By Robert Allan Davis, ADN, RN

The Big Chill: Accidental Hypothermia

How to identify and manage this life-threatening reaction to prolonged exposure.

Overview: A potential cause of such emergent issues as cardiac arrhythmias, hypotension, and fluid and electrolyte shifts, accidental hypothermia can be deadly, is common among trauma patients, and is often difficult to recognize. The author discusses predisposing conditions, the classic presentation, and the effects on normal thermoregulatory processes; explains how to conduct a systems assessment of the hypothermic patient; and describes crucial management strategies.

Keywords: accidental hypothermia, cold weather emergencies, exposure, rewarming

On May 10, 1996, around midnight, the renowned high-altitude guide Rob Hall led a team of 10 climbers from their high camp to summit the world's tallest mountain, Mount Everest. After extensive delays, some team members approached the peak at around 2 PM—too late to allow for a safe descent back to camp before nightfall. A storm engulfed the mountain at around 3 PM, casting the climbers into blizzard conditions. Overnight temperatures dipped below -60°F (-51.1°C) with 80 mile-per-hour winds. Hall survived overnight near the top, but could not descend because of severely frostbitten hands and feet. After a tearful goodbye to his wife over the radio, Rob Hall died, along with three other members from his team. Four climbers from two other teams also died that day, making it the deadliest day in Everest history.[1]

Jon Krakauer's personal account of this climb in his book *Into Thin Air* paints a classic picture of accidental hypothermia, in which extreme environmental conditions cause core body temperature to drop below 95°F (35°C). Not all cases of accidental hypothermia are so dramatic or played out on such a grand stage, yet they can be just as devastating and may be more common than previously believed.

According to the Centers for Disease Control and Prevention, between 1979 and 2002, an average of 689 deaths per year in the United States were attributed to prolonged exposure to cold temperatures.[2] Some clinicians, however, believe accidental hypothermia is underreported as a cause of hospital admission and death because of flawed reporting procedures and its frequent classification as a secondary cause.[3,4] For example, patients with traumatic injuries, who may be predisposed to accidental hypothermia because their injuries leave them unable to move into warm areas or because hypovolemic shock impairs their thermoregulatory system, are also at elevated risk for death if hypothermic.

Photo by Matt Rourke / Associated Press.

In a 1998 study, 661 (9.4%) of the 7,045 patients in a Nashville surgical ICU were hypothermic.[5] More than half (395) of the hypothermic patients were being treated for trauma, and of those patients nearly 53% (208) died. The authors concluded that hypothermia in trauma patients is "significant and independent of the month of admission," and "mortality is high." A study of 732 patients treated for major trauma in 2008 found that more than 13% had accidental hypothermia, and for these patients the mortality rate was nearly 30%—more than three times as high as the 9.15% overall mortality rate for the group.[6] To care for patients with accidental hypothermia, nurses need to know the predisposing conditions; recognize the signs and symptoms; and understand the normal mechanisms of thermoregulation, necessary aspects of assessment, and essential management techniques.

HOW HEAT IS LOST

Mechanisms of heat loss include

- radiation, the transfer of heat to the environment in the form of infrared rays.
- conduction, the movement of heat from the body to cooler objects in direct contact with it (the ground, a tree, or water in which it is submersed, for example).
- convection, the wind's wicking away of heat from exposed skin.
- insensible water loss, the release of heat through the evaporation of bodily fluids, as occurs during perspiration and breathing.

When ambient temperature drops, receptors in the skin and hypothalamus stimulate the desire to seek warmth, as well as such autonomic heating mechanisms as vasoconstriction and shivering. Shivering increases heat production fivefold.[7] The respiratory rate increases with shivering, along with the heart rate.

