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RELIAS MEDIA

Hypothermia and Frostbite

All emergency providers should be familiar with hypothermia regardless of the climate in which they practice. Hypothermia can occur in a variety of climates, indoors or outdoors, and in patients of all ages regardless of health status. Screening for hypothermia is as simple as taking a patient's temperature, yet the condition often goes unnoticed, especially in the setting of critical patients. The recognition and correction of hypothermia is imperative as part of the resuscitation process.

Accidental hypothermia occurs when the body is unable to balance environmental as well as physiologic factors contributing to heat loss and heat production to maintain normothermia. Even though controlled therapeutic hypothermia can be advantageous in very specific settings, such as cardiac arrest and cerebrovascular accidents, these benefits generally do not apply to accidental hypothermia. Therapeutic hypothermia will not be discussed in this article.

Frostbite, chilblains, trench foot, and cold urticaria are cold-related injuries that may present to any emergency department during any time of year. Although predominantly described in the armed forces, these conditions now are recognized as commonplace in civilians, athletes, and homeless individuals across all age groups. All emergency providers, both in the field and in the hospital, must be aware of these injuries and their treatments to prevent further harm.

Hypothermia

Definitions

Hypothermia is defined as a core body temperature below 35° C (95° F). A rectal thermometer is the least invasive method of obtaining a core body temperature, but esophageal and bladder probes also may be used. Although temporal, tympanic, and oral thermometers are used regularly, they may not give an accurate representation of core temperature.[1] Hypothermia may be classified as mild, moderate, or severe. Mild hypothermia ranges from 32° C to 35° C (90° F to 95° F), moderate hypothermia ranges from 28° C to 32° C (82° F to 90° F), and severe hypothermia is defined by temperatures below 28° C (82° F).[1]

Mechanisms of Thermoregulation

Humans maintain normothermia through a balance between heat production and heat loss directed by the anterior hypothalamus, both through physiological and behavioral responses.[2,3] The ability to preserve a normal body temperature is dependent on both the capacity to sense the need for temperature regulation and to respond to those signals.

Heat is generated mostly through increasing energy metabolism as well as through the shiver response. Conditions that can limit the production of heat include endocrine disorders, trauma, hypothalamic dysfunction, adrenal

EXECUTIVE SUMMARY

- Hypothermia can occur in any environment. It is defined as a core body temperature below 35° C. Initially, as the body temperature falls, shivering can generate heat. Once this fails, the body temperature will continue to fall.
- A cold myocardium can be very irritable, so patients should be handled gently. However, cardiopulmonary resuscitation and intubation may be necessary. Patients in cardiac arrest, whose core temperatures are less than 32° C and whose potassium is less than 12 mmol/L should receive aggressive active rewarming until they are warmed.
- Many cardiac drugs do not work well in cold tissues. During cardiac resuscitation, use only two to three rounds of epinephrine and avoid other medications to prevent a bolus of drug when the blood is heated.
- Frostbite occurs when tissue is frozen. It is difficult to determine the degree of frostbite until the tissue is rewarmed. Hemorrhagic bullae suggest deep tissue injury and should be left intact. The area should not be allowed to refreeze, so any discharge plans should include appropriate shelter.

dysfunction, hypoglycemia, poor nutrition, and extremes of age.[1,4] The shiver response is initiated at *skin* temperatures from 17° C to 20° C and terminates at *core* temperatures below 30° C.[5] The shiver response is dependent on factors such as body mass index (BMI) and body fat percentage, with lower values correlating to a stronger intensity of shiver response.[6] In addition to heat production, the body also can gain heat from the environment.

The body loses heat through radiation, evaporation, conduction, or convection. Radiation is the body's major mechanism of heat loss. The amount of radiation is directly proportional to the difference between the temperatures of the body and the outside environment.[1]

Evaporation occurs as a substance changes from liquid to gas. Evaporative cooling is the second largest mechanism of heat loss, occurring as the body uses energy to convert water into vapor, both from the skin's surface and from the lungs.[7]

Conduction is the exchange of heat between two surfaces in direct contact with each other, such as between the skin and the air. Because of the higher conductivity of water compared to gases in the air, a patient in wet clothing loses heat much faster than a patient in dry clothing. Complete water submersion produces up to 25 times higher rates of heat loss when compared to air.

The final mechanism of heat loss is convection, or the heat interchange caused by the movement of liquids or gases over a surface.[1] Because of the high potential for convective heat loss during transport as air flows over the patients' bodies when they are moved, patients should be wrapped in tarps or blankets prior to moving them.

Epidemiology and Etiology

In the United States, an estimated three to five out of every 1 million people per year die as a direct result of hypothermia.[8,9] The incidence of hypothermia is highest in adults between 30 and 49 years of age, with men affected 10 times more often than women.[1] However, the mortality rate of hypothermia in the elderly is substantially higher than it is in younger patients.[9,10] Those with lower body fat percentages, lower BMI, and lower muscle mass are more susceptible to hypothermia because of their reduced capability to produce and retain body heat.[10] Because of an inability to sense the need for shelter and/or the inability to achieve such shelter, in addition to a higher incidence of comorbidities and polypharmacy, people at higher risk for environmental hypothermia include those with: extremes of age, mental disability, homelessness, psychiatric conditions, and drug or alcohol use. Ethanol is a vasodilator that blunts the shiver response and produces anesthesia to the cold, thus altering the behavioral response to cold exposure.[1]

Predisposing medical conditions for hypothermia include poor nutrition, hypoglycemia, adrenal insufficiency, burns, sepsis, shock, diabetes, hypothyroidism, cerebrovascular accidents, and some neoplasms.[1,11] Burns disrupt the body's protective outer layer, resulting in impaired insulation, vasodilation, and increased heat loss due to radiation; evaporation of insensible fluid losses; and convection during transfer.[12] Sepsis generates an immune response requiring the use of energy, which is released as heat. Hypothermia in sepsis is associated with a delay in diagnosis and administration of antibiotics, leading to increased length of stay, increased rates of admission to intensive care units, and increased mortality compared to sepsis presenting with fever.[13] Hypothermia is seen in some patients with neurologic dysfunction, such as neurosarcoidosis or spinal cord injury.[14,15]

In a 2007 study, researchers identified cases of hypothermia related to the use of antipsychotic medications.[11] Dopamine reduces body temperature while 5-hydroxytryptamine-2 (5-HT2) elevates body temperature. Atypical antipsychotics, in particular, antagonize both of these receptors but have a higher affinity for antagonism at the 5-HT2 receptor, producing a net decrease in body temperature.[16] Because thermoregulation is controlled by the hypothalamus, medications that affect the hypothalamus can cause hypothermia even when taken at therapeutic doses. Several case reports have implicated both typical and atypical antipsychotics as well as antiepileptics and benzodiazepines in the development of hypothermia.[17,18,19]

