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as necessary. For cases of arterial gas embolism, evidence supports the use of intravenous lidocaine to achieve standard antiarrhythmic plasma levels. Occasionally, very sick divers require intubation, ventilation, and intensive care.

The presentation of sick divers to physicians or hospitals without diving medicine expertise creates a risk of misinterpretation of non specific manifestations and of consequent mistakes in diagnosis and management. Physicians finding themselves in this situation are strongly advised to expeditiously contact the 24-h worldwide diving emergency advisory service provided by the Divers Alert Network (DAN) at +1-919-684-9111. Acknowledgment The authors gratefully acknowledge the major contributions of Professor Michael Bennett (1956–2023) to previous editions of this chapter. ■ ■FURTHER READING Boet S et al: Efficacy and safety of hyperbaric oxygen treatment to treat COVID-19 pneumonia: A living systematic review update. Diving Hyperb Med 52:126, 2022. Brouwer R et al: Economic analysis of hyperbaric oxygen therapy for the treatment of ischaemic diabetic foot ulcers. Diving Hyperb Med 54:265, 2024. Chen H-R et al: Application of hyperbaric oxygen therapy in diabetic foot ulcers: A meta-analysis. Int Wound J 21:e14621, 2024. Huang E (ed): Undersea and Hyperbaric Medicine Society Hyperbaric Medicine Indications Manual, 15th ed. North Palm Beach, FL, Best Publishing, 2024. Krzyżak J, Korzeniewski K: Medical assessment of fitness to dive. PART 15 Disorders Associated with Environmental Exposures Parts I and II. Int Marit Health 72:36 and 115, 2021. Lin ZC et al: Hyperbaric oxygen for late radiation tissue injury. Cochrane Database Syst Rev 8:CD005005, 2023. Miller IL et al: Hyperbaric oxygen for lower limb trauma (HOLLT): An international multi-centre randomised clinical trial. Diving Hyperb Med 52:164, 2022. Mitchell SJ: Decompression illness: A comprehensive overview. Div ing Hyperb Med 54:1, 2024. Mitchell SJ et al: Decompression illness. N Eng J Med 386:1254, 2022. Moses RA et al: Patient reported outcome measures following hyper baric oxygen therapy for radiation cystitis: Early results from the mul ticenter registry for hyperbaric oxygen therapy. J Urol 211:765, 2024. Weaver LK et al: Hyperbaric oxygen for acute carbon monoxide poi soning. N Engl J Med 347:1057, 2002. Wendling J et al (eds). Medical Assessment for Work Under Pressure, 2nd ed. London, International Marine Contractors Association, 2024. Whelan HT (ed): Hyperbaric Medicine Practice, 5th ed. North Palm Beach, FL, Best Publishing, 2024. Daniel F. Danzl

Hypothermia and

Peripheral Cold Injuries ■ ■HYPOTHERMIA Accidental hypothermia occurs when there is an unintentional drop in the body’s core temperature below 35°C (95°F). At this temperature, many of the compensatory physiologic mechanisms that conserve heat begin to fail. Primary accidental hypothermia is a result of the direct exposure of a previously healthy individual to the cold. The mortality rate is much higher for patients who develop secondary hypothermia as a complication of a serious systemic disorder or injury.