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Although skin and subcutaneous fat provide insulation from the cold, even at room temperature and at rest the body loses 55% to 65% of its heat through radiation and about 20% to 27% through insensible water loss.[8] During activity, insensible water loss rises; with increased humidity, insensible water loss (and subsequent heat loss) is minimized.[9] High altitudes, which drastically deepen and raise the respiratory rate, exacerbate

Table 1. Medical Disorders That Predispose to Hypothermia

Endocrinologic disorders
- hypopituitarism
- hypoadrenalism
- hypothyroidism
- hypoglycemia
- renal failure

Malnutrition

Nervous system disorders
- spinal cord transections
- neuropathies
- trauma
- stroke

Disorders affecting thermoregulation
- subarachnoid hemorrhage
- Parkinsonism
- multiple sclerosis
- Wernicke's syndrome
- agenesis of the corpus callosum

Skin disorders
- psoriasis
- exfoliative dermatitis

Sepsis

insensible water loss. Conduction occurs rapidly through wet clothes, which transmit body heat to the environment five times faster than dry clothes.[8] Similarly, when immersed in water, the body loses heat 25 to 30 times faster than it does when dry.[10] Heat loss by convection increases exponentially as wind speed rises.

WHO'S AT GREATEST RISK

Certain medical disorders predispose a person to hypothermia by reducing the body's ability to produce heat through their effects on hypothalamic, metabolic, or vascular processes (see Table 1). Likewise, extremes of age and drug intoxication increase the risk of accidental hypothermia.

Extremes of age. Infants are at high risk for accidental hypothermia because they have inadequate subcutaneous fat tissue, an immature thermoregulatory system that's incapable of producing a shivering response, and a surface area that's greater than their mass. Older adults are at elevated risk as a result of the normal physiologic changes associated with aging, such as a reduced ability to generate heat by metabolism and impaired temperature regulation, including a reduced shivering response. Such risks may be compounded by ineffective vasoconstriction and decreased subcutaneous fat, total body water, and muscle mass, which are also frequently seen in advanced age.[3, 11-13]

Drug intoxication. In one study involving 88 patients treated for accidental hypothermia in an ED, 55 were intoxicated with alcohol.[14] Alcohol dilates blood vessels, suppressing the shivering response, and lowers the thermoregulatory set point by directly affecting the hypothalamus.[12] In addition, because alcohol interferes with judgment, it affects a person's desire to seek shelter or put on warm clothing. Likewise, pharmacologic agents such as benzodiazepines, tricyclic antidepressants, opioids, barbiturates, and phenothiazines can reduce core temperature, as can lithium toxicity and, rarely, valproic acid.[12]

BODY SYSTEMS ASSESSMENT

Certain presentations and recent patient histories should immediately raise clinical suspicion of accidental hypothermia. These include being pale and cold to the touch with an altered level of consciousness and a depressed respiratory and heart rate. After identifying accidental hypothermia and conducting an airway, breathing, and circulation assessment, the health care team needs to address potential emergent conditions, such as cardiac arrhythmias, hypotension, and fluid and electrolyte shifts, while delivering supportive measures such as maintaining the airway, ensuring adequate oxygen delivery, and stabilizing hemodynamic status.

Throughout the systems assessment and rewarming process, it's crucial to measure temperature accurately, so as to avoid underestimating the severity of hypothermia. Because standard thermometers don't record temperatures below 93.2°F (34°C), and temperature measurement at any site is subject to error, it's best to monitor multiple sites continuously with a combination of esophageal, rectal, and bladder temperature probes. Keep in mind that esophageal probes are inaccurate when warmed, humidified oxygen is used; rectal probes may be inaccurate if a warm peritoneal lavage is performed or if the probe is wedged near cold stool; and both rectal and bladder temperatures tend to underreport core temperature because the probes are placed distal to the heart. Tympanic thermometers haven't proven to be accurate in hypothermic patients.[15]

