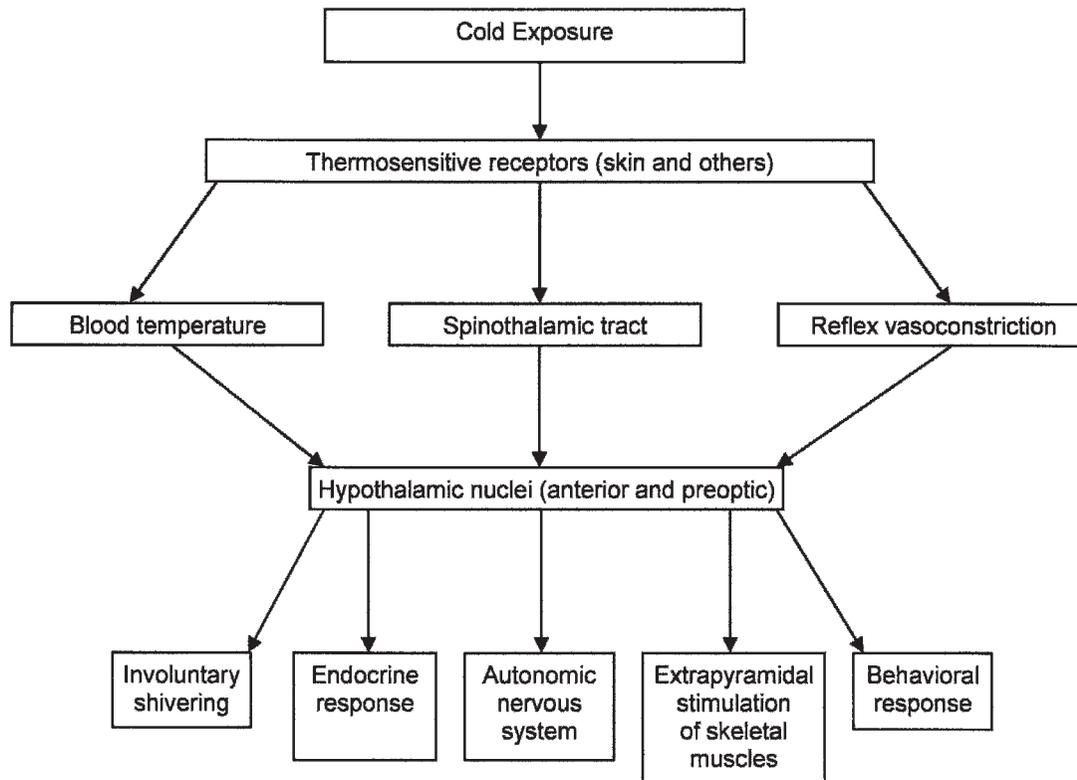


Figure 6. Physiologic responses to cold exposure.

losses. However, with inappropriate choices or when proper choices are unavailable, heat losses are exacerbated, with potentially fatal consequences.

Radiation is heat lost directly to the environment by long-wave (infrared) radiation. Wind, wetness, and other factors do not affect it. Radiative heat loss is greatest at night, especially with the absence of the moon or cloud cover. Uncovered surfaces of the body, especially the head, face, neck, and hands, also increase radiative heat loss. Heat lost through uncovered skin is perceived to be greater because skin temperature is lower and can account for 50% to 65% of all heat losses from the human body in a resting state.⁵²

Convection is heat lost through the movement of air or water across the skin. The human body maintains a thin layer of warm air adjacent to the skin, called the boundary layer. Convection enhances the rate of evaporative heat loss. Air moving across the skin removes this warm layer, replacing it with a cold layer of air that must then be warmed. Air movement may be from wind or a person moving through the air, as in running, skiing, cycling, etc. Depending upon the speed of the air moving across the skin, convective heat losses may be small or large. An estimate of convective heat loss is the wind-chill factor. The amount and insulative properties of clothing worn may reduce or intensify convective heat losses.⁵²

Conduction is heat lost by direct contact with a cold surface. It is exacerbated by moisture, either in the environment (rain, snow, or water immersion) or in wet clothing. Conduction can increase heat loss by up to 5 times with wet clothing and up to 25 times with water immersion. Proper selection of clothing, footwear, layering, and activity level can drastically reduce conductive heat losses. Subcutaneous fat stores also help to reduce

conductive heat loss. Conductive and convective heat losses usually account for approximately 15% of all heat losses; however, high air speed, inadequate amounts of dry clothing, wet clothing, and water immersion all drastically increase conductive and convective heat losses.⁵²

Evaporative heat losses account for 15% to 25% of the total and occur through respiration and exposed skin. Little can be done to reduce evaporative heat losses from respiration, because ambient air must be warmed to core temperature and humidified to 100% as it moves into the respiratory tract. However, like conductive heat losses, losses from the skin and sweating can be controlled through proper selection of clothing and activity level.⁵²

The amount of heat lost from the body can be calculated using the classical heat balance equation. The equation refers to the balancing of heat being produced by metabolism (thermogenesis) and the rate at which it is being lost to the environment via radiation, convection, conduction, and evaporation (thermolysis). This equation provides a conceptual understanding of the interplay between the production of heat within the body and the loss of heat to the environment. When an imbalance develops between the rates of heat production and heat loss, total body heat content changes.⁵² For example, when an athlete ceases exercise, the level of heat production diminishes, while the level of heat loss remains unchanged. This leads to an imbalance between the rates of heat production and heat loss.

$$S = M - R - C - K - E$$

S=heat gain/loss

M=metabolic heat production

R=radiative heat loss

C=convective heat loss

K=conductive heat loss

E=evaporative heat loss

Pathophysiology of Cold Injury

Hypothermia. Hypothermia (defined as a decrease in core body temperature below 95°F [35°C]) can develop in cold and dry or cold and wet conditions and can arise either slowly over many hours or quite suddenly. Generally, slow-onset hypothermia occurs on land, whereas sudden hypothermia results from cold-water immersion or exposure to cold rain.^{1,7-9} The body loses heat faster than it can generate heat and core temperature begins to drop. Although we associate hypothermia with temperatures below freezing, it also occurs at temperatures as high as 50°F to 60°F (10°C–16°C).³ For example, at the 1983 Bostonfest Marathon, 20% of the runners presenting for treatment at the finish-line medical tent were diagnosed with hypothermia³ despite an ambient temperature of approximately 50°F (10°C). During the 1985 Boston Marathon, 75 runners (1.3% of entrants) were treated for hypothermia, even though the ambient temperature was 76°F (24°C).¹