TABLE 477-1 Risk Factors for Hypothermia Age extremes Elderly Neonates Environmental exposure Occupational Sports-related Inadequate clothing Immersion Toxicologic and pharmacologic Ethanol Anesthetics Antipsychotics Antidepressants Anxiolytics Benzodiazepines Neuromuscular blockers Insufficient fuel Malnutrition Marasmus Kwashiorkor Endocrine-related Diabetes mellitus Hypoglycemia Hypothyroidism Adrenal insufficiency Hypopituitarism Neurologic Cerebrovascular accident Hypothalamic disorders Parkinson’s disease Spinal cord injury Multisystemic Trauma Sepsis Shock Hepatic or renal failure Carcinomatosis Burns and exfoliative dermatologic disorders Immobility or debilitation ■ ■CAUSES Primary accidental hypothermia is geographically and seasonally per vasive. Although most cases occur in the winter months and in colder climates, this condition is surprisingly common in warmer regions as well. Multiple variables render individuals at the extremes of age—both the elderly and neonates—particularly vulnerable to hypothermia (Table 477-1). The elderly have diminished thermal perception and are more susceptible to immobility, malnutrition, and systemic illnesses that interfere with heat generation or conservation. Dementia, psychi atric illness, and socioeconomic factors often compound these prob lems. Neonates have high rates of heat loss because of their increased surface-to-mass ratio and their lack of effective shivering and adaptive behavioral responses. At all ages, malnutrition can contribute to heat loss because of diminished subcutaneous fat and as a result of depleted energy stores used for thermogenesis. Individuals whose occupations or hobbies entail extensive exposure to cold weather are at increased risk for hypothermia. Military history is replete with hypothermic tragedies. Hunters, sailors, skiers, and climbers also are at great risk of exposure, whether it involves injury, changes in weather, or lack of preparedness. Ethanol causes vasodilation (which increases heat loss), reduces thermogenesis and gluconeogenesis, and may impair judgment or lead to obtundation. Some antipsychotics, antidepressants, anxiolytics, benzodiazepines, and other medications reduce centrally mediated vasoconstriction. Many hypothermic patients are admitted to inten sive care because of drug overdose. Anesthetics can block shivering responses; these effects are compounded when patients are not insu lated adequately in the operating or recovery units. Several types of endocrine dysfunction cause hypothermia. Hypo thyroidism—particularly when extreme, as in myxedema coma— reduces the metabolic rate and impairs thermogenesis and behavioral responses. Adrenal insufficiency and hypopituitarism also increase susceptibility to hypothermia. Hypoglycemia, most commonly caused by insulin or oral hypoglycemic agents, is associated with hypothermia, in part because of neuroglycopenic effects on hypothalamic function. Increased osmolality and metabolic derangements associated with uremia, diabetic ketoacidosis, and lactic acidosis can lead to altered hypothalamic thermoregulation. Neurologic injury from trauma, cerebrovascular accident, subarach noid hemorrhage, and a hypothalamic lesion increases susceptibility to hypothermia. Agenesis of the corpus callosum (Shapiro’s syndrome) is one cause of episodic hypothermia. In this syndrome, profuse perspira tion is followed by a rapid fall in temperature. Acute spinal cord injury

disrupts the autonomic pathways that lead to shivering and will prevent cold-induced reflex vasoconstrictive responses. Hypothermia associated with sepsis is a poor prognostic sign. Hepatic failure causes decreased glycogen storage and gluconeogenesis as well as a diminished shivering response. In acute myocardial infarc tion associated with low cardiac output, hypothermia may be reversed after adequate resuscitation. With extensive burns, psoriasis, erythro dermas, and other skin diseases, increased peripheral-blood flow leads to excessive heat loss. ■ ■THERMOREGULATION Heat loss occurs through five mechanisms: radiation (55–65% of heat loss), conduction (10–15% of heat loss, increased in cold water), con vection (increased in the wind), respiration, and evaporation; both of the latter two mechanisms are affected by the ambient temperature and the relative humidity. The preoptic anterior hypothalamus normally orchestrates thermo regulation (Chap. 20). The immediate defense of thermoneutrality is via the autonomic nervous system, whereas delayed control is mediated by the endocrine system. Autonomic nervous system responses include the release of norepinephrine, increased muscle tone, and shivering, leading to thermogenesis and an increase in the basal metabolic rate. Cutaneous cold thermoreception causes direct reflex vasoconstriction to conserve heat. Prolonged exposure to cold also stimulates the thy roid axis, leading to an increased metabolic rate. ■ ■CLINICAL PRESENTATION In most cases of hypothermia, the history of exposure to environmental factors (e.g., prolonged exposure to the outdoors without adequate cloth ing) makes the diagnosis straightforward. In urban settings, however, the presentation is often more subtle, and other disease processes, toxin exposures, or psychiatric diagnoses should be considered. Predicting the core temperature based on the clinical presentation is very difficult. After initial stimulation by hypothermia, there is progressive depression of all organ systems. The timing of the appearance of these clinical manifestations varies widely (Table 477-2). Without knowing the core temperature, it can be difficult to interpret other vital signs. For example, tachycardia disproportionate to the core temperature sug gests secondary hypothermia resulting from hypoglycemia, hypovole mia, or a toxin overdose. Because carbon dioxide production declines progressively, the respiratory rate should be low; persistent hyperven tilation suggests a central nervous system (CNS) lesion or an organic TABLE 477-2 Physiologic Changes Associated with Accidental Hypothermia BODY TEMPERATURE CENTRAL NERVOUS SYSTEM CARDIOVASCULAR RESPIRATORY RENAL AND ENDOCRINE NEUROMUSCULAR SEVERITY Mild 35°C (95°F)–