In trauma patients, hypothermia correlates with high rates of morbidity and mortality because of its effects on coagulation, drug metabolism, and even monitoring capability. The combination of hypothermia with coagulopathy and acidosis in the setting of trauma has such a poor prognosis that the combination is referred to collectively as the "lethal triad," with each factor compounding the others.[20] The mortality rate is significantly higher among trauma patients with hypothermia

compared to patients with the same body temperature from environmental hypothermia alone.[21]

Hypothermia-related deaths occur over a broad range of temperatures. Some research has shown that regions with warmer, more variable climates and rapid temperature shifts actually have a higher rate of cold-related deaths than areas with consistently colder climates, possibly because of inadequate preparedness.[22] Although hypothermia is more common in those exposed to the elements and without appropriate clothing or shelter, hypothermia-related deaths also occur indoors.[20]

Pathophysiology

Hypothermia affects nearly all major body systems. Its most profound effects occur on the central nervous system, the respiratory system, the cardiovascular system, the renal system, and the coagulation cascade. All of these effects can be reversible once the patient is warmed to normal body temperatures, typically without lasting sequelae.

Patients shiver to generate heat to counteract the cold. They become tachycardic, tachypneic, and hypertensive, but maintain a normal level of consciousness.[23] Mild hypothermia sets in when compensation mechanisms fail and normothermia no longer can be maintained. Although electroencephalogram changes develop at core temperatures below 34° C, clinically evident alteration of mental status occurs below 32° C as moderate hypothermia ensues.[1] As their core temperature continues to fall, patients become confused, lethargic, and difficult to arouse, with slurred speech and slow mentation.[23] The "umbles" of hypothermia — stumbles, mumbles, fumbles, and grumbles — help identify patients in the early stages of hypothermia. Hyperreflexia also develops around 32° C. Heart rate and blood pressure begin to decrease. Shivering may cease around 30° C when autoregulation of cerebral blood flow becomes nonfunctional and the hypothalamic control of vasoconstriction is lost. This may lead to paradoxical undressing as the patient begins to feel warm because of inappropriate vasodilation, further contributing to the development of severe hypothermia as the core temperature falls below 28° C.[1] Severely hypothermic patients may lose consciousness. Areflexia develops at temperatures below 26° C. Eventually, vital signs will be lost as well.

Hypothermia has a biphasic effect on the respiratory system. Early tachypnea slows as hypothermia worsens, resulting in increased retention of carbon dioxide and respiratory acidosis. At 33° C, the respiratory epithelium undergoes a decrease in mucociliary functioning and the cough reflex is suppressed, resulting in an increased risk of aspiration and pneumonia. Apnea may occur at temperatures below 24° C.[1]

The cardiovascular response to hypothermia also differs based on severity. Initially, mild hypothermia causes a sympathetic response, leading to tachycardia and vasoconstriction. A reduction in mean arterial pressures, cardiac contractility, and cardiac output occurs around 32° C, resulting in hypotension and profound bradycardia.[1] As the core temperature continues to fall and the patient enters severe hypothermia, sinus bradycardia progresses to atrial fibrillation with a slow ventricular response, which may develop into ventricular fibrillation. Cardiac arrest is common in severe hypothermia, as the potential for deadly arrhythmia increases.[23] At temperatures below 20° C, asystole ensues.[1]

Of note, an elevation of the junction between the termination of the QRS complex and the beginning of the ST segment known as the Osborn wave or J wave may be seen on electrocardiogram (ECG) at temperatures below 33° C. This becomes more apparent as hypothermia worsens. Although quite common in moderate to severe hypothermia, J point elevation is not pathognomonic and may be seen in normothermic patients with hypercalcemia, Brugada syndrome, early repolarization syndrome, or brain injury.[24,25]

Peripheral vasoconstriction leads to increased renal blood flow and decreased release of antidiuretic hormone (ADH) and causes cold diuresis. This decrease in ADH leads to decreased reabsorption of water back into circulation and causes the water to be excreted in the urine. Furthermore, as hypothermia worsens and begins to affect the central nervous system, less ADH is produced by the hypothalamus, thereby causing even more profound diuresis.[1]

The efficacy of the coagulation cascade is directly proportional to the enzymatic ability of the coagulation factors. Drastic decreases in enzymatic function are seen at temperatures below 34° C, resulting in prolonged prothrombin and partial thromboplastin times. The level of coagulation deficiency often is underestimated or unrecognized when blood from hypothermic patients is analyzed because blood typically is warmed to a standard 37° C before laboratory testing. In addition, platelet function and count deteriorate at cold temperatures. Platelets become sequestered in the portal venous system, resulting in platelet counts as low as 15,000/mm^3. The platelet count often does not normalize until at least one hour after rewarming. Because of hypothermia's effects on the hypothalamus and the kidneys, the inappropriate increase in diuresis and fluid loss further causes hemoconcentration, leading to increased blood viscosity.[1]

Prehospital Management

The management of hypothermia depends on the patient's cardiovascular status as well as the degree of hypothermia. Prehospital management of hypothermia involves appropriate resuscitation, prevention of further heat loss, and transport to the proper facility. To prevent further heat loss, patients should be dried and wrapped in field wraps, tarps, or blankets. For prolonged evacuations, "hypowraps" with both insulative and protective layers can be used.

Patients with mild hypothermia (32-35° C) and who have a normal level of consciousness without any signs of trauma, significant comorbidities, or suspected secondary hypothermia may be treated in the field and do not necessarily require transport. They should be dried and protected from further heat loss, and their shivering should be supported with food and hydration. Some gentle active rewarming can be done with warm blankets, heat packs, and hot water bottles. Patients with moderate (28-32° C) or severe hypothermia (< 28° C), signs of trauma, comorbidities, suspected secondary hypothermia,

or who fail to improve with field treatments should be transported to a hospital. They should be handled gently, dried, and wrapped with an insulation and vapor layer for transport. Patients should be transported preferentially to a hospital with extracorporeal membrane oxygenation (ECMO) capability if they have altered mental status with hypotension, a ventricular arrhythmia, or a core temperature of less than 28° C.[23]

In the absence of vital signs, begin cardiac resuscitation (see below). Transport the patient to the nearest hospital if cardiac arrest appears to have occurred prior to the onset of hypothermia. Aggressive rewarming in the prehospital setting is controversial, but most sources recommend active external rewarming if resources such as heating pads or warm blankets are available. If primary environmental hypothermia is the suspected cause of cardiac arrest, the core temperature is less than 32° C, and the serum potassium is known to be less than 12 mmol/L, transfer the patient to a hospital with ECMO capability. If the core temperature is greater than 32° C or the serum potassium is greater than 12 mmol/L without the presence of vital signs, consider cessation of resuscitative efforts.[23]