Cardiovascular concerns. Cardiovascular compromise is present in all stages of accidental hypothermia. Initially, cold temperatures stimulate the sympathetic nervous system, but when hypothermia is moderate or severe, this protective mechanism fails.[10] At 95.9°F (35.5°C), a 700% increase in norepinephrine is released, causing massive peripheral vasoconstriction, which shunts blood to core organs.[16,17] As thermoregulation mechanisms deteriorate, heart and respiratory rates decline, reducing mean arterial pressure, cardiac output, and myocardial contractility—all of which may remain low until the patient is normothermic.[10] In moderate and severe hypothermia, dehydration, fluid shifts, and

elevated urine output diminish intravascular volume, concentrate the blood, and produce hypotension.[18] Changes in core temperature are accompanied by major electrolyte shifts, especially in potassium and magnesium, which can cause ectopy and cardiac arrhythmias.[18] While atrial or ventricular arrhythmias usually convert spontaneously with rewarming, below a temperature of 75.2°F (24°C), the risk of asystole is high.

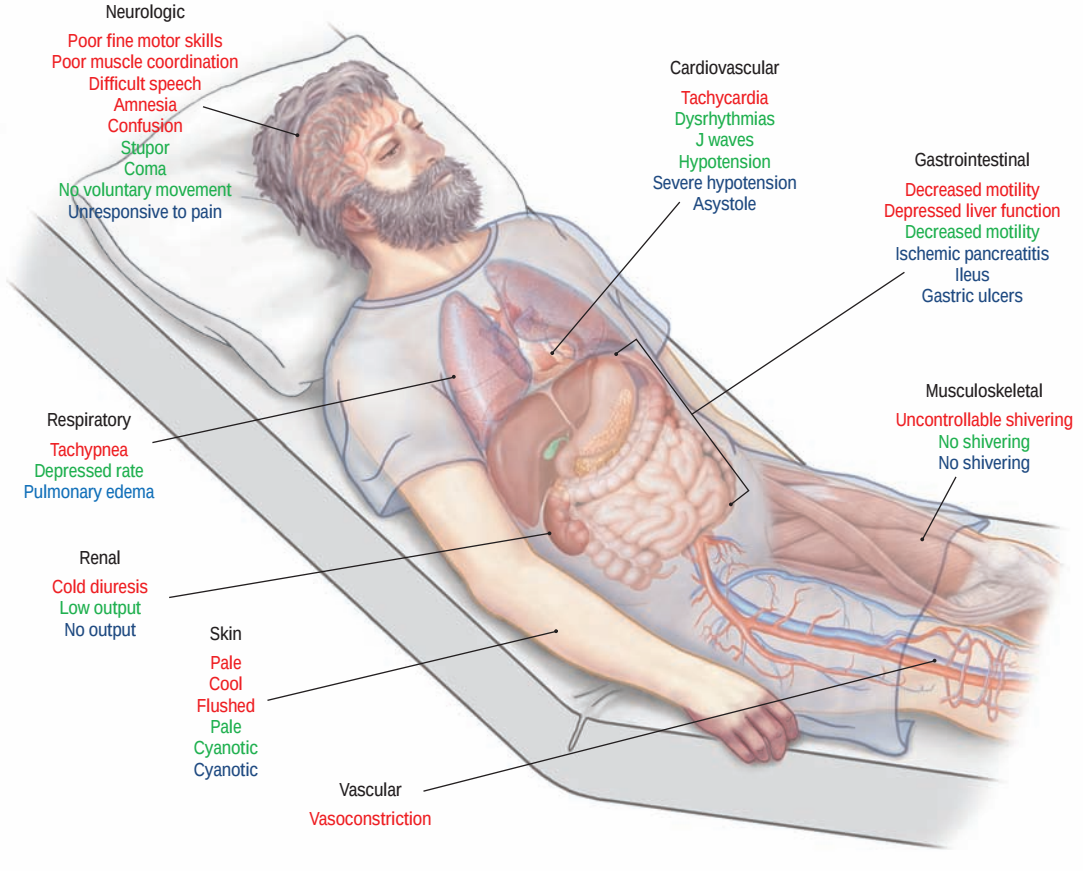
The hypothermic patient must be handled gently because sudden movement can trigger an arrhythmia. Because bradycardia is severe in hypothermia, the pulse at the carotid or femoral site should be assessed for 45 seconds to two minutes. Arterial and central venous blood pressures, as well as electrocardiographic activity, must be continuously monitored. An increased PR interval, a widened QRS complex, and a prolonged QT interval signal progressing hypothermia.