32.2°C (90°F) Linear depression of cerebral metabolism; amnesia; apathy; dysarthria; impaired judgment; maladaptive behavior Tachycardia, then progressive bradycardia; cardiac cycle prolongation; vasoconstriction; increase in cardiac output and blood pressure Moderate <32.2°C (90°F)–

28°C (82.4°F) EEG abnormalities; progressive depression of level of consciousness; pupillary dilation; paradoxical undressing; hallucinations Progressive decrease in pulse and cardiac output; increased atrial and ventricular arrhythmias; suggestive (J-wave) ECG changes Severe <28°C (<82.4°F) Loss of cerebrovascular autoregulation; decline in cerebral blood flow; coma; loss of ocular reflexes; progressive decrease in EEG abnormalities Progressive decrease in blood pressure, heart rate, and cardiac output; reentrant dysrhythmias; maximal risk of ventricular fibrillation; asystole Abbreviations: ECG, electrocardiogram; EEG, electroencephalogram. Source: From DF Danzl, RS Pozos: Accidental hypothermia. N Engl J Med 331:1756, 1994.

acidosis. A markedly depressed level of consciousness in a patient with mild hypothermia suggests an overdose or CNS dysfunction due to infection or trauma.

Physical examination findings will also be altered by hypothermia. For instance, the assumption that areflexia is solely attributable to hypo thermia can obscure the diagnosis of a spinal cord lesion. Patients with hypothermia may be confused or combative; these symptoms abate more rapidly with rewarming than with chemical or physical restraint. A classic example of maladaptive behavior in patients with hypothermia is paradoxical undressing, which involves the inappropriate removal of clothing in response to a cold stress. The cold-induced ileus and abdominal rectus spasm can mimic or mask the presentation of an acute abdomen (Chap. 16). When a patient in hypothermic cardiac arrest is first discovered, cardiopulmonary resuscitation (CPR) is indicated unless (1) a do-notresuscitate status is verified, (2) obviously lethal injuries are identified, or (3) the depression of a frozen chest wall is not possible. Continuous CPR is normally recommended, and interruptions should be avoided if possible. In the field, when the core temperature is <28°C (82.4°F), intermittent CPR may also be effective. As the resuscitation proceeds, the prognosis is grave if there is evidence of widespread cell lysis, as reflected by potassium levels >12 mmol/L (12 meq/L). Other findings that may preclude continuing resuscitation include a core temperature <10–12°C (<50–54°F), a pH <6.5, and evidence of intravascular thrombosis with a fibrinogen value <0.5 g/L (<50 mg/dL). The decision to terminate resuscitation before rewarming the patient past 33°C (91°F) should be predicated on the type and severity of the precipitants of hypothermia. Survival has occurred with a cardiac arrest time over 7 h. A history of asphyxia, as in an avalanche, with secondary cooling is the most important negative predictor of survival. CHAPTER 477 ■ ■DIAGNOSIS AND STABILIZATION Hypothermia is confirmed by measurement of the core temperature. When feasible, placement of a temperature probe in the lower third of the esophagus is preferable, and continuous monitoring essential. Ancillary rectal or bladder monitoring often lags behind core tempera ture changes. Hypothermia and Peripheral Cold Injuries Pulses may be undetectable in perfusing patients, and the ampli tude of the QRS complex is decreased. Maximal amplification on the monitor is often helpful. Chest compressions, when not indicated, may convert a perfusing rhythm to a nonperfusing one. Bedside Tachypnea, then progressive decrease in respiratory minute volume; declining oxygen consumption; bronchorrhea; bronchospasm Diuresis; increase in catecholamines, adrenal steroids, triiodothyronine, and thyroxine; increase in metabolism with shivering Increased preshivering muscle tone, then fatiguing Hypoventilation: 50% decrease in carbon dioxide production per 8°C (17.6°F) drop in temperature; absence of protective airway reflexes 50% increase in renal blood flow; renal autoregulation intact; impaired insulin action Hyporeflexia; diminishing shivering-induced thermogenesis; rigidity Pulmonic congestion and edema; 75% decrease in oxygen consumption; apnea Decrease in renal blood flow that parallels decrease in cardiac output; extreme oliguria; poikilothermia; 80% decrease in basal metabolism No motion; decreased nerve-conduction velocity; peripheral areflexia; no corneal or oculocephalic reflexes