Hypothermia is caused by the body's inability to maintain a normal core temperature, with resulting changes in the function of the nervous system, cardiovascular system, respiratory system, and renal system.⁶² The central nervous system is susceptible to depression during cold exposure. This depressed activity is typically manifested as changes in motor function (eg, clumsiness, loss of finger dexterity, slurred speech), cognition (eg, confusion, poor decision making, memory loss) and level of consciousness. Significant changes in central nervous system function occur as core temperature drops below 95°F (35°C), and show a linear decrease as core temperature continues to drop. Brain function becomes measurably abnormal below 92.3°F (34°C) and ceases at 66°F to 68°F (19°C–20°C).^{7,62}

Cardiovascular system functional change is manifested as an initial tachycardia and then a progressive bradycardia that decreases the resting heart rate by 50%. This drop in heart rate is due to decreased depolarization of the heart pacemaker cells. Other changes include increased myocardial oxygen demand and decreased arterial pressure and cardiac output. The conduction system changes are apparent during an electrocardiogram as prolonged PR, QRS, and QT intervals. Additional arrhythmias may include ventricular fibrillation and asystole, which can occur when the core temperature is below 77°F (25°C), and atrial fibrillation, which is common at core temperatures below 89.6°F (32°C).^{7,62}

The renal system responds to cold exposure by excreting large amounts of glomerular filtrate. This cold-induced diuresis appears to be due to a large increase in central volume secondary to peripheral vasoconstriction. Diuresis may occur to balance fluid levels as the central circulation becomes overloaded. The urine produced is very dilute, regardless of hydration status. Cold-water immersion can further increase urine output by 3.5 times normal. These renal responses occur during rest and light activity.^{7,62}

The respiratory system reacts to cold exposure by initially producing a hyperventilation response. After the initial response, however, the ventilation rate progressively decreases as core temperature drops, reaching 5 to 10

breaths per minute below a core temperature of 86°F (30°C). Carbon dioxide production also decreases by up to 50%, which can lead to respiratory acidosis. These respiratory responses occur during rest and light activity.^{7,62}

Frostbite. The pathophysiology of deep frostbite (defined as actual freezing of body tissues) typically consists of 3 distinct phases: frostnip, mild frostbite, and deep frostbite. The frostnip, or prefreeze, phase occurs with superficial skin cooling below 50°F (10°C), resulting in loss of sensation, constriction of the microvasculature, plasma leakage, and increased viscosity of vascular contents.⁶⁵

Mild frostbite, or freeze-thaw phase, begins as skin temperature drops below 28°F (–2°C) and extracellular ice crystals form. The location and rate of crystal formation depends on the severity of the cold stress (combination of air temperature, moisture presence, and wind). Water migrates across the cell membrane, resulting in intracellular dehydration and increased intracellular electrolyte concentrations. As the cell volume decreases, the cell eventually collapses, the membrane ruptures, and cell death occurs. As crystallization progresses, surrounding cells and vascular structures are compressed.^{65–67}

The third phase, or severe frostbite, results in microvascular collapse at the arteriole and venule levels. As microvascular tissue fails, blood viscosity increases, resulting in microthrombi, plasma leakage, increased tissue pressure, ischemia, and tissue death. Nerve and muscle tissue may also be affected. As edema resolves over approximately 72 hours from onset, the most noticeable sign of frostbite is gangrenous tissue.^{65–67}

Blood flow to the skin of the extremities (acral skin) (eg, fingers, toes, tip of nose) is under much stronger local control of vasoconstriction and vasodilation than nonacral skin and is very sensitive to local changes in temperature. This local control is independent of the central nervous system and produces significant changes in blood flow to skin that is cooled, regardless of changes in core temperature. Nonacral skin blood flow is controlled by the central nervous system, and changes in flow are likely due to changes in core temperature. These differences in control of blood flow produce the scenario of potential freezing injury to acral skin without a significant drop in core body temperature.⁶⁸

Chilblain (Pernio) and Immersion (Trench) Foot. Non-freezing cold injuries, such as chilblain and immersion foot, appear to share a common pathophysiology and generally result in cellular damage without ice crystal formation.⁶⁷ The abnormal inflammatory response typically leads to intracellular and then extracellular fluid build-up. Increased extracellular fluid results in damage to neurologic and vascular tissue. Classic symptoms of swelling, numbness, and itching, followed by hypersensitivity to cold after rewarming, appear related to a dermal edema, migration of lymph fluid, microvascular damage, and vascular and neurologic hypersensitivity to cold exposure.⁶⁷

Nonenvironmental Risk Factors

Health care providers should be aware of the following nonenvironmental risk factors, which may make athletes more susceptible to cold injury and may affect normal physiologic responses to cold exposure.

Previous Cold Injuries. Having sustained a previous cold injury increases the chance of subsequent cold injuries by 2 to 4 times, even if prior injuries were not debilitating or resolved with no or minimal medical care.^{3,6,69} For example, an athlete who sustained frostnip or frostbite is 2 to 4 times more likely to develop frostbite in the same area again, given similar environmental conditions.

Low Caloric Intake, Dehydration, and Fatigue. Low caloric intake (less than 1200 to 1500 kcal/day) or hypoglycemia (or both) directly decreases metabolism and concomitant heat production,⁵ contributing to the inability to maintain body temperature balance through physical activity. Dehydration does not negatively affect peripheral vasoconstriction or shivering and, therefore, does not appear to increase susceptibility to cold injury.^{5,70–80} Fatigue associated with hypoglycemia is linked to impaired peripheral vasoconstriction and shivering responses and can lead to faulty decision making and inadequate preparations, indirectly resulting in cold injuries.

Race. Black individuals have been shown to be 2 to 4 times more likely than individuals from other racial groups to sustain cold injuries. These differences may be due to cold weather experience, but are likely due to anthropometric and body composition differences, including less-pronounced CIVD, increased sympathetic response to cold exposure, and thinner, longer digits.^{3,6}

Nicotine, Alcohol, and Drug Use. Nicotine inhaled through smoking causes a reflex peripheral vasoconstriction, possibly negating the CIVD and later enhancing the cold-induced vasoconstriction to maintain core temperature. Alcohol reduces the glucose concentration in the blood, which tends to decrease the shivering response. Alcohol also may lead to faulty decision making due to its effects on the central nervous system. Drugs with a depressive effect may impair the thermoregulatory system and so inhibit the body's reaction to cold by blunting the peripheral vasoconstriction and shivering responses. As with alcohol, they may also lead to faulty decision making.^{77,81,82}

Body Size and Composition. Body fat and muscle mass appear to be instrumental in providing protection for maintaining core body temperature with exposure to cold air and water. This effect appears in both males and females regardless of the amount of clothing worn. Strong evidence suggests that percentage of body fat (>25%, approximately) and amount of muscle mass are reliable predictors of the ability to maintain and protect core body temperature during prolonged exposure to a wide range of cold air and water temperatures. The greater the level of body fat and muscle mass, the better the ability to protect core body temperature through passive (eg, insulative properties of fat) and active (eg, shivering thermogenesis) mechanisms.^{83–86}