Hospital Arrival

On arrival at the hospital, repeat the primary survey and begin or continue cardiac resuscitation. In general, the cold myocardium is an irritable myocardium. Despite myocardial irritability, treatment of unstable patients requiring intubation or cardiopulmonary resuscitation (CPR) takes precedence over gentle handling. Treatment is determined based on the cardiac rhythm. In patients with severe hypothermia, the American Heart Association recommends delivery of only up to three doses of epinephrine and one attempt at defibrillation if indicated per Advanced Cardiovascular Life Support (ACLS) standards. If unsuccessful, focus efforts on rewarming the patient. Because cardiac medications may not be effective at core temperatures below 28° C, withhold other drugs until the patient has been warmed to prevent an accidental bolus of these medications once they are no longer inactivated by the cold temperatures.[1] Similarly, ventricular fibrillation often is resistant to treatment until after rewarming. Continue resuscitative efforts until the core temperature reaches at least 32° C.[23] As the saying goes, "they're not dead until they're warm and dead." Prioritize rewarming over correction of arrhythmias.

Handle stable patients with great care. Arrhythmias such as atrial fibrillation and sinus bradycardia are common. Aim efforts toward warming the patient rather than treating the heart rhythm. Order bloodwork and diagnostic testing as clinically indicated. All hypothermic patients should have a blood glucose level measured. Obtain an ECG and a chest X-ray in obtunded patients to evaluate arrhythmia and aspiration, respectively. In cases of suspected secondary hypothermia, order laboratory and imaging tests to elicit the cause of hypothermia, such as metabolic derangement, sepsis, hypothyroidism, or intoxication. Appropriate tests may include complete blood count (CBC), complete metabolic panel (CMP), lactate, blood culture, thyroid function panel, ethanol level, acetaminophen level, salicylate level, or urine toxicology screen.[23] Trauma patients require type and screen and coagulation panel, although the coagulation panel may be inaccurate, as previously discussed.

Rewarming

Treatment of hypothermia is a spectrum of escalating therapies ranging from passive external warming to active external warming to active internal warming. Passive modalities of external rewarming include removing the patient from the cold environment, removing wet clothing, and covering the patient, including his or her head. In a patient with intact hypothalamic and endocrine functions, passive external warming alone can increase body temperature by 0.5-2° C per hour. However, if the shivering reflex is not intact or if passive external rewarming is unsuccessful, escalate to active external warming. Active external rewarming involves application of heat directly to the skin and includes the use of warm blankets, the application of heating pads to the axilla and groin, and the use of forced-air warming blankets. These methods can increase core temperature by 0.8-2.5° C per hour.[1]

Any patient with hypotension, cardiac arrest, or ventricular arrhythmia requires active internal rewarming.[23] Base treatment primarily on hemodynamic status rather than temperature, but note that the core body temperature in these patients tends to be below 32° C. While the use of warmed intravenous fluids is emphasized strongly and still is recommended, overall, warmed fluids are ineffective in raising the temperature of a patient who does not require aggressive fluid resuscitation. This is because of the small difference in temperature as well as the large mass difference between the human body and the infused fluids. For example, infusing one liter of normal saline warmed to 40° C into a 70-kilogram patient with a body temperature of 32° C would increase the core temperature by only 0.14° C.[1] Use warmed intravenous fluids in conjunction with other warming methods whenever possible. In emergency departments in which warmed intravenous fluids are not readily available, room temperature bags of normal saline may be placed in a regular microwave oven for 2.5 minutes to achieve a temperature of approximately 40° C. Use this method only in emergency situations because microwave ovens vary and the fluid is not heated uniformly, necessitating mixing the contents thoroughly before administration. It is important to remember that fluids containing dextrose cannot be heated in a microwave because of the risk of caramelization.[1] Provide warm, humidified air to reduce the loss of heat from the lungs.

Active core rewarming includes warm intracavitary lavage.[26] Closed thoracic lavage involves placing one thoracostomy tube on both sides of the chest wall in the second or third intercostal space in the midclavicular line for instillation of warmed fluids. A second thoracostomy tube is placed on both sides of the chest wall in the fourth, fifth, or sixth intercostal space in the posterior axillary line for fluid drainage. This method warms the myocardium and must include intubation of the patient. Peritoneal, bladder,

and gastric lavage are other methods of intracavitary lavage that can increase core temperature by 1-3° C per hour.[1]

The fastest and most effective active internal warming techniques include hemodialysis, continuous arteriovenous rewarming (CAVR), ECMO, and cardiopulmonary bypass. Hemodialysis usually can be initiated quickly and can raise the core temperature by 2-3° C per hour. Compared to other methods of active internal rewarming, hemodialysis is more widely available, more cost effective, and less invasive, and can correct electrolyte abnormalities as well as acidosis.[27] However, hemodialysis requires an adequate blood pressure for use and cannot be instituted by emergency department staff alone. CAVR raises core temperature by 3-4° C per hour. The advantages of CAVR are that it does not require systemic anticoagulation and can be started in the emergency department by appropriately trained staff. CAVR involves cannulation of the femoral artery and the contralateral femoral vein. Specialized tubing is primed with the patient's arterial blood and run through a fluid warmer before being reconnected to the femoral vein catheter.[28] However, because this system is driven by the patient's blood pressure, a systolic pressure of at least 60 mmHg is required for this method to be effective.[1,28] Therefore, neither hemodialysis nor CAVR can be used for patients in cardiac arrest.

For patients presenting in hypothermic cardiac arrest, ECMO and cardiopulmonary bypass are viable options. ECMO can raise the core body temperature by 3-4° C per hour. Cardiopulmonary bypass remains the fastest mechanism of increasing core body temperature and can raise the temperature by 7-10° C per hour.[1,29,30] However, not all hospitals have the capability to institute these measures, so transfer to a higher acuity center may be required. ECMO and cardiopulmonary bypass have the advantage of providing complete hemodynamic support, quickly resolving any end organ damage due to ischemia during cardiac arrest. Since ECMO typically involves cannulation of the femoral or internal jugular veins, emergency providers accelerate the process when they place central venous catheters.[29] With the vessel already accessed, a larger ECMO cannula can be placed in the vessel quickly. In some large centers, emergency physicians can upsize the cannula in preparation for ECMO. Cardiopulmonary bypass requires more complicated cannulation and generally is performed by a cardiac surgeon.[30] In the proper setting, candidates for cardiopulmonary bypass may undergo thoracotomy in the emergency department with mediastinal irrigation and open cardiac massage while arrangements are being made.[29,30] Both ECMO and cardiopulmonary bypass require anticoagulation, which represents an increased risk of bleeding. These risks must be weighed against the potential benefits before deciding to pursue one of these more invasive methods.