echocardiography and end-tidal carbon dioxide ($ETCO_2$) monitoring may confirm the perfusion status. If the core temperature is below 30°C (86°F) and there is no perfusion, consider up to three attempts at maximal power. If unsuccessful, rewarm to at least 30°C (86°F) before further attempts. Although cardiac pacing for hypothermic bradydysrhythmias is rarely indicated, the transthoracic technique is preferable. The J or Osborn wave at the junction of the QRS complex and ST segment suggests the diagnosis. Obvious J waves are routinely misdiagnosed by automated

readings as injury current.

Supplemental oxygenation is always warranted, since tissue oxygen ation is affected adversely by the leftward shift of the oxyhemoglobin dissociation curve. Pulse oximetry is often unreliable in patients with vasoconstriction. If protective airway reflexes are absent, gentle endo tracheal intubation should be performed. Adequate preoxygenation will prevent ventricular arrhythmias. Insertion of a gastric tube prevents dilation secondary to decreased bowel motility. Indwelling bladder catheters facilitate monitoring of cold-induced diuresis and can provide an ancillary approach for temperature monitoring. Dehydration is encountered commonly with chronic hypothermia, and most patients benefit from an intravenous or intraosseous crystalloid bolus. Normal saline is preferable to lactated Ringer's solution, as the liver in hypothermic patients inefficiently metabolizes lactate. The placement of a pulmonary artery catheter can cause perforation of the less compliant pulmonary artery. Insertion of a central venous catheter deeply into the cold right atrium should be avoided since this procedure, similar to transvenous pacing, can pre cipitate refractory arrhythmias. Arterial blood gases should not be corrected for temperature (Chap. 58). An uncorrected pH of 7.42 and a PCO_2 of 40 mmHg reflect appropriate alveolar ventilation and acid-base balance at any core temperature. Acid-base imbalances should be corrected gradually, since the bicarbonate buffering system is inefficient. A common error is overzealous hyperventilation in the setting of depressed CO_2 produc tion. When the PCO_2 decreases by 10 mmHg at 28°C (82°F), it doubles the pH increase of 0.08 that occurs at 37°C (99°F). Consider $ETCO_2$ monitoring to prevent hyperventilation. PART 15 Disorders Associated with Environmental Exposures The severity of anemia may be underestimated because the hema tocrit increases 2% for each 1°C drop in temperature. White blood cell sequestration and bone marrow suppression are common, potentially masking an infection. Although hypokalemia is more common in chronic hypothermia, hyperkalemia also occurs; the expected electro cardiographic changes are often obscured by hypothermia. Patients with renal insufficiency, metabolic acidosis, or rhabdomyolysis are at greatest risk for electrolyte disturbances. Coagulopathies are common because cold inhibits the enzymatic reactions required for activation of the intrinsic cascade. In addition, thromboxane B2 production by platelets is temperature dependent, and platelet function is impaired. The administration of platelets and fresh-frozen plasma is therefore not effective. Coagulation studies can be deceptively normal and contrast with the observed in vivo coagu lopathy. This contradiction occurs because all coagulation tests are rou tinely performed at 37°C (99°F), and the enzymes are thus rewarmed. ■ ■REWARMING STRATEGIES The key initial decision is whether to rewarm the patient passively or actively. Passive external rewarming simply involves covering and insu lating the patient in a warm environment. With the head also covered, the rate of rewarming is usually 0.5°–2°C (1.10°–4.4°F) per hour. This technique is ideal for previously healthy patients who develop acute, mild primary accidental hypothermia. The patient must have sufficient glycogen to support endogenous thermogenesis. The application of heat directly to the extremities of patients with chronic severe hypothermia should be avoided because it can induce peripheral vasodilation and precipitate core temperature "afterdrop," a response characterized by a continual decline in the core temperature after removal of the patient from the cold. Truncal heat application reduces the risk of afterdrop. Active rewarming is necessary under the following circumstances: core temperature <32°C (<90°F) (poikilothermia), cardiovascular

instability, age extremes, CNS dysfunction, hormone insufficiency, and suspicion of secondary hypothermia. Active external rewarming is best accomplished with forced-air heating blankets.