Aerobic Fitness Level and Training. Overall, physical training and fitness level appear to have only minor influence on thermoregulatory responses to cold. Most cross-sectional comparisons of aerobically fit and less-fit persons show no relationship between maximal aerobic exercise capability and temperature regulation in the cold. In those studies purportedly demonstrating a relationship,^{87,89} differences in thermoregulation appear more likely attributable to anthropometric (body size and composition) differences between aerobically fit and

less-fit participants, rather than an effect of maximal aerobic exercise capabilities per se. The primary thermoregulatory advantage provided by increased endurance resulting from physical training is that a fitter individual can sustain higher-intensity, longer-duration voluntary activity than a less-fit person and, thus, maintain higher sustained rates of metabolic heat production, keeping the former warmer. In addition, exercise training has been suggested to enhance the peripheral vasoconstriction response, which would conserve body heat but possibly increase peripheral cold injury risks.^{87–91}

Sex. The hypothermia injury rate for females is 2 times higher than for males. Sex differences in thermoregulatory responses during cold exposure are influenced by interactions among total body fat content, subcutaneous fat thickness, amount of muscle mass, and surface area-to-mass ratio. For example, among men and women with equivalent total body mass and surface areas, women's greater fat content enhances insulation and reduces the fall in core temperature. In women and men of equivalent subcutaneous fat thickness, women typically have a greater surface area but smaller total body mass and smaller muscle mass (thus, lower total body heat content) than men and lose heat at a greater rate. Women's thermogenic response to cold exposure also appears less able to generate metabolic heat than men of similar body composition due to less total muscle mass. Therefore, total heat loss is greater in women due to the larger surface area for convective heat loss, and body temperature tends to fall more rapidly for any given cold stress.^{91–94} As a result, the prevention and recognition recommendations in this position statement should be interpreted and used more conservatively for female athletes than male athletes.

Clothing. The role of clothing in preventing cold injuries lies in its ability to reduce heat loss to the environment by trapping warm air. Cold-weather clothing typically has an internal layer that allows evaporation of sweat without absorption, a middle layer that provides insulation, and an external layer that is wind and water resistant and allows evaporation of moisture. The internal layer is in direct contact with the skin and uses a moisture-wicking material such as polyester or polypropylene. This layer should not retain moisture but should transfer the moisture to other layers, from which it can evaporate. The middle layer provides the primary insulation against heat loss and can be a fleece or wool material. The outer layer should have venting abilities (eg, zippers or mesh in the armpits or low back area) to allow moisture transfer to the environment.⁹⁵

Clothing requirements for cold environment exercise depend on ambient temperature; presence of wind, rain, and water; and activity intensity. Generally speaking, as exercise intensity increases at any given air temperature, the amount of clothing insulation needed to maintain body temperature equilibrium decreases. The insulative protection offered by different clothing combinations is represented in units of clo. One clo of insulation is the clothing needed to permit a person to rest comfortably when the air temperature is 70°F (21°C).⁹⁶ See Table 4 for a list of the clo values of common clothing combinations. Interactions between exercise intensity and ambient conditions dictate the selection of clothing based upon clo values. For example, if the ambient temperature is 20°F (−7°C) and the person is at rest (1 metabolic equivalent of exercise

intensity), then clothing with a clo value of approximately 5 is needed to maintain core temperature. If activity is increased to an exercise intensity of 4 metabolic equivalents, then clothing with a clo value of approximately 2 would be adequate.⁵ However, imposing a standard clothing requirement for a group of individuals could result in overheating in some and inadequate protection in others. Individuals should be able to adjust clothing according to their specific responses and comfort levels while following general guidelines.

Other clothing considerations include the ability to adapt when weather conditions change, especially if rain, snow, or water immersion is a possibility. In these instances, waterproof or water-resistant clothing should be available, and the athlete should be able to change into dry clothing as needed. Socks should not constrict blood flow, should allow the evaporation of moisture, and should be changed frequently after sweating or water immersion. Another consideration is the heat lost from an uncovered head. Even if the remainder of the body is well insulated in ambient temperatures of 25°F (−4°C),⁹⁷ fleece, knit caps, or other complete head coverings can significantly reduce heat loss.

Predisposing Medical Conditions

The following medical conditions add to the risk of cold injury or are exacerbated by exposure to cold environments.

Exercise-Induced Bronchospasm. Exercise-induced bronchospasm (EIB), also called exercise-induced asthma or airway hyperresponsiveness, is a narrowing of the respiratory tract airways. It is exacerbated by exposure to cold, dry air. Predisposing factors include asthma and allergies. However, not all individuals with EIB have asthma or allergies, and not all individuals with asthma or allergies have EIB. This condition can affect any individual, from a small child at play to an elite athlete.^{98–101} Authors¹⁰² have noted a high prevalence of EIB in cold-weather athletes, in women, and in athletes training and competing in indoor ice facilities. Two possible mechanisms have been suggested to explain EIB. The osmotic theory postulates that excessive airway drying due to increased breathing rates enhances the secretion of vasoconstrictor mediators in the breathing passageways. This increased vasoconstrictor response then limits air flow.¹⁰³ A second theory suggests that the combination of cold air and increased breathing rate cools airways; after airways rewarm, increased blood flow leads to edema formation and airway flow reductions.¹⁰³ The reduction of airway flow reduces maximal ventilation and, subsequently, maximal performance.

Raynaud Syndrome. As with EIB, Raynaud syndrome is caused by cold exposure and characterized by intermittent vasospasm of the digital vessels. This vasospasm significantly reduces blood flow to the extremities. The affected area may present with tingling, swelling, or a throbbing pain. The skin may turn a shade of white, then possibly blue, and then becomes red upon rewarming. Raynaud syndrome describes a spectrum of disorders whose causes are usually idiopathic, although infrequently, autonomic dysfunction or an underlying condition such as thoracic outlet syndrome or collagen vascular disease may be responsible.⁶⁷

Anorexia Nervosa. Anorexia nervosa results in a deficiency of body fat stores, potential malnutrition, decreased metabolic rate, and peripheral vasoconstriction. These changes limit the ability to maintain a normal core temperature.^{104–106}

Cold Urticaria. Cold urticaria may be the most common form of urticaria.^{5,107} The condition has a rapid onset, presenting with wheals (hives) that may be local or generalized, redness, itching, and edema. Other symptoms may include fatigue, headache, dyspnea, and in rare cases, anaphylactic shock. Two forms of the condition, primary acquired and secondary acquired (hereditary), have been identified and differ in speed of onset: within minutes or 24 to 48 hours after cold exposure, respectively.¹⁰⁷

Cardiovascular Disease. Individuals with cardiovascular disease are sensitive to increased demands on the myocardium and increases in blood pressure, as well as having potentially decreased flow to cutaneous and subcutaneous tissues. Cold exposure coupled with exercise increases the demand on the cardiovascular system by increased sympathetic nervous system activity, peripheral resistance, blood pressure, and myocardial oxygen demands. This increased stress is in contrast to the demands of rest and exercise in warm environments.^{108,109} Therefore, individuals with diminished cardiovascular system function should be cautious when exercising in the cold and should be monitored closely for symptoms associated with a myocardial infarction.