Additional Considerations

The foremost concern in hypothermic patients is rewarming to appropriate temperatures. The root cause of the hypothermia may include hypoglycemia, hypothyroidism, sepsis, trauma, cerebrovascular accident, myocardial infarction, or alcohol or drug intoxication. Because the history is likely to be extremely limited, perform a thorough physical exam. Patients brought in for hypothermia are likely to have other cold-related injuries.

Disposition

Admit patients who require hemodialysis, CAVR, ECMO, or cardiopulmonary bypass to the intensive care unit. Admit to the hospital wards any patient with hypothermia resulting in confusion, tachypnea or bradypnea, hypertension or hypotension, hyperactive or diminished reflexes, or cardiac dysrhythmias. Admit any patient with secondary hypothermia because of a pre-existing medical condition. Discharge otherwise healthy patients with mild hypothermia due to environmental exposure only after determining that they have a place of warmth and shelter where they may return and after ruling out any other cold-related injuries.[23]

Frostbite

Definitions

Frostbite is a localized thermal injury that occurs when tissue freezes and commonly affects distal structures such as the fingers, toes, ears, and nose. Although it is more common in colder climates, frostbite also occurs from prolonged use of easily accessible household items such as ice packs. As with burns, frostbite generally is divided into four categories based on the extent of injury: first degree, second degree, third degree, and fourth degree. First-degree frostbite is a partial-thickness injury causing erythema and edema. First-degree frostbite lacks skin blistering and does not result in loss of tissue. Second-degree frostbite is a full-thickness injury that causes numbness followed by throbbing as well as the formation of clear blisters. Healing usually occurs within months, but permanent damage to superficial nerves may be sustained. Third-degree frostbite involves skin and deeper subcutaneous tissues, resulting in the formation of hemorrhagic blisters as well as skin necrosis with permanent nerve damage. Fourth-degree frostbite extends into the muscle, tendon, or bone, and results in the formation of a dry, black, mummified eschar and tissue necrosis. Over time, the delineation between viable and nonviable tissue will become apparent and autoamputation may result.[23] Note that most of the characteristic features that separate the degrees of frostbite will not be apparent until after rewarming. Because distinguishing the degree of injury can be difficult upon initial presentation, sometimes frostbite is classified more simply into the two categories of superficial (first or second degree) or deep (third or fourth degree).[31] This two-tier classification system may be more appropriate in early frostbite injury. (*See Table 1.*)

Epidemiology and Etiology

Frostbite carries a high degree of morbidity, with severe frostbite frequently resulting in loss of digits or limbs. Historically, frostbite has been a disease of war. Now, frostbite is seen more commonly among civilians who engage in recreational exposure to the elements, such as mountaineering or

winter sports. Outdoor temperature, humidity level, degree of wind chill, and duration of exposure all contribute to the overall risk of cold injury.[32] A review of medical events and traumas reported to medical facilities on the Alaskan mountain Denali over a 20-year period found that frostbite was the most common individual diagnosis, accounting for 18.1% of visits.[31] A review of the first 10 years at a Mount Everest base camp medical clinic revealed that 27.5% of all traumatic dermatologic complaints were attributed to frostbite.[33]

The following factors increase the risk for frostbite injuries: extremes of age, mental disabilities, homelessness, psychiatric conditions, and drug or alcohol intoxication. Peripheral vascular disease, diabetes mellitus, and Raynaud's disease particularly increase frostbite risk, as do some medications such as beta-blockers.[31,32,34] Similar to hypothermia, frostbite occurs most commonly in men and in adults 30-49 years of age, possibly because of increased workplace exposure and psychosocial factors.[34] Occupational exposures that contribute to frostbite risk include access to refrigerants or pressurized liquid chemicals, such as ammonia or carbon dioxide. However, frostbite also can occur from inappropriate use of commercial household products, such as ice packs and fire extinguishers.[34,35] Given the common use of the rest, ice, compression, elevation (RICE) protocol for musculoskeletal injuries, physicians should counsel patients on the proper use of cold packs to prevent iatrogenic cold injuries.

Most incidences of frostbite can be avoided with proper planning and precautions, including wearing appropriate garments and being aware of frostbite signs and symptoms. Wearing well-fitting clothing and footwear decreases the incidence of frostbite. To retain the optimal amount of body heat, clothing and footwear should be form-fitted and moisture-wicking without being too constricting or creating a moist environment. Adding extra layers of clothing for insulation can be counterproductive and can contribute to the development of frostbite if blood flow is restricted.[32]

Insulation of the core to decrease central heat loss can help prevent frostbite by decreasing the severity of peripheral vasoconstriction.

Table 1. Two- and Four-Tier Classification of Frostbite According to Clinical Manifestations

Two-Tier Category and Clinical Manifestations	Four-Tier Category
Superficial	**First or second degree**
Partial-thickness skin freezing, erythema and hyperemia, mild edema, no blisters or necrosis	First degree
Full-thickness skin freezing, erythema, substantial edema, superficial blisters containing clear or milky fluid	Second degree
Deep	**Third or fourth degree**
Skin and subcutaneous tissue freezing, blue or black appearance, substantial edema, hemorrhagic blisters with some necrosis	Third degree
Freezing extending through subcutaneous tissue into muscle, tendon, and bone; deep red and mottled appearance with eventual gangrene; minimal edema; extensive necrosis	Fourth degree

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Table 2. Radiographic Stages of Frostbite Injuries

Stage and Time Frame	Radiographic Findings
Early: immediate to weeks after injury	May be normal, depending on injury severity Soft-tissue swelling Tissue atrophy and distortion in severely affected areas Subcutaneous emphysema No bone or joint changes
Intermediate: weeks to months after injury	Bone demineralization Periostitis
Late: months to years after injury	Acroosteolysis Sclerosis at ends of involved bone Asymmetric early osteoarthrosis of the affected limb Small periarticular erosions In children, epiphyseal fragmentation and/or premature fusion with resulting deformities