A J wave, also known as an Osborn wave, is a positive deflection often seen at the J point (where the QRS meets the ST segment) on the electrocardiograms of hypothermic patients. Caused by repolarization abnormalities, a J wave resembles a camel's hump. Detectable in 80% of patients with core body temperatures below 86°F (30°C), J waves are indicative of hypothermia but cannot be used to diagnose it because they're also seen in patients with cerebral injury, especially subarachnoid hemorrhage, and in myocardial ischemia. Care should be taken not to mistake J waves for an ST-segment myocardial infarction: J waves in hypothermia resolve with rewarming.[12]

Pulmonary assessment. The hypothermic patient is at high risk for aspiration pneumonia (from excessive bronchial secretions, impaired mucociliary function, and depressed cough reflex) and, after rewarming, for pulmonary edema.[7,10] The severely hypothermic patient may present with respiratory acidosis from hypoxia and metabolic acidosis secondary to lactic acid generation from poor tissue perfusion, impaired hepatic metabolism, and reduced acid excretion.[3,10]

It may take 30 to 45 seconds to assess the patient's respiratory rate; hypothermia depresses the medullary

Figure 1. Physiologic Effects of Hypothermia [20,21]



Red = mild (91.4°F–95°F [33°C–35°C]); green = moderate (85.2°F–89.6°F [29°C–32°C]); blue = severe (< 85.2°F [< 29°C]).

Illustration by Anne Rains.

respiratory center and slows breathing considerably.[7] To secure the airway, carefully insert an endotracheal or oropharyngeal tube. During intubation, ventricular fibrillation may result from poor preoxygenation.[12] Monitor pulse oximetry continuously.[15] Probes should be placed on the patient's ear or forehead because if they're placed on the finger, the oximeter will reflect peripheral vasoconstriction.

lead to inappropriate intervention because acid-base complications related to respiratory and metabolic pathways resolve when the patient is normothermic,[4, 19] and mild alkalosis (an uncorrected pH of 7.42 and $PaCO_2$ of 40 mmHg) is preferred and can be maintained through ventilator management.[4] Hyperventilation to correct respiratory acidosis is not recommended as it can rapidly change the blood pH, leading to ventricular fibrillation.[20]

Rewarming the hypothermic patient is a top priority, because most sequelae resolve when the patient is normothermic.

Monitor arterial blood gases on admission and during the rewarming stages. There is debate over whether arterial blood gas measurements should be adjusted to account for the patient's temperature.[3] Blood pH, partial pressure of oxygen, and partial pressure of carbon dioxide ($PaCO_2$) are temperature dependent, whereas bicarbonate remains constant.[4] Since blood gas analyzers heat blood samples to 98.6°F (37°C), they don't reflect the patient's true temperature.[3] You can adjust for the patient's temperature by applying a calculation to the results, and some analyzers make the correction automatically if you input the patient's temperature; however, some clinicians believe that corrected results

Table 2. Necessary Blood Tests in Accidental Hypothermia[4, 8]

At Admission	Every Four Hours
Complete blood count	Complete blood count
Complete metabolic panel	Basic metabolic panel
Magnesium	Potassium
Phosphorous	Magnesium
Ionized calcium	Arterial blood gases
Arterial blood gas studies	Coagulation studies
Lactate	
Fibrinogen	
Creatinine phosphokinase	
Amylase	
Creatinine kinase	
Creatinine kinase–muscle brain	
Troponin	
Toxicology screen	
Cortisol	
Thyroid function	

Neurologic evaluation. As hypothermia progresses, the patient's consciousness steadily declines from alertness to confusion, amnesia, and, finally, to coma.[7] With every 1.8°F (1°C) drop in temperature, cerebral blood flow is reduced 6% to 7%.[7] An electroencephalogram reveals abnormal waveforms at temperatures below 93.2°F (34°C) that flatten out at temperatures below 84.2°F (29°C).[10] Assess hypothermic patients for alertness, orientation, and motor function, and for level of consciousness by using the Glasgow Coma Scale.