Environmental Risk Factors

Environmental cold stress results from a combination of low air temperature; humidity, rain, or immersion; and little thermal radiation and air movement. An index of cold stress is the wind-chill temperature index (WCT) (Figure 4). This index gives an indication of how cold a person feels when exposed to a combination of cold air and wind. This index estimates the danger of extreme cooling of exposed skin (ie, the risk of frostbite) while walking at 3 mph (1.3 m·s^{−1}) in various combinations of conditions. When the WCT is below −18°F (−27°C), the risk of developing frostbite in exposed skin in less than 30 minutes increases, warranting closer observation. However, the environmental wind speed in the WCT does not account for wind produced during movement. Biking or running produce wind across the body at the same rate as body movement and should be taken into account when estimating risk. For example, if a light wind is present (less than 5 mph [2.24 m·s^{−1}]) with cold temperatures (25°F [−4°C]), the risk of frostbite for most people is low. Yet if the athlete is cycling at 15 mph (16.71 m·s^{−1}), the relative risk of developing frostbite increases. Therefore, the effect of air movement produced by the body should be taken into account when using the wind-chill recommendations. The WCT is calculated using the following formula²⁷:

$$\text{Wind chill (}^\circ\text{F)} = 35.74 + 0.6215T - 35.75V^{0.16} + 0.4275T(V^{0.16})$$

Where T = Air temperature (°F), and V = Wind speed (mph)

This index is a useful tool to monitor the potential thermal stress athletes must deal with when exposed to cold temperatures.

Influence of Wind, Rain, and Immersion

Exercise during windy or rainy conditions or water immersion poses several unique challenges to the body's ability to maintain a normal body temperature. The transfer of body heat in water may be 70 times greater than in air.⁵² This transfer can lead to a significant loss of body heat for those exercising in rainy conditions or in the water. Maintaining normal body temperature in those conditions depends on several factors, including exercise intensity, exercise mode, anthropometric factors, insulative properties of clothing and equipment, and the magnitude of heat loss caused by wind speed, amount of rain, or water temperature.⁵²

During exercise in cold and wet conditions, the ability to generate adequate metabolic heat to maintain body temperature depends on exercise intensity and mode.⁵¹ More heat is lost from the arms and legs due to smaller diameter and shorter distance from the limb center to the surface, which allows rapid heat conduction compared with the trunk. This increased conduction is outweighed when exercise intensity is greater than 75% of maximal oxygen uptake ($\dot{V}O_2$ max).^{53,54} Exercise mode also affects the ability to generate metabolic heat. The more muscle mass involved in performing the exercise, the greater the heat generated. Therefore, exercise involving only the lower body generates more heat than exercise involving only the upper body, and whole-body exercise generates more heat than lower or upper body exercise alone.

The combination of windy and wet conditions can also affect body temperature maintenance. Light to moderate exercise in the rain leads to decreases in core temperature compared with resting conditions.⁷⁹ When air temperature is -15.0°F (5°C) and clothes are wet, heat losses may double those observed in dry conditions.⁵⁵ Decreases in core temperature have been observed with the addition of wind during light-intensity exercise. With high-intensity exercise (greater than 60% of peak oxygen consumption), body temperature can be maintained in cold, windy, and wet conditions.⁵⁶

The influence of water temperature and amount of wet or immersed skin also influences heat loss. The lower the water temperature and the larger the surface area of the body immersed in or in contact with water, the greater the heat loss and the more rapid the decreases in core temperature.⁵⁷

The best survival strategy for an individual exposed to accidental, prolonged cold-water immersion (eg, boating or water craft accident) has been debated: try to swim for safety, move vigorously in place to generate body heat, or remain quiet and move very little to conserve body heat. Recent researchers¹¹⁰ suggested the following strategies:

1. Stay calm. Unless you're wearing an immersion suit, you'll experience cold shock when you go into cold water due to rapid cooling of the skin. You won't be able to control your breathing, and you won't get far if you try to swim at this point. Your breathing will return to normal in 2 to 3 minutes.
2. Make a plan. While you're waiting for the cold shock to subside, consider your situation and decide whether to swim or stay in the present location.
3. If you decide to swim, look for the shore and decide if you can make it. Most people studied could swim

between 800 and 1500 m (874.9 and 1640.4 y) in cold water, or for 45 minutes, before their arms and leg muscles cooled to the point that they could no longer swim.

4. If you decide to stay in place, try to get out of the water as much as possible. Complete any tasks that require the use of your hands, such as tying knots or turning on flares, as soon as possible. As your hands cool, they lose dexterity.
5. Stick to your decision; don't change your mind midway. After 30 minutes in cold water, you may become hypothermic, and you won't make the best decisions.

After immersion in cold water, athletes should be monitored for the phenomenon of afterdrop. Once physical activity concludes, the body still dissipates significant amounts of heat but no longer generates metabolic heat through activity. This imbalance between heat loss and heat production could lead to drops in core temperature during the rewarming process. Afterdrop is commonly found in individuals after prolonged water immersion in which core temperature has already decreased.³⁰⁻³²

Role of Cold Acclimatization. Cold acclimatization may play a role in tolerance of cold exposure, but observed adjustments are modest and rely upon the severity of previous exposure. Adjustments found in persons with recurring exposure to cold are habituation, metabolic acclimatization, and insulative acclimatization.¹¹¹

Cold-induced habituation manifests itself as a decreased shivering and vasoconstriction response during cold exposure compared with nonacclimatized exposures. Some individuals may also have a greater decrease in core temperature than nonacclimatized persons; this is known as hypothermic habituation. Short, intense exposures that occur a few times per week appear to elicit habituation. Hypothermic habituation, however, occurs with longer exposures in moderate temperatures during consecutive days over a period greater than 2 weeks.¹¹¹

Metabolic acclimatization is characterized by a more pronounced shivering response to cold and typically occurs after long-term exposures. Insulative acclimatization is produced by greater conservation of heat during exposure to cold. This includes a larger and more rapid decline in skin temperature, resulting in less heat conduction at the skin. Another response may be improved convective heat loss due to a circulatory countercurrent heat exchange mechanism.¹¹¹

In comparison with the acclimatization observed with repeated environmental heat exposure, adjustment to the physiologic response during cold exposure appears to be more difficult to acquire, varies more from individual to individual, develops more slowly, and has less of a preventive effect.