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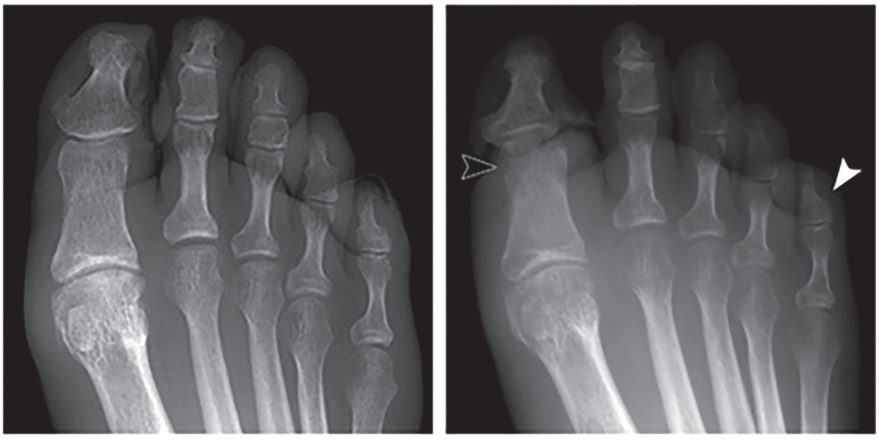
Pathophysiology

The pathologic changes that result in frostbite occur in multiple interconnected phases. The initial phase is the prefreeze phase in which decreased temperature causes local vasoconstriction and tissue ischemia. Alteration of sensation, both paresthesia and hyperesthesia, occurs during this phase.[34] The second phase is the freeze-thaw phase in which direct cellular injury occurs as the result of intracellular ice crystal formation as the tissues are exposed to temperatures below their freezing point. This can cause cell dehydration and contraction, electrolyte abnormalities, and

Figure 1. Right Toe Frostbite in a 55-Year-Old Man



(a) Initial dorsoplantar radiograph shows soft-tissue irregularities of the first, second, and fifth toes without underlying bone changes. (b) Dorsoplantar radiograph obtained 4 months later at follow-up shows progression of the first and second toe soft-tissue defects and a new periarticular erosion of the distal first proximal phalanx (black arrowhead). Note the interval healing of the soft tissue of the fifth toe, with new subtle osteopenia of the fifth distal phalanx (white arrowhead).

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destabilization of the cell membrane.[31,34] The body responds to the freezing of the extremities with alternating cycles of vasoconstriction and vasodilation, also known as the hunting reaction. Rewarming during cycles of vasodilation increases the level of inflammatory mediators, compounding the damage to tissues and resulting in a prothrombotic, proinflammatory microenvironment that promotes indirect cellular injury. The next phase is the vascular stasis phase in which vasoconstriction prevails, damaging tissues through both hypoxia and acidosis. The resultant endothelial damage, proinflammatory mediators, edema, and thrombosis lead to the final late ischemic phase.[34]

Management

Early recognition is paramount to minimize permanent damage from frostbite. The earliest symptom may be paresthesia alone. Erythema, edema, and blister formation do not occur until after rewarming. Assess any patient presenting with environmental hypothermia for frostbite.[36] In reference to winter sports, the presentation of any team member with a cold injury should prompt evaluation of all team members. The first step in the treatment of frostbite is removal from the cold environment. Never directly apply dry heat or rub the skin, which can damage the tissues.

Field management of frostbite differs slightly from hospital management. Generally, avoid rewarming until it can be guaranteed that the tissue will not refreeze. When possible, patients with frostbitten feet or toes should not be allowed to walk until they receive definitive care because walking can result in partial rewarming, swelling, and damage to the tissues.[31,32] However, when walking is necessary because of the nature of the evacuation, several sources recommend leaving the shoes in place and ambulating on "frozen" feet. Rewarming causes frostbitten limbs to swell.[31,37] Once swelling sets in, replacing the shoes may be impossible. Aspirate *clear* blisters only if they are at high risk of self-rupturing during extraction and transportation. Do not aspirate or debride hemorrhagic blisters in the field. Prior to hospital arrival, protect frostbitten limbs with loose, bulky dressings that allow for substantial swelling.[37]

In the hospital, once the patient is stable and warmed, evaluate and attend to frostbitten limbs. Note in the history when the insult occurred, the mechanism of injury, any thaw-refreeze events that may have occurred, and any treatment prior to hospital arrival. Give a tetanus booster vaccine if necessary. Clinical photography at admission and throughout treatment aids in documentation and reduces the need for repeated dressing removal. Because of the swelling that occurs following rewarming, remove all potentially constricting items, such as jewelry, clothing, and circumferential dressings.[31]

Rapid rewarming is paramount to achieving the best tissue viability because slow thawing increases anoxic reperfusion injury.[37] Use warm running or circulating water set to 37-39° C and containing an antibacterial agent such as iodine or chlorhexidine.[31,38,39] Continue rewarming until the tissue achieves a red or purple appearance, typically half an hour.[31] Administer strong analgesia for comfort. Start all patients without major contraindications on a nonsteroidal anti-inflammatory medication because systemic antiprostaglandin activity can mediate inflammatory damage.[38] Oral ibuprofen 12 mg/kg twice daily is a sufficient dose to provide these anti-inflammatory effects, but dose up to 2,400 mg/day for better analgesia.[31] Parenteral opiates may be required for further pain control.

Hospital management of blisters is a controversial topic with minimal available research and is largely at the discretion of the provider. Clear blisters contain prostaglandins and thromboxanes that may cause further damage to frostbitten tissues. Aspiration of these fluids may promote healing. Some sources recommend unroofing clear blisters completely while others endorse needle aspiration. Conversely, most sources agree that hemorrhagic blisters should be left intact because they may cause desiccation and further damage if debrided or aspirated.[38,39]

Once rewarming is completed, apply topical aloe vera cream or gel to the affected areas before loosely wrapping them in a protective dressing and elevating the extremity. Aloe vera is an antiprostaglandin that helps reduce inflammation and subsequent tissue damage. Because prophylactic systemic

antibiotic therapy is controversial, only institute antibiotic therapy in cases of proven or suspected infection as directed by swab or culture results.

All patients with frostbite require a high-calorie diet rich in protein to promote optimal healing. The healing of frostbite injuries is a long, dynamic process, and the true extent of tissue damage and possible need for amputation will not be evident until days to weeks after rewarming.[39]

Compartment syndrome can occur. Because of the intrinsic nature of frostbite injury, the hallmark signs and symptoms of compartment syndrome, including pain out of proportion, paresthesias, pallor, paralysis, and poikilothermia, may be extremely difficult to assess. Pulselessness is a late sign. For this reason, maintain a high suspicion for compartment syndrome, especially in areas of circumferential edema. The definitive treatment is fasciotomy.[39]

Diagnostic Studies

Blood work is not required in the acute management of frostbite except in the case of suspected infection. Order diagnostic imaging studies to guide management.