Renal considerations. At the onset of accidental hypothermia, the body excretes large amounts of urine, regardless of its total fluid status. The likely cause of this "cold diuresis" is the initial peripheral vasoconstriction, which in mild hypothermia increases core intravascular volume and renal blood flow, thereby decreasing the antidiuretic hormone (ADH).[18] The subsequent drop in core temperature causes hypothalamic dysfunction, further reducing ADH levels and promoting more diuresis.[18] Other possible contributors to diuresis include malabsorption in the distal tubules of the kidneys, decreased glomerular filtration rates, and renal insensitivity to ADH.[7, 21] Insert a Foley catheter to monitor fluid status and renal function.

Gastrointestinal risks. At temperatures below 89.6°F (32°C), the gastrointestinal system slows, putting the patient at risk for an ileus.[7, 11] Cold temperatures also reduce hepatic function and may cause ischemic pancreatitis.[21, 22] Below 80°F (26.7°C), the pancreas may hemorrhage[7] and the gastrointestinal tract may develop punctate hemorrhages.[23]

To relieve gastric distension and prevent aspiration, after the endotracheal tube is placed, an orogastric or nasogastric tube can be inserted set to low, intermittent wall suction.[21] Because insulin action is impaired at temperatures below 86°F (30°C), initiate an IV insulin drip and check bedside blood sugar every one to two hours—from a central or arterial line, because capillary circulation is depressed.

Skin assessment. Note the color, temperature, moisture, and turgor of the patient's skin. Accidental

Table 3. Rewarming Options for Accidental Hypothermia

Passive External	Active External	Active Internal
Remove wet clothing	Warm blankets	Warmed IV saline
Blankets	Increased room temperature	Gastric lavage
Sleeping bag	Forced-air blanket	Colonic lavage
Tent	Heated water-circulating blanket or suit	Bladder lavage
Dry clothing	Fire	Peritoneal lavage
Insulation	Warm-water immersion	Thoracic lavage
	Heated fluidized-bead beds	Cardiopulmonary bypass
		Warmed inhaled oxygen
		Warmed blood
		Portable and percutaneous cardiopulmonary bypass
		Continuous arteriovenous rewarming
		Endovascular warming catheters
		Extracorporeal membrane oxygenation

hypothermia is frequently associated with local cold injuries such as frostnip or frostbite.

LABORATORY STUDIES

Once the hypothermic patient has been admitted to the hospital, admission blood work should be drawn. During rewarming, the nurse should monitor electrolyte shifts and acid-base status, watching for signs of infection or clotting disorders, repeating certain blood tests and coagulation studies every four hours (see Table 2 [4, 8]).

Clotting disorders develop in accidental hypothermia for several reasons. Cold conditions not only inhibit enzymes within the clotting cascade, but they also impair platelet function and reduce the overall platelet count through marrow suppression and the sequestration of platelets in the liver, portal venous system, and spleen.[10, 16] Hypothermic patients should be rewarmed and platelet function restored before platelets or clotting factors are transfused to treat bleeding.[10, 12] If patients are not actively bleeding, clotting will return to normal with rewarming.[10, 18]

On the opposite side of the clotting spectrum, thromboembolisms can occur as a result of hemoconcentration, a slowed circulation, and a release of tissue thromboplastins.[24] Disseminated intravascular coagulation has been reported in the context of severe clotting disorders.[10] For every 1°C drop in body temperature, blood viscosity rises by 2%.[7]