SPECIAL CONCERNS

Children (Prepubescents)

Exposure to cold environments poses unique challenges for young athletes due to their higher surface area-to-mass ratios and smaller adipose tissue deposits. These factors result in a faster cooling rate than for adults in water and similar cooling rates as adults in cold air. These rates

appear to be a function of a higher level of metabolic heat production and a stronger peripheral vasoconstriction response.^{112,113} Therefore, children should take similar preventive measures as those suggested for adults, but they should be encouraged to take more frequent breaks from a cold environment, especially water immersion.

Older Individuals (More Than 50 Years Old)

As one ages, the ability to tolerate cold decreases and risk of hypothermia increases.^{5,6} This increased risk for hypothermia appears to be due to diminished sympathetic nervous system-mediated reflex vasoconstriction, which allows greater heat loss.¹¹⁴ Additionally, an older individual is more likely to have health concerns such as diabetes, hypothyroidism, hypopituitarism, or hypertension, which increase the likelihood of cold injury. As older individuals continue an active lifestyle and as advances in medical and surgical treatments extend life, more persons with histories of myocardial infarction and stroke are becoming active in outdoor environments.^{6,55,62,64,115,116} Thus, ATs working with older athletes should apply prevention and recognition recommendations more conservatively in this population.

Spinal Cord Injuries

The presence of a spinal cord injury (SCI) resulting in some form of paralysis is associated with an increased risk for hypothermia during cold exposure.^{117,118} Athletes with SCI tend to be less sensitive to the sensation of cold on the skin surface and have a diminished perception of skin temperature change.^{119,120} Overall, individuals with SCI have a diminished capability to stabilize core temperature¹²¹; specifically, they have a diminished autonomic response to cold, which results in a decreased vasoconstriction response^{117,122} and a decreased effector response to the muscle to generate metabolic heat.¹²² Those with SCI also have abnormal blood pressure responses to cold exposure,¹²³ as well as increased complaints of muscle spasticity, pain, and numbness.^{124–127} Consequently, athletes with SCI should be monitored closely for both core temperature changes and skin changes associated with nonfreezing cold injury.

CONCLUSIONS

Certified athletic trainers and other health care providers must be able to identify the signs and symptoms of hypothermia, frostbite, chilblains, and immersion (trench) foot in athletes. This position statement outlines the current recommendations to prevent the occurrence and improve the recognition and treatment of cold injury in athletes.

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REFERENCES

1. Sallis R, Chassay CM. Recognizing and treating common cold-induced injury in outdoor sports. *Med Sci Sports Exerc.* 1999;31(10):1367–1373.
2. Rav-Acha M, Heled Y, Moran DS. Cold injuries among Israeli soldiers operating and training in a semiarid zone: a 10-year review. *Mil Med.* 2004;169(9):702–706.
3. Reynolds K, Williams J, Miller C, Mathis A, Dettori J. Injuries and risk factors in an 18-day Marine winter mountain training exercise. *Mil Med.* 2000;165(12):905–910.
4. Jones BH, Rock PB, Smith LS, et al. Medical complaints after a marathon run in cool weather. *Physician Sportsmed.* 1985;13(10):103–110.
5. Castellani JW, Young AJ, Ducharme MB, Giesbrecht GG, Glickman E, Sallis RE. American College of Sports Medicine position stand: prevention of cold injuries during exercise. *Med Sci Sports Exerc.* 2006;38(11):2012–2029.
6. DeGroot DW, Castellani JW, Williams JO, Amoroso PJ. Epidemiology of U.S. Army cold weather injuries, 1980–1999. *Aviat Space Environ Med.* 2003;74(5):564–570.
7. Danzl DF. Accidental hypothermia. In: Marx JA, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice.* Vol 3. St Louis, MO: Mosby; 2006:2236–2254.
8. Hamlet MP. Human cold injuries. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes.* Indianapolis, IN: Benchmark Press; 1988:435–466.
9. Ulrich AS, Rathlev NK. Hypothermia and localized cold injuries. *Emerg Med Clin North Am.* 2004;22(2):281–298.
10. Murphy JV, Banwell PE, Roberts AHN, McGrouther DA. Frostbite: pathogenesis and treatment. *J Trauma.* 2000;48(1):171–178.
11. Hamlet MP. Prevention and treatment of cold injury. *Int J Circumpolar Health.* 2000;59(2):108–113.
12. Hassi J. Frostbite, a common cold injury: challenges in treatment and prevention. *Int J Circumpolar Health.* 2000;59(2):90–91.
13. Mills WJ. Clinical aspects of freezing cold injury. In: Pandolf KB, Burr RE, eds. *Textbooks of Military Medicine: Medical Aspects of Harsh Environments.* Vol 1. Falls Church, VA: Office of The Surgeon General, US Army; 2002:429–466.
14. Thomas JR, Oakley EHN. Nonfreezing cold injury. In: Pandolf KB, Burr RE, eds. *Textbooks of Military Medicine: Medical Aspects of Harsh Environments.* Vol 1. Falls Church, VA: Office of The Surgeon General, US Army; 2002:467–490.
15. Strength of Recommendation Taxonomy (SORT). American Academy of Family Physicians. <http://www.aafp.org/afp/20040201/548.html>. Accessed July 2, 2008.
16. Hassi J, Raatikka VP, Huurre M. Health-check questionnaire for subjects exposed to cold. *Int J Circumpolar Health.* 2003;62(4):436–443.
17. Askew EW. Environmental and physical stress and nutrient requirements. *Am J Clin Nutr.* 1995;61(3 suppl):631S–637S.

18. Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *J Athl Train*. 2000;35(2):212–224.
19. United States Department of Agriculture. MyPyramid.gov. www.mypyramid.gov. Accessed February 1, 2006.
20. Knight DR, Horvath SM. Urinary responses to cold temperature during water immersion. *Am J Physiol*. 1985;248(5, pt 2):R560–R566.
21. Allen DE, Gellai M. Mechanisms for the diuresis of acute cold exposure: role for vasopressin? *Am J Physiol*. 1993;264(3, pt 2):R524–R532.
22. Sramek P, Simeckova M, Jansky L, Savlikova J, Vybiral S. Human physiological responses to immersion into water of different temperatures. *Eur J Appl Physiol*. 2000;81(5):436–442.
23. Kenefick RW, Hazzard MP, Mahood NV, Castellani JW. Thirst sensations and AVP responses at rest and during exercise-cold exposure. *Med Sci Sports Exerc*. 2004;36(9):1528–1534.
24. Kuehn LA. Managing and monitoring cold stress in sport. In: *Sports Science Periodical on Research and Technology in Sport*. Ottawa, ON, Canada: The Coaching Association of Canada; 1985:51–62.
25. Priest S, Dixon T, Strong M. Preventing cold injuries. *J Phys Educ Rec Dance*. 1989;60(8):45–50.
26. Danielsson U. Windchill and the risk of tissue freezing. *J Appl Physiol*. 1996;81(6):2666–2673.
27. National Weather Service. Windchill terms and definition. <http://www.nws.noaa.gov/om/windchill/windchillglossary.shtml>. Accessed February 15, 2006.
28. Moran DS. Current opinion: potential applications of heat and cold stress indices to sporting events. *Sports Med*. 2001;31(13):909–917.
29. Holmer I. Work in the cold: review of methods for assessment of cold exposure. *Int Arch Occup Environ Health*. 1993;65(3):147–155.
30. Rissanen S, Rintamaki H. Thermal responses and physiological strain in men wearing impermeable and semipermeable protective clothing in the cold. *Ergonomics*. 1997;40(2):141–150.
31. Smolander J, Louhevaara V, Ahonen M. Clothing, hypothermia, and long-distance skiing. *Lancet*. 1986;2(8500):226–227.
32. Tanaka M, Tochihara Y, Yamazaki S, Ohnaka T, Yoshida K. Thermal reaction and manual performance during cold exposure while wearing cold-protective clothing. *Ergonomics*. 1983;26(2):141–149.
33. Nuckton TJ, Claman DM, Goldreich D, Wendt FC, Nuckton JG. Hypothermia and afterdrop following open water swimming; the Alcatraz/San Francisco swim study. *Am J Emerg Med*. 2000;18(6):703–707.
34. Romet TT. Mechanism of afterdrop after cold water immersion. *J Appl Physiol*. 1988;65(4):1535–1538.
35. Giesbrecht GG, Goheen MS, Johnston CE, Kenny GP, Bristow GK, Hayward JS. Inhibition of shivering increases core temperature afterdrop and attenuates rewarming in hypothermic humans. *J Appl Physiol*. 1997;83(5):1630–1634.
36. Moran DS, Mendal L. Core temperature measurement: methods and current insights. *Sports Med*. 2002;32(14):879–885.
37. Danzl DF, Lloyd EL. Treatment of accidental hypothermia. In: Pandolf KB, Burr RE, eds. *Textbooks of Military Medicine: Medical Aspects of Harsh Environments*. Vol 1. Falls Church, VA: Office of The Surgeon General, US Army; 2002:491–529.
38. Petrone P, Kuncir EJ, Asensio JA. Surgical management and strategies in the treatment of hypothermia and cold injury. *Emerg Med Clin North Am*. 2003;21(4):1165–1178.
39. Kempainen RR, Brunette DD. The evaluation and management of accidental hypothermia. *Respir Care*. 2004;49(2):192–205.
40. Delaney KA, Howland MA, Vassallo S, Goldfrank LR. Assessment of acid-base disturbances in hypothermia and their physiologic consequences. *Ann Emerg Med*. 1989;18(1):72–82.
41. Giesbrecht GG, Bristow GK, Uin A, Ready AE, Jones RA. Effectiveness of three field treatments for induced mild (33.0 degrees C) hypothermia. *J Appl Physiol*. 1987;63(6):2375–2379.
42. Daanen HA, Van de Linde FJ. Comparison of four noninvasive rewarming methods for mild hypothermia. *Aviat Space Environ Med*. 1992;63(12):1070–1076.
43. Kornberger E, Schwarz B, Lindner KH, Mair P. Forced air surface rewarming in patients with severe accidental hypothermia. *Resuscitation*. 1999;41(2):105–111.
44. Walpoth BH, Walpoth-Aslan BN, Mattle HP, et al. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. *New Engl J Med*. 1997;337(21):1500–1505.
45. Visetti E, Pastorelli M, Bruno M. Severe accidental hypothermia successfully treated by warmed peritoneal lavage. *Minerva Anestesiol*. 1998;64(10):471–475.
46. Aslam AF, Aslam AK, Vasavada BC, Khan IA. Hypothermia: evaluation, electrocardiographic manifestations, and management. *Am J Med*. 2006;119(4):297–301.
47. Collins AM, Danzl DF. Hypothermia with profound anemia and pancreatitis. *Wilderness Environ Med*. 2006;17(1):31–35.
48. Biem J, Koehncke N, Classen D, Dosman J. Out of the cold: management of hypothermia and frostbite. *CMAJ*. 2003;168(3):305–311.
49. Bird D. Identification and management of frostbite injuries. *Emerg Nurse*. 2000;7(8):17–19.
50. Murphy JV, Banwell PE, Roberts AHN, McGroutner DA. Frostbite: pathogenesis and treatment. *J Trauma*. 2000;48(1):171–178.
51. Bruen KJ, Ballard JR, Morris SE, Cochran A, Edelman LS, Saffle JR. Reduction of the incidence of amputation in frostbite injury with thrombolytic therapy. *Arch Surg*. 2007;142(6):546–551.
52. Toner MM, McArdle WD. Human thermoregulatory responses to acute cold stress with special reference to water immersion. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology: Environmental*. Vol 1. New York, NY: Oxford Press; 1996:379–397.
53. Ferretti G, Veicsteinas A, Rennie DW. Conductive and convective heat flows of exercising humans in cold water. *J Appl Physiol*. 1989;67(6):2473–2480.
54. Toner MM, Sawka MN, Pandolf KB. Thermal responses during arm and leg and combined arm-leg exercise in water. *J Appl Physiol*. 1984;56(5):1355–1360.
55. Kaufman WC, Bothe DJ. Wind chill reconsidered, Siple revisited. *Aviat Space Environ Med*. 1986;57(1):23–26.
56. Weller AS, Millard CE, Stroud MA, Greenhaff PL, Macdonald IA. Physiological responses to cold stress during prolonged intermittent low- and high-intensity walking. *Am J Physiol*. 1997;272(6, pt 2):R2025–R2033.
57. Lee DT, Toner MM, McArdle WD, Vrabas JS, Pandolf KB. Thermal and metabolic responses to cold-water immersion at knee, hip, and shoulder levels. *J Appl Physiol*. 1997;82(5):1523–1530.
58. Young AJ, Castellani JW, Sawka MN. Human physiological responses to cold exposure. In: Nose H, Nadel ER, Morimoto T, eds. *Proceedings of 1997 Nagano Symposium on Sports Science*. Carmel, IN: Cooper Publishing Group; 1998:273–286.
59. Jacobs I, Martineau L, Vallerand AL. Thermoregulatory thermogenesis in humans during cold stress. *Exerc Sports Sci Rev*. 1994;22:221–250.
60. Tikuisis P, Bell DG, Jacobs I. Shivering onset, metabolic response, and convective heat transfer during cold air exposure. *J Appl Physiol*. 1991;70(5):1996–2002.
61. Rothwell NJ. CNS regulation of thermogenesis. *Crit Rev Neurobiol*. 1994;8(1–2):1–10.
62. Savage MV, Brengelmann GL. Control of skin blood flow in the neutral zone of human body temperature regulation. *J Appl Physiol*. 1996;80(4):1249–1257.
63. Veicsteinas A, Ferretti G, Rennie DW. Superficial shell insulation in resting and exercising men in cold water. *J Appl Physiol*. 1982;52(6):1557–1564.
64. O'Brien C. Reproducibility of the cold-induced vasodilation response in the human finger. *J Appl Physiol*. 2005;98(4):1334–1340.
65. Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PA, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St Louis, MO: Mosby; 1995:51–103.