X-rays taken in the acute phase of injury usually will be normal or show only soft-tissue swelling, but they may show fractures or radiopaque foreign bodies. In the months following cold injury, radiography can show bone involvement in cases of fourth-degree frostbite. (*See Table 2 and Figure 1.*)

The preferred imaging study for patients presenting within 24 hours of deep frostbite injury with suspicion of vascular compromise is digital subtraction angiography (DSA). DSA visualizes vessel patency through cannulation and injection of iodinated contrast.[40] It recognizes potential areas that could benefit from thrombolytic therapy and can be used for surgical planning.[41] Most hospitals with interventional radiology should have DSA capability. Magnetic resonance angiography (MRA) is a noninvasive alternative to DSA. However, unlike DSA, MRA is diagnostic only and has no therapeutic potential.[31,34]

Triple phase bone scintigraphy accurately predicts the level of amputation in 84% of cases only two days after frostbite injury, weeks before the level of demarcation is clinically apparent.[31,34] Most hospitals with nuclear medicine capability are able to perform bone scans.

Single photon emission computed tomography in conjunction with conventional computed tomography (SPECT/CT) is a newer diagnostic modality that provides information regarding tissue and bone viability with better anatomical precision. This allows for more accurate determination of the viability of the tips of the distal phalanges.[34] See Figures 2 and 3 for frostbite visualized on advanced imaging studies.

With the demarcation of viability clearly identified, patients can undergo earlier surgical intervention, which may lead to decreased risk of infection, shorter hospital stays, and earlier rehabilitation.

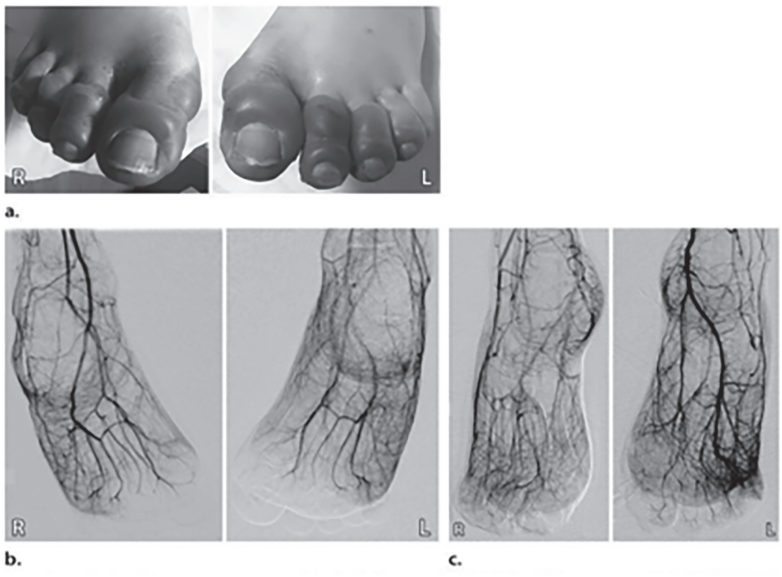
Thrombolytics

The early use of thrombolytics can reverse the microvascular thrombosis and ischemia caused by cold injury, thus reducing tissue loss and improving outcomes. The decision to use thrombolytic therapy in patients without major contraindications is based on identifying areas of hypoperfusion demonstrated on diagnostic imaging such as DSA and bone scintigraphy.[42,43] These decisions are made in conjunction with a surgeon or interventional radiologist.[44] Thrombolytics are given as soon as possible once a patient has been deemed an appropriate candidate. However, the outer range of efficacy has not been determined clearly. Most current literature recommends the use of thrombolytics within 24 hours of cold exposure. (*See diagram figure on the Rapid Reference Card insert.*)

Additional Aspects

Several complications may result following even optimal healing of a frostbite injury, leading to a high rate of morbidity. These complications include functional loss, cold sensitivity, chronic pain, chronic ulceration, and arthritis. Approximately half of patients with significant frostbite injury will have either cold hypersensitivity or decreased

Figure 2. Third-Degree Frostbite of the Toes in a 22-Year-Old Man



(a) Dorsal photographs show bilateral diffuse discoloration of the toes and hemorrhagic blisters. (b) DSA images obtained at presentation show absent perfusion beyond the distal interphalangeal joint bilaterally. (c) DSA images obtained after 24 hours of continuous intra-arterial tPA infusion via the popliteal arteries show near-complete resolution. The patient made a full recovery without tissue loss or surgery.

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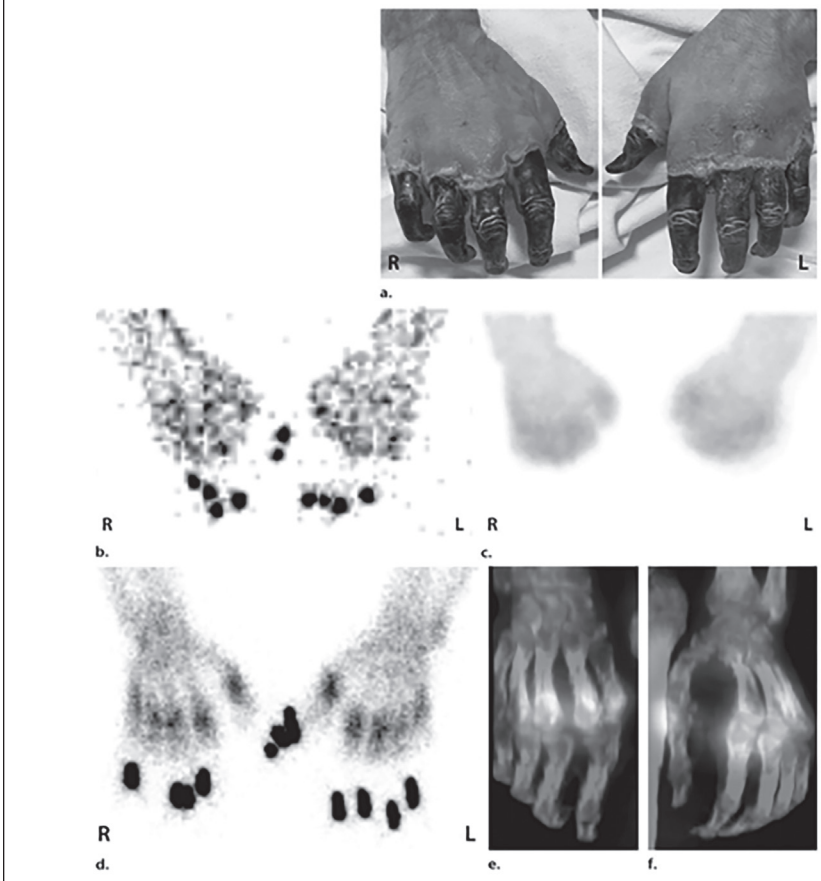
Figure 3. Fourth-Degree Frostbite of the Hands in a 55-Year-Old Man