Clotting problems in accidental hypothermia are discovered through physical assessment. Serum laboratory studies for prothrombin time, partial thromboplastin time, and international normalized ratio produce deceptively normal results, despite obvious coagulopathies, because coagulation tests are warmed to 98.6°F (37°C).[10]

Low serum potassium, magnesium, and phosphorus are the most common electrolyte abnormalities in accidental hypothermia. Severe hypothermia and increased mortality risk are associated with elevated levels of amylase.[21]

REWARMING METHODS

Rewarming the hypothermic patient is a top priority, because most sequelae resolve when the patient is normothermic. The severity of the hypothermia and the availability of equipment determine the rewarming method. Treatment options range from blankets and water-circulating suits to warmed IV fluids and extracorporeal methods.

Patients who are warmed slowly have a higher mortality rate.[25] Rewarming that results in hyperthermia, however, is often detrimental, especially if the patient has ischemic or hypoxic brain damage.[26] There are three basic types of hypothermia treatment: passive external, active external, and active internal rewarming (see Table 3).

Passive external rewarming is used for patients with mild hypothermia, who are neither neurologically nor cardiovascularly compromised and are still able to generate heat. With this method, the patient's body is insulated—perhaps with blankets, reflective "space" blankets, or sleeping bags—in order to trap the heat it produces. Any wet clothing should be removed immediately to reduce heat loss. Passive external rewarming is the treatment of choice in the field and during transport because it's quick and easy to initiate and monitor.

Active external rewarming uses external mechanical means, such as increasing the ambient temperature or applying forced-air blanket systems, warm-water–circulating blankets or bodysuits, or heat packs to the

groin and neck to produce heat. Active external rewarming may be used in conjunction with passive external rewarming and is preferred for patients who present with moderate accidental hypothermia and poor cardiac performance. Confining active external rewarming to the trunk prevents a phenomenon known as *afterdrop*, in which the warming of extremities causes peripheral blood vessels to dilate, returning cold acidotic blood from the periphery to the core, further reducing core temperature and possibly triggering fatal ventricular arrhythmias.[4] Patients with moderate or severe accidental hypothermia are at elevated risk for afterdrop. However, some clinicians have found afterdrop to be clinically insignificant in their practice.[27]

Forced-air blanket systems are readily available in hospitals and are easy to use and maintain at the bedside.[18] These devices blow heated air through a disposable plastic or paper sheet, allowing for convective heat transfer, while shielding the patient from radiant heat loss. In one study, forced air blankets rewarmed patients at a rate of 4.32°F (2.4°C) per hour,[27] in another study by only 0.7°F (0.4°C).[28]

Similar to forced-air blankets, fluid-circulating blanket systems increase the patient's temperature by radiation, convection, and conduction. The blankets are connected to a temperature-regulating pump the size of a small refrigerator. Some systems use fluid-circulating garments that wrap around the patient's torso and legs, creating a larger heat-exchanging surface area. In one study, the garments raised core temperatures by 2.2°F (1.2°C) per hour.[28]

Active internal rewarming works faster and has better survival rates than noninvasive rewarming.

Active external rewarming rarely involves immersing the hypothermic patient in warm water. This method is not recommended because it makes it difficult to continuously monitor the patient and maintain a water temperature of 104°F to 106.7°F (40°C to 41.5°C). Heated fluidized-bead beds have been used as well, but are not readily available in the hospital setting because they need to be specially ordered or rented.

Active internal rewarming is reserved for patients who are hemodynamically unstable. Although these methods are invasive and associated with greater risks than noninvasive rewarming, they work faster and have better survival rates. This type of rewarming is performed primarily in the hospital setting and can include instilling heated saline into body cavities intravenously or through lavage. Extracorporeal methods can rewarm patients through an implanted central line or through cardiopulmonary bypass.