66. Danzl DF. Frostbite. In: Marx JA, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. Vol 3. St Louis, MO: Mosby; 2006:2228–2236.
67. McCauley RL, Smith DJ Jr, Robson MC, Hegggers JP. Frostbite and other cold-induced injuries. In: Auerbach PA, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St Louis, MO: Mosby; 1995:129–145.
68. Charkoudian N. Skin blood flow in adult human thermoregulation: how it works, when it does not, and why. *Mayo Clin Proc*. 2003;78(5):603–612.
69. Healy JD. Excess winter mortality in Europe: a cross country analysis identifying key risk factors. *J Epidemiol Community Health*. 2003;57(10):784–789.
70. Freund BJ, Sawka MN. Influence of cold stress on human fluid balance. In: Marriot BM, Carlson SJ, eds. *Nutritional Needs in Cold and High-Altitude Environments: Applications for Military Personnel in Field Operations*. Washington, DC: National Academy Press; 1996:161–180.
71. O'Brien C, Young AJ, Sawka MN. Hypohydration and thermoregulation in cold air. *J Appl Physiol*. 1998;84(1):185–189.
72. Layden JD, Patterson MJ, Nimmo MA. Effects of reduced ambient temperature on fat utilization during submaximal exercise. *Med Sci Sports Exerc*. 2002;34(5):774–779.
73. Makinen TT, Gavhed D, Holmer I, Rintamaki H. Effects of metabolic rate on thermal responses at different air velocities in -10°C . *Comp Biochem Physiol A Mol Integr Physiol*. 2001;128(4):759–768.
74. Fellows IW, Macdonald IA, Bennett T, Allison SP. The effect of undernutrition on thermoregulation in the elderly. *Clin Sci (Lond)*. 1985;69(5):525–532.
75. Ainslie PN, Campbell IT, Lambert JP, MacLaren DPM, Reilly R. Physiological and metabolic aspects of very prolonged exercise with particular reference to hill walking. *Sports Med*. 2005;35(7):619–647.
76. Tikuisis P, Ducharme MB, Moroz D, Jacobs I. Physiological responses of exercised-fatigued individuals exposed to wet-cold conditions. *J Appl Physiol*. 1999;86(4):1319–1328.
77. Castellani JW, Young AJ, Sawka MN, Pandolf KB. Human thermoregulatory responses during serial cold water immersions. *J Appl Physiol*. 1998;85(1):204–209.
78. Young AJ, Castellani JW, O'Brien C, et al. Exertional fatigue, sleep loss, and negative energy balance increase susceptibility to hypothermia. *J Appl Physiol*. 1998;85(4):1210–1217.
79. Castellani JW, Young AJ, Degroot DW, et al. Thermoregulation during cold exposure after several days of exhaustive exercise. *J Appl Physiol*. 2001;90(3):939–946.
80. Castellani JW, Stulz DA, Degroot DW, et al. Eighty-four hours of sustained operations alter thermoregulation during cold exposure. *Med Sci Sports Exerc*. 2003;35(1):175–181.
81. Fox GR, Hayward JS, Hobson GN. Effect of alcohol on thermal balance of man in cold water. *Can J Physiol Pharmacol*. 1979;57(8):860–865.
82. Centers for Disease Control and Prevention (CDC). Hypothermia-related deaths, United States, 2003–2004. *MMWR Morb Mortal Wkly Rep*. 2005;54(7):173–175.
83. Glickman-Weiss EL, Nelson AG, Hearon CM, Goss FL, Robertson RJ, Cassinelli DA. Effects of body morphology and mass on thermal responses to cold water: revisited. *Eur J Appl Physiol Occup Physiol*. 1993;66(4):299–303.
84. Glickman-Weiss EL, Nelson AG, Hearon CM, Prisby R, Caine N. Thermal and metabolic responses of women with high fat versus low fat body composition during exposure to 5 and 27°C for 120 minutes. *Aviat Space Environ Med*. 1999;70(3, pt 1):284–288.
85. Prisby R, Glickman-Weiss EL, Nelson AG, Caine N. Thermal and metabolic responses of high and low fat women to cold water immersion. *Aviat Space Environ Med*. 1999;70(9):887–891.
86. Tikuisis P, Gonzalez RR, Oster RA, Pandolf KB. Role of body fat in the prediction of the metabolic response for immersion in cold water. *Undersea Biomed Res*. 1988;15(2):123–134.
87. Moriya K, Suda T, Hattori M, Yokoyama S. The correlation of thermoregulatory reactions in young men exposure to cold air with their physical fitness. *Jpn J Biometeorol*. 1993;30:77–84.
88. Savourey G, Bittel J. Thermoregulatory changes in the cold induced by physical training in humans. *Eur J Appl Physiol Occup Physiol*. 1998;78(5):379–384.
89. Sugahara M, Taimura A. Relationship between thermoregulation and cold induced vasodilation during cold exposure with regard to maximal oxygen uptake. *Jpn J Phys Fitness Sports Med*. 1996;46(1):101–110.
90. Jacobs I, Romet T, Frim J, Hynes A. Effects of endurance fitness on responses to cold water immersion. *Aviat Space Environ Med*. 1984;55(8):715–720.
91. Bittel JHM, Nonotte-Varly C, Livecchi-Gonnot GH, Savourey GLMJ, Hanniquet AM. Physical fitness and thermoregulatory reactions in a cold environment in men. *J Appl Physiol*. 1988;65(5):1984–1989.
92. McArdle WD, Magel JR, Gergley TJ, Spina RJ, Toner MM. Thermal adjustment to cold-water exposure in resting men and women. *J Appl Physiol*. 1984;56(6):1565–1571.
93. McArdle WD, Magel JR, Spina RJ, Gergley TJ, Toner MM. Thermal adjustment to cold-water exposure in exercising men and women. *J Appl Physiol*. 1984;56(6):1572–1577.
94. Graham TE. Thermal, metabolic and cardiovascular changes in men and women during cold stress. *Med Sci Sports Exerc*. 1998;20(5 suppl):S185–S192.
95. Holmer I. Assessment of cold stress in terms of required clothing insulation-IREQ. *Int J Indust Ergon*. 1988;3(2):159–166.
96. International Organization for Standardization. *Ergonomics of the Thermal Environment: Estimation of the Thermal Insulation and Evaporative Resistance of A Clothing Ensemble*. Geneva, Switzerland: International Organization for Standardization; 1993. ISO 9920.
97. Froese G, Burton AC. Heat losses from the human head. *J Appl Physiol*. 1957;10(2):235–241.
98. Langdeau JB, Boulet LP. Prevalence and mechanisms of development of asthma and airway hyperresponsiveness in athletes. *Sports Med*. 2001;31(8):601–616.
99. Storms WW. Review of exercise-induced asthma. *Med Sci Sports Exerc*. 2003;35(9):1464–1470.
100. Helenius IJ, Tikkanen HO, Haahtela T. Occurrence of exercise induced bronchospasm in elite runners: dependence on atopy and exposure to cold air and pollen. *Br J Sports Med*. 1998;32(2):125–129.
101. Zeitoun M, Wilk B, Matsuzaka A, Knopfli BH, Wilson BA, Bar-Or O. Facial cooling enhances exercise-induced bronchoconstriction in asthmatic children. *Med Sci Sports Exerc*. 2004;36(5):767–771.
102. Wilber RL, Rundell KW, Szmedra L, Jenkinson DM, Im J, Drake SD. Incidence of exercise-induced bronchospasm in Olympic winter sport athletes. *Med Sci Sports Exerc*. 2000;32(4):732–737.
103. Evans TM, Rundell KW, Beck KC, Levine AM, Baumann JM. Cold air inhalation does not affect the severity of EIB after exercise or eucapnic voluntary hyperventilation. *Med Sci Sports Exerc*. 2005;37(4):544–549.
104. Davies CTM, Fohlin L, Thoren C. Temperature regulation in anorexia nervosa [proceedings]. *J Physiol (Lond)*. 1977;268(1):8P–9P.
105. Luck P, Wakeling A. Increased cutaneous vasoreactivity to cold in anorexia nervosa. *Clin Sci (Colch)*. 1981;61(5):559–567.
106. Beard JL, Borel MJ, Derr J. Impaired thermoregulation and thyroid function in iron-deficiency anemia. *Am J Clin Nutr*. 1990;52(5):813–819.
107. Neittaanmaki H. Cold urticaria: clinical findings in 200 patients. *J Am Acad Dermatol*. 1985;13(4):636–644.
108. Epstein SE, Stampfer M, Beiser GD, Goldstein RE, Braunwald E. Effects of a reduction in environmental temperature on the circulatory response to exercise in man: implications concerning angina pectoris. *New Engl J Med*. 1969;280(1):7–11.
109. Neill WA, Duncan DA, Kloster F, Mahler DJ. Response of coronary circulation to cutaneous cold. *Am J Med*. 1974;56(4):471–476.
110. Ducharme MB, Lounsbury DS. Self-rescue swimming in cold water: the latest advice. *J Appl Physiol Nutr Metab*. 2007;32(4):799–807.