(a) Dorsal photographs show gangrene involving all digits of both hands. (b-d) Multiphase ^{99m}Tc-MDP bone scintigraphic images of the hands show (b) absent tracer uptake throughout the bilateral phalanges in the palmar blood flow phase at 80 seconds (with markers delineating the distal aspects of the fingers), (c) palmar soft-tissue phase (without markers), and (d) 4-hour delayed phase (with markers). Note the increased tracer uptake at the metacarpophalangeal joints proximal to the level of necrosis on the delayed phase image. (e-f) Fused SPECT/CT images of the (e) right and (f) left hands show absent tracer uptake distal to the metacarpophalangeal joints bilaterally, with the exception of increased uptake in the left second and third proximal phalanges. SPECT/CT enables delineation of the precise level of tracer cutoff. The patient ultimately underwent amputation of the bilateral thumbs at the end of the mid proximal phalanges and total amputation of the other fingers.

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sensitivity to touch. Occasional electrical shock-like sensations also have been reported.[45] Chronic neuropathic pain is a common sequela following cold injury. Chronic ulceration may result from subsequently diminished vascularization, poor wound healing, and the creation of new foci of pressure following amputation. Malignant transformation is more prevalent in previously frostbitten skin than in normal, uninjured skin. Finally, arthritis is a well-documented complication of frostbite, likely due to localized osteoporosis stemming from vascular insult.[31]

Disposition

Patients with only localized areas of superficial frostbite can be discharged with strict return precautions and close outpatient follow-up. Patients who develop an expanding area of injury, circumferential injury, intractable pain, or change in sensation need to return to the emergency department immediately. Provide proper wound care instructions, including application of topical aloe vera every six hours.

Patients with deep frostbite injuries should be admitted for further evaluation and wound care at centers with general surgery and advanced imaging capability.[23,44] Patients with suspected vascular injury should be admitted or transferred to a center with interventional radiology and/or vascular surgery.

Nonfreezing Cold Injuries

Frostnip

Frostnip is the least severe of the cold-related injuries. Frostnip causes temporary skin pallor and numbness but does not result in permanent injury or loss of tissue. Unlike with frostbite, the tissue has not yet frozen in a frostnip injury, and symptoms are easily reversible with removal from the cold environment alone.[34]

Trench Foot

Trench foot, also known as immersion foot, occurs following repeated exposure to a wet environment in nonfreezing temperatures, typically 0-15° C, and is characterized by skin irritation or ulceration and rash formation.[32] The foot may appear pale and mottled with decreased sensation that does not resolve rapidly with rewarming. The extremity becomes red, warm, painful, and edematous as perfusion returns over the next hours to days, and blisters may form. Treatment should include drying, rewarming, and preventing re-exposure to cold. Long-term complications include diminished sensation or cold sensitivity.[23] Historically, trench foot occurred in members of the armed forces.[46] Now, the condition occurs in civilian populations, especially in people who are homeless.[47,48]

Chilblains

Chilblains, also known as pernio, are inflammatory lesions caused by acute or chronic exposure to cold, moist, nonfreezing environments. They are thought to be the result of cold-induced vasoconstriction leading to hypoxemia and an inflammatory response.[49] These cutaneous eruptions typically involve the hands, feet, and ears. They may develop as quickly as one to five hours after exposure and may take weeks to resolve.[32] The afflicted area becomes red, edematous, and occasionally cyanotic. Lesions may be macular or papular and are associated with pruritus and burning paresthesias. After rewarming, tender blue nodules

may form and last for days.[23] In addition to drying, warming, and preventing re-exposure to cold, use vasodilators and topical steroids to improve outcomes. Lesions that do not improve with warming may require biopsy.[49]

Cold Urticaria

Cold urticaria is an allergic response to cold exposure resulting in localized or systemic eruption of hives within minutes of cold exposure. Extremely rarely, angioedema or anaphylaxis may occur. Treatment with second-generation antihistamines and removal from the cold environment generally is sufficient for symptom relief.[46] Development of cold urticaria indicates a predisposition to future episodes, although formation of urticaria does not necessarily occur with every exposure to cold.

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CME/CE Questions

1. Which of the following is a risk factor for accidental hypothermia?
   a. Female gender
   b. Living in a consistently cold climate
   c. History of drug or alcohol abuse
   d. Having a high body mass index and body fat percentage
2. Which of the following is true of the J or Osborn wave?
   a. It is diagnostic of hypothermia.
   b. It is seen in all cases of severe hypothermia.
   c. It is caused by cold diuresis.
   d. It is seen with increasing severity of hypothermia.
3. Which of the following is an example of an appropriate method of active external rewarming?
   a. Forced-air warming blankets
   b. Removal of wet clothing
   c. Warm intravenous fluids
   d. Hemodialysis
4. Which of the following is correct regarding management of severely hypothermic patients without a pulse?
   a. Do not intubate or perform cardiopulmonary resuscitation.
   b. Give 1 mg epinephrine intravenously every three to five minutes until rewarmed to 28° C.
   c. Discontinue resuscitative efforts if the patient remains pulseless after the core temperature has reached 28° C.
   d. Attempt defibrillation only once for ventricular fibrillation if the core temperature is less than 28° C.
5. Which type of frostbite is a deep tissue injury characterized by hemorrhagic blisters and skin necrosis resulting in permanent nerve damage?
   a. First degree
   b. Second degree
   c. Third degree
   d. Chilblains
6. Which of the following represents the best modality for rewarming frostbitten limbs?
   a. Warm dry air set to 37-39° C
   b. Forced air warming blanket
   c. Hot running water set to 40-42° C
   d. Warm running water set to 37-39° C
7. Which of the following is correct regarding the use of thrombolytics for frostbite?
   a. Systemic therapy has been proven to have better outcomes than catheter-directed intra-arterial therapy.
   b. Most of the current literature recommends use of thrombolytics within 24 hours of cold exposure.
   c. Concurrent use of vasodilators generally worsens outcome by increasing the levels of inflammatory cytokines.
   d. Thrombolytics may be administered during magnetic resonance angiography.