Other options include devices that circulate water through external heat exchange pads, radi ant heat sources, and hot packs. Monitoring a patient with hypother mia in a heated tub is extremely difficult. Electric blankets should be avoided because vasoconstricted skin is easily burned. There are numerous widely available options for active core rewarm ing. Airway rewarming with heated humidified oxygen (40°–45°C [104°–113°F]) via mask or endotracheal tube is a convenient option. Although airway rewarming provides less heat than do some other forms of active core rewarming, it eliminates respiratory heat loss and adds 1°–2°C (2.2°–4.4°F) to the overall rewarming rate. Crystalloids should be heated to 40°–42°C (104°–108°F), but the quantity of heat provided is significant only during massive volume resuscitation. The most efficient method for heating and delivering fluid or blood is with a countercurrent in-line heat exchanger. Heated irrigation of the gastrointestinal tract or bladder transfers minimal heat because of the limited available surface area. These methods should be reserved for patients in cardiac arrest and then used in combination with all avail able active rewarming techniques. If extracorporeal life support (ECLS) is unavailable, closed thoracic lavage is far more efficient in severely hypothermic patients with car diac arrest. The hemithoraxes are irrigated through two inserted largebore thoracostomy tubes. Thoracostomy tubes should not be placed in the left chest of a spontaneously perfusing patient for purposes of rewarming. Peritoneal lavage with the dialysate at 40°–45°C (104°– 113°F) efficiently transfers heat when delivered through two catheters with outflow suction. Another option involves the use of endovascular temperature control catheters. The HOPE (Hypothermia Outcome Prediction after Extracorporeal Life Support) score is a tool based on six covariates to help predict which patients may benefit from ECLS. ECLS options (Table 477-3) should be considered in severely hypothermic patients, especially those with primary accidental hypothermia. ECLS, including bypass, should be considered in nonperfusing patients without documented contraindications to resuscitation. Circulatory support may be the only effective option in patients with completely frozen extremities or those TABLE 477-3 Options for Extracorporeal Life Support EXTRACORPOREAL REWARMING TECHNIQUE CONSIDERATIONS Continuous venovenous (CVV) rewarming Circuit: CV catheter to CV, dual-lumen CV, or peripheral catheter No oxygenator/circulatory support Flow rates 150–400 mL/min ROR 2°–3°C (4.4°–6.6°F)/h Hemodialysis Circuit: single- or dual-vessel cannulation Stabilizes electrolyte or toxicologic abnormalities or rhabdomyolysis Exchange cycle volumes 200–500 mL/min ROR 2°–3°C (4.4°–6.6°F)/h Continuous arteriovenous rewarming (CAVR) Circuit: percutaneous 8.5-Fr femoral catheters Requires systolic blood pressure of 60 mmHg No perfusionist/pump/anticoagulation Flow rates 225–375 mL/min ROR 3°–4°C (6.6°–8.8°F)/h Cardiopulmonary bypass (CPB) Circuit: full circulatory support with pump and oxygenator Perfusate-temperature gradient 5°–10°C (11°–22° F) Flow rates 2–7 L/min (average 3–4 L/min) ROR up to 9.5°C (20.9°F)/h Venoarterial extracorporeal membrane oxygenation (VA-ECMO) Decreased risk of post-rewarming cardiorespiratory failure Improved neurologic outcome Abbreviations: CV, central venous; ROR, rate of rewarming.

with significant tissue destruction coupled with rhabdomyolysis. There is no evidence that extremely rapid rewarming improves survival in perfusing patients. TREATMENT Hypothermia When a patient is hypothermic, target organs and the cardiovas cular system respond minimally to most medications. Generally, medications are withheld below 30°C (86°F). In contrast to antiar rhythmics, low-dose vasopressor medications may improve the intra-arrest rates of return of spontaneous circulation. Because of increased binding of drugs to proteins as well as impaired metabo lism and excretion, either a lower dose or a longer interval between doses should be used to avoid toxicity. As an example, the adminis tration of repeated doses of digoxin or insulin would