111. Young AJ. Homeostatic responses to prolonged cold exposure: human cold acclimatization. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology: Environmental*. Vol. 1. New York, NY: Oxford Press; 1996:419–438.
112. Sloan REG, Keatinge WR. Cooling rates of young people swimming in cold water. *J Appl Physiol*. 1973;35(3):371–375.
113. Smolander J, Bar-Or O, Korhonen O, Ilmarinen J. Thermoregulation during rest and exercise in the cold in pre- and early pubescent boys and young men. *J Appl Physiol*. 1992;72(4):1589–1594.
114. Thompson-Torgerson CS, Holowatz SA, Kenney WL. Altered mechanisms of thermoregulatory vasoconstriction in aged human skin. *Exerc Sports Science Rev*. 2008;36(3):122–127.
115. Holmer I. Strategies for prevention of cold stress in the elderly. *Int J Circumpolar Health*. 2000;59(3–4):267–272.
116. Potkanowicz ES, Caine-Bish N, Otterstetter R, Glickman EL. Age effects on thermal, metabolic, and perceptual responses to acute cold exposure. *Aviat Space Environ Med*. 2003;74(11):1157–1162.
117. Ferrara MS, Buckley WE, Peterson CL. Epidemiology of sport related injuries for athletes with disability. *Athl Ther Today*. 1997;2(1):28–33.
118. Colachis SC III. Hypothermia associated with autonomic dysreflexia after traumatic spinal cord injury. *Am J Phys Med Rehabil*. 2002;81(3):232–235.
119. Aguilera EJ, Glenn AM, Lynch WJ, Molinari CJ Jr, Nguyen CH. Exposure hypothermia and the winter SCI participant. *J Am Paraplegia Soc*. 1987;10(1):14–17.
120. Dykes RW, Saddiki-Traki F, Tremblay N, Boureau F, Morel-Fatio X. Differences in the sensations of cold on the anterior torso of control and spinal-cord-transected individuals. *Behav Neurosci*. 2006;120(2):463–473.
121. Krassioukov A, Wolfe DL, Hsieh JT, Hayes KC, Durham CE. Quantitative sensory testing in patients with incomplete spinal cord injury. *Arch Phys Med Rehabil*. 1999;80(10):1258–1263.
122. Claus-Walker J, Halstead LS. Metabolic and endocrine changes in spinal cord injury, II (section 1): consequences of partial decentralization of the autonomic nervous system. *Arch Phys Med Rehabil*. 1982;63(11):569–575.
123. Sawka MN, Latzka WA, Pandolf KB. Temperature regulation during upper body exercise: able-bodied and spinal cord injured. *Med Sci Sports Exerc*. 1989;21(5 suppl):S132–S140.
124. Nicotra A, Young TM, Asahina M, Mathias CJ. The effect of different physiological stimuli on skin vasomotor reflexes above and below the lesion in human chronic spinal cord injury. *Neurorehabil Neural Repair*. 2005;19(4):325–331.
125. Mizushima T, Tajima F, Okawa H, Umezumi Y, Furusawa K, Ogata H. Cardiovascular and endocrine responses during the cold pressor test in subjects with cervical spinal cord injuries. *Arch Phys Med Rehabil*. 2003;84(1):112–118.
126. Shirado O, Shundo M, Kaneda K, Strax TE. Outdoor winter activities of spinal-cord injured patients: with special reference to outdoor mobility. *Am J Phys Med Rehabil*. 1995;74(6):408–414.
127. Finnerup NB, Gyldensted C, Fuglsang-Frederiksen A, Bach FW, Jensen TS. Sensory perception in complete spinal cord injury. *Acta Neurol Scand*. 2004;109(3):194–199.

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