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Hypothermia and Frostbite

Two- and Four-Tier Classification of Frostbite According to Clinical Manifestations

Two-Tier Category and Clinical Manifestations	Four-Tier Category
Superficial	**First or second degree**
Partial-thickness skin freezing, erythema and hyperemia, mild edema, no blisters or necrosis	First degree
Full-thickness skin freezing, erythema, substantial edema, superficial blisters containing clear or milky fluid	Second degree
Deep	**Third or fourth degree**
Skin and subcutaneous tissue freezing, blue or black appearance, substantial edema, hemorrhagic blisters with some necrosis	Third degree
Freezing extending through subcutaneous tissue into muscle, tendon, and bone; deep red and mottled appearance with eventual gangrene; minimal edema; extensive necrosis	Fourth degree

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Radiographic Stages of Frostbite Injuries

Stage and Time Frame	Radiographic Findings
Early: immediate to weeks after injury	May be normal, depending on injury severity Soft-tissue swelling Tissue atrophy and distortion in severely affected areas Subcutaneous emphysema No bone or joint changes
Intermediate: weeks to months after injury	Bone demineralization Periostitis
Late: months to years after injury	Acroosteolysis Sclerosis at ends of involved bone Asymmetric early osteoarthrosis of the affected limb Small periarticular erosions In children, epiphyseal fragmentation and/or premature fusion with resulting deformities

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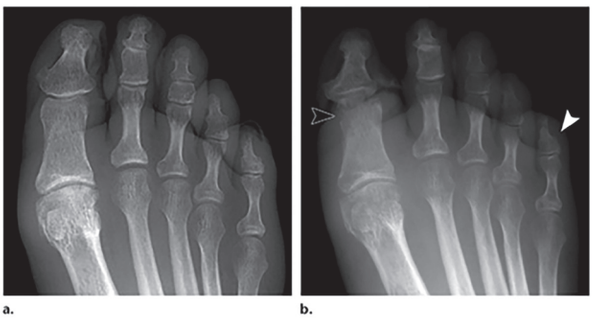
Right Toe Frostbite in a 55-Year-Old Man



a. **b.**

(a) Initial dorsoplantar radiograph shows soft-tissue irregularities of the first, second, and fifth toes without underlying bone changes. (b) Dorsoplantar radiograph obtained 4 months later at follow-up shows progression of the first and second toe soft-tissue defects and a new periarticular erosion of the distal first proximal phalanx (black arrowhead). Note the interval healing of the soft tissue of the fifth toe, with new subtle osteopenia of the fifth distal phalanx (white arrowhead).

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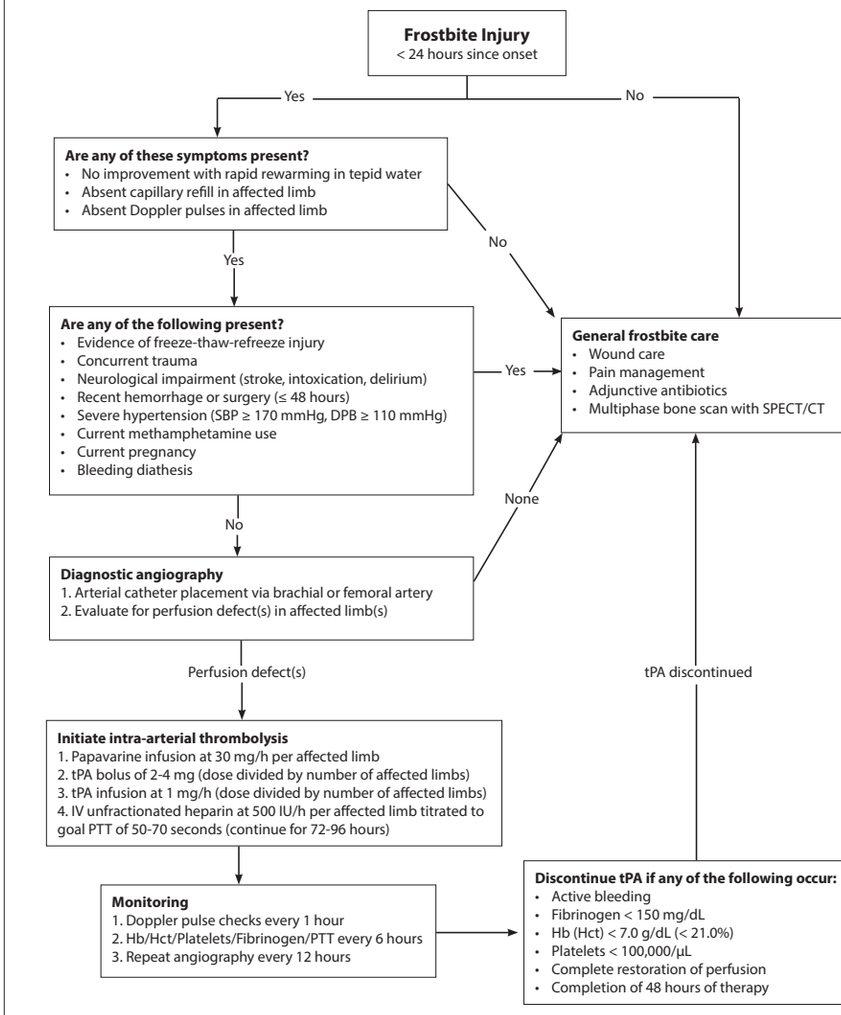
Diagram Shows an Algorithm for Management of Frostbite With Thrombolytic Therapy



Abbreviations: CT = computed tomography; DBP = diastolic blood pressure; Hb = hemoglobin; Hct = hematocrit; IV = intravenous; PTT = partial thromboplastin time; SBP = systolic blood pressure; SPECT = single photon emission computed tomography

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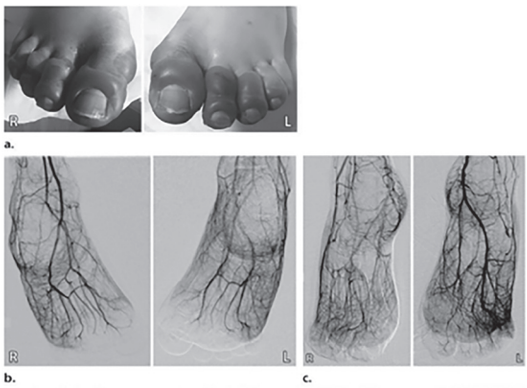
Third-Degree Frostbite of the Toes in a 22-Year-Old Man



(a) Dorsal photographs show bilateral diffuse discoloration of the toes and hemorrhagic blisters. (b) DSA images obtained at presentation show absent perfusion beyond the distal interphalangeal joint bilaterally. (c) DSA images obtained after 24 hours of continuous intra-arterial tPA infusion via the popliteal arteries show near-complete resolution. The patient made a full recovery without tissue loss or surgery.

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