Saline warmed between 104°F and 109.4°F (40°C and 43°C) provides an easy means of increasing core temperature. Ideally, saline is warmed for IV administration using an in-line heater that monitors temperature, though a microwave oven may be used if an in-line heater isn't available.[10,11] Although one liter of warmed saline may increase a patient's temperature by only 0.25°F (0.14°C), infusing warm IV fluids is a standard of care for hypothermic patients and can be helpful if the patient requires substantial fluid resuscitation.[10] Lactated Ringer's solution is contraindicated in hypothermia because the hypothermic liver is unable to metabolize lactate, and fluids containing dextrose shouldn't be heated because heat causes dextrose to caramelize. Blood to be transfused should not be warmed above 107.6°F (42°C) and should not be microwaved because microwaving causes morphologic changes in red blood cells and subsequent hemolysis.[29]

If the patient is using a ventilator, inhaled air can be heated up to 113°F (45°C).[21] Using the alveolar surfaces for heat exchange in this way can raise the patient's core temperature by 0.9°F (0.5°C) per hour.[18] Heated oxygen is a standard treatment for all hypothermic patients, as it is readily available and easy to institute.[11,18,21]

Warm lavage is an active internal rewarming method that transfers heat from instilled warm fluids to the membranes of internal cavities. Gastric, colonic, and bladder lavage transfer minimal heat due to small surface areas.[21,30] Gastric lavage may provoke cardiac arrhythmias[23] and also carries a risk of aspiration.[21]

Peritoneal lavage, using 104°F to 113°F (40°C to 45°C) isotonic dialysate, not only rewarms the patient, but rewarms the liver directly, restoring hepatic function and eliminating metabolic byproducts and toxins.[10,21] It also dialyzes the patient, who may have renal hypoperfusion or insufficiency due to vasoconstriction and hypovolemia.[21] Peritoneal lavage can be used during cardiopulmonary resuscitation.

Thoracic lavage can be accomplished using a closed or open approach. A closed approach consists of inserting two chest tubes, one for instilling heated normal saline and the other for drainage.[30] An open approach into the thorax is performed in the operating room, where a left thoracotomy approach is used. Thoracic lavage directly warms the heart, even if circulation is impaired, and the rate of rewarming ranges from 5.4°F to 10.8°F (3°C to 6°C) per hour.[30] With thoracic lavage, cardiopulmonary resuscitation can be performed by external compressions or internal cardiac massage. Possible complications include bleeding and infection.[30]

Cardiopulmonary bypass has been used to treat patients with moderate or severe hypothermia who are in cardiac arrest or whose hemodynamic status is unstable.[22,31] Cardiopulmonary bypass allows for rapid

rewarming of 1.8°F to 3.6°F (1°C to 2°C) every three to five minutes.[21] The peripheral method accesses the femoral artery and vein, allowing for chest compressions; it requires relatively little preparation time (for cannulation) compared with thoracotomy; and it avoids potential complications associated with an open chest.[22, 31] The thoracotomy method cannulates the right atrium of the heart and the ascending aorta. It allows for good cardiac massage, left ventricular venting, internal defibrillation, and greater perfusion rates.[22] The greatest disadvantages of cardiopulmonary bypass are its complexity, cost, and unavailability in certain areas. Multiple organ failure is common after rewarming by cardiopulmonary bypass.[22] Considering the condition of patients that require cardiopulmonary bypass, it has an impressive survivability rate, calculated to be 60% in one analysis of 68 hypothermic patients who underwent the procedure.[31]

Portable and percutaneous cardiopulmonary bypass systems are available in some hospitals. They work the same way cardiopulmonary bypass does but can be initiated in the ED and set up in about 42 minutes (versus 134 minutes for cardiopulmonary bypass), and they rewarm at a rate of 7.2°F (4°C) per hour.[32]

Continuous arteriovenous rewarming, another option for rewarming severely hypothermic patients, accesses a femoral artery and vein and runs the patient's blood through an external heating element. Because the patient's own blood pressure circulates the blood through the circuit, it requires a minimum systolic blood pressure of 60 mmHg. The benefit of continuous arteriovenous rewarming is that heat exchangers are readily available in hospitals and the system is nonheparinized.[10] Rewarming occurs at a rate of 5.4°F to 7.2°F (3°C to 4°C) per hour.[10] If the patient has a dialysis shunt, it can be used for access.[24]