be ineffective while the patient is hypothermic, but the residual drugs would be potentially toxic during rewarming. Achieving a mean arterial pressure of at least 60 mmHg should be an early objective. If the hypotension is disproportionate for temperature and does not respond to crystalloid/colloid infusion and rewarming, low-dose dopamine support (2–5 μg/kg per min) should be considered. Perfusion of the vasoconstricted cardiovas cular system also may improve with low-dose IV nitroglycerin. Atrial arrhythmias should be monitored initially without inter vention, as the ventricular response should be slow and, unless preexistent, most will convert spontaneously during rewarming. The role of prophylaxis and treatment of ventricular arrhythmias is complex. Preexisting ventricular ectopy may be suppressed by hypothermia and reappear during rewarming. There is limited evi dence to recommend any specific antiarrhythmic treatment. Initiating empirical therapy for adrenal insufficiency usually is not warranted unless the history suggests steroid dependence or hypoadrenalism or efforts to rewarm with standard therapy fail. The administration of parenteral levothyroxine to euthyroid patients with hypothermia, however, is potentially hazardous. Because labo ratory results can be delayed and confounded by the presence of the sick euthyroid syndrome (Chap. 394), historic clues or physical findings suggestive of hypothyroidism should be sought. When myxedema is the cause of hypothermia, the relaxation phase of the Achilles reflex is prolonged more than is the contraction phase. Hypothermia obscures most of the symptoms and signs of infec tion, notably fever and leukocytosis. Shaking rigors from infection may be mistaken for shivering. Except in mild cases, extensive cultures and repeated physical examinations are essential. Unless an infectious source is identified, empirical antibiotic prophylaxis is most warranted in the elderly, neonates, and immunocompromised patients. FROSTBITE Peripheral cold injuries include both freezing and nonfreezing cold injuries to tissue. Tissue freezes quickly when in contact with thermal conductors such as metal and volatile solutions. Other predisposing factors include constrictive clothing or boots, immobility, and vaso constrictive medications. Frostbite occurs when the tissue temperature drops below 0°C (32°F). Ice-crystal formation subsequently distorts and destroys the cellular architecture. Once the vascular endothelium is damaged, stasis progresses rapidly to microvascular thrombosis. After the tissue thaws, there is progressive dermal ischemia. The microvasculature begins to collapse, arteriovenous shunting increases tissue pressures, and edema forms. Finally, thrombosis, ischemia, and superficial necrosis appear. The development of mummification and demarcation may take weeks to months. CLINICAL PRESENTATION The initial presentation of frostbite can be deceptively benign. The symptoms always include a sensory deficiency affecting light touch, pain, or temperature perception. The acral areas and distal extremities

are the most common insensate areas. Some patients describe a clumsy or “chunk of wood” sensation in the extremity.

Deep frostbitten tissue can appear waxy, mottled, yellow, or vio laceous-white. Favorable presenting signs include some warmth or sensation with normal color. The injury is often superficial if the subcutaneous tissue is pliable or if the dermis can be rolled over bony prominences. Clinically, frostbite is superficial or deep. Superficial frostbite does not entail tissue loss but rather causes only anesthesia and erythema. The appearance of vesiculation surrounded by edema and erythema implies deeper involvement (Fig. 477-1). Hemorrhagic vesicles reflect a serious injury to the microvasculature and indicate severe frostbite. Damages in subcuticular, muscular, or osseous tissues may result in amputation. An alternative classification establishes grades based on the location of presenting cyanosis; that is grade 1, absence of cyanosis; grade 2, cyanosis on the distal phalanx; grade 3, cyanosis up to the metacarpophalangeal (MP) joint; and grade 4 cyanosis

proximal to the MP joint. The two most common nonfreezing peripheral cold injuries are chilblain (pernio) and immersion (trench) foot. Chilblain results from neuronal and endothelial damage induced by repetitive exposure to damp cold above the freezing point. Young females, particularly those with a history of Raynaud’s phenomenon, are at greatest risk. Persistent vasospasticity and vasculitis can cause erythema, mild edema, and pru ritus. Eventually plaques, blue nodules, and ulcerations develop. These lesions typically involve the dorsa of the hands and feet. In contrast, nonfreezing cold injury includes trench foot and immersion foot that results from repetitive exposure to wet cold above the freezing point. The feet initially appear cyanotic, cold, and edema tous. The subsequent development of bullae is often indistinguishable CHAPTER 477 Hypothermia and Peripheral Cold Injuries FIGURE 477-1 Frostbite with vesiculation, surrounded by edema and erythema.

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