Endovascular warming catheters are central venous catheters used to circulate heated water to raise core temperature. The catheter is attached to a small refrigerator-sized control unit with temperature control. The rewarming rate averages 4.5°F (2.5°C) per hour. Endovascular catheters are less invasive than cardiopulmonary bypass, portable and percutaneous cardiopulmonary bypass, and continuous arteriovenous rewarming: initiation requires only the placement of a single central venous line.[33] Complications are the same as for central venous catheters and include deep vein thrombosis and line infections.[33]

Extracorporeal membrane oxygenation, normally used in the neonatal intensive care setting to facilitate oxygen delivery and carbon dioxide removal in newborns with meconium aspiration syndrome or respiratory distress syndrome and in those undergoing cardiac surgery, is used to treat accidental hypothermia as well. It can rewarm at rates similar to cardiopulmonary bypass, while lowering the risk of multiple organ failure associated with reperfusion.[22] The downside to extracorporeal membrane oxygenation is that it is not available in many hospitals and requires expert training.

In treating hypothermia, the goal is to rewarm the patient quickly without overshooting normal body temperature. Even mild hyperthermia can cause severe neurologic outcomes, with ischemic or hypoxic brain injuries.[26] Some clinicians set a temperature goal for active rewarming of 95°F (35°C).[26] When this temperature is achieved, passive external rewarming is sufficient to bring the patient up to normal temperatures. To improve neurologic outcomes in accidental hypothermia, researchers have suggested rewarming hypothermic patients to a mild hypothermic state and sustaining that state for 24 hours, as is the practice with induced hypothermia after cardiac arrest.[33]

Normal advanced cardiac life support pathways are modified for hypothermic patients because medications and defibrillation are ineffective at such low body temperatures.

ADVANCED CARDIAC LIFE SUPPORT

Cardiopulmonary resuscitation efforts should continue until the patient is warmed to between 89.9°F and 95°F (32°C and 35°C) even if unresponsive (as it is often said in the stark language of the ED, hypothermic patients aren't dead until they're "warm and dead"). Prolonged cardiopulmonary resuscitation times of more than six hours have shown good neurologic outcomes.[18]

Normal advanced cardiac life support pathways are modified for hypothermic patients because medications and defibrillation are ineffective at such low body temperatures. A defibrillator can be attached to the patient in case of ventricular fibrillation or pulseless ventricular tachycardia,[20] but until the core temperature is raised to between 78.8°F and 84.2°F (26°C and 29°C), only one shock is advised because defibrillation is ineffective below those temperatures.[23, 30] Transvenous pacing may cause ventricular arrhythmias and is not recommended.[18]

Normal advanced cardiac life support drugs, such as lidocaine, procainamide, propranolol, diltiazem, verapamil, and neuromuscular blockers, are ineffective below temperatures of 86°F (30°C).[12, 18] In porcine models, epinephrine didn't improve outcomes during cardiopulmonary resuscitation,[34] and there have been reports that it has precipitated ventricular fibrillation in humans.[12] Vasopressin, on the other hand, has improved defibrillation.[18]

Vasopressors have been shown to cause arrhythmias and precipitate ventricular fibrillation.[12, 35] They are virtually ineffective in the hypothermic body because the vasculature is maximally constricted in response to the cold.[12] To raise blood pressure, vascular volume must be increased.[9]

THE LESSONS OF EVEREST

Rob Hall and the climbers who died with him on Mount Everest did so because definitive treatment was unavailable. For the hypothermic patients admitted to hospitals under similar though less spectacular conditions, it's important that nurses know how to recognize, assess, and manage this challenging condition that's associated with such a wide variety of pathophysiologic states. ▼

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