

The Hypothermic Patient: “It’s Really Not That Cool . . .”

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Objectives

At the end of the CE module, the EMS provider will be able to:

1. Discuss the pathophysiology of hypothermia.
2. Identify the differences between mild, moderate and severe hypothermia.
3. Recognize the differences in management of patients with hypothermia versus the non-hypothermic patient.
4. Identify two general causes of hypothermia.



photo by Audra Horton

Case Scenario

It is a cool February morning and you and your partner are evaluating Mr. Sanders, a 72-year-old male, at his son’s home. The son states that Mr. Sanders lives with him and his family. The son tells you that his dad has been diagnosed with “dementia” and has a tendency to wander from the home if the house is not secured prior to bedtime. The son tells you that he found his dad asleep in his pajamas in a chair on the front porch this morning when he went out to get the paper. The forecasted temperature for the previous night was 30° Fahrenheit. Your evaluation of Mr. Sanders reveals a male in what appears to be good physical condition. He has a confused look on his face and responds to your questions with slow, slurred speech that varies in pitch. His skin is cool to the touch, his blood pressure is 106/68 and his pulse rate is 108. His respiratory rate is 24 breaths per minute. Your partner applies the ECG monitor, which reveals an irregular rhythm with what appears to be an anomaly in the ST segment. The son tells you his father takes only an aspirin a day and no prescription medications. He also tells you that he is not allergic to any medications. At this point you decide to prepare the patient for treatment and transport.

Introduction

The incidence of hypothermia is always greater in areas with cooler climates; however, we cannot always rule out hypothermia on a patient simply because the ambient temperature is above 32° C (90° F). EMS providers must be able to recognize hypothermia and understand the pathophysiology of hypothermia.

Hypothermia is defined as core body temperature less than 32° C (95° F), however, some authorities believe an alternate definition of hypothermia is any drop in the core temperature of 2° C (3.5° F) from the normal core temperature (normal core temperature is 37° C or 98.6° F). We can further break hypothermia into three classifications: mild, moderate and severe.

- Mild hypothermia is defined as a core temperature of 32°–35° C (90°–95° F).
- Moderate hypothermia is defined as a core temperature of 28°–32° C (82°–90° F).
- Severe hypothermia is defined as a core temperature less than 28° C (82° F).

Each of the above classifications present with unique conditions and causes and will be discussed in detail later in this article.

Causes of Hypothermia

Causes of hypothermia can be divided into two general categories: temperature regulation and increased heat loss. Let's look at each of them in more detail.

Temperature Regulation

Cold receptors located throughout the body — in the skin, mucous membranes and deep tissues — recognize temperature changes in the body and send a signal to the hypothalamus. The hypothalamus, located in the base of the brain, is the body's temperature regulation center. It is very sensitive and controls the body mechanisms that generate heat (e.g., shivering) or promote heat loss (e.g., sweating). For the purposes of this discussion we will focus only on heat generation.

Heat is generated through the release of a chemical mediator, norepinephrine. Norepinephrine, a catecholamine, exerts both primary and secondary stimulatory responses that result in vasoconstriction, which shunts blood away from the surface of the body and toward vital organs and muscles. Another mechanism of heat generation and body temperature control is the basal metabolic rate, or the rate at which the body's metabolism is occurring when completely at rest. The body continuously adjusts the metabolic rate in order to maintain the normal core temperature. In states of hypothermia, the chemical mediator adrenal cortisone (a hormone) increases the body's metabolic rate to promote the generation of heat.

Several endocrine abnormalities may cause decreased heat production, including hypopituitarism, hypoadrenalism and hypothyroidism. Other causes include severe malnutrition or hypoglycemia and neuromuscular inefficiencies seen in the extremes of age. A disruption in heat generation, regardless of air temperature, can lead to hypothermia.

When not related to external temperatures, a failure of the hypothalamus to regulate core body temperature commonly results in hypothermia; however, impaired temperature regulation may also occur with central nervous system (CNS) trauma, strokes, toxicologic and metabolic derangements, intracranial bleeding,

Parkinson's disease, CNS tumors, Wernicke disease and multiple sclerosis.

Increased Heat Loss

As stated previously, the body strives to maintain a constant core temperature at all times. However, external stimuli constantly act on the body and can accelerate heat loss, which can result in hypothermia if the body is unable to control or act on those stimuli.

We lose heat through several methods: conduction, convection, radiation, evaporation and respiration.

- **Conduction:** method of heat loss occurring as a result of direct contact of the body, or body parts, with a cooler object. Heat flows from a higher temperature object to a lower temperature object.
- **Convection:** method of heat loss occurring as a result of air currents passing over the body. Heat is first conducted to the air before convection can occur.
- **Radiation:** method of heat loss occurring as a result of heat dissipating from the body into the air. Heat transfers from an area of higher temperature to an area of lower temperature.
- **Evaporation:** method of heat loss occurring as a result of water evaporating from the skin or from the lungs during breathing.
- **Respiration:** method of heat loss through convection, radiation and evaporation during the breathing cycle. This particular heat loss method is not often considered a direct method of heat loss because it is the combination of other methods. But, physiologically, respiration or breathing can be a critical means of heat loss.

A rapid and unexpected increase in heat loss is probably the most common cause of hypothermia. This includes accidental hypothermia because of either submersion in or exposure to cold ambient temperatures. Patients may also present with peripheral vasodilation caused by pharmacologic or toxicologic agents. Patients may have burns or



psoriasis, which decrease the body's ability to preserve heat, or they may be hypothermic as a result of our own treatment or management of a different condition. Cold fluid infusions, overenthusiastic treatment of heatstroke or emergency obstetrical deliveries may cause increased-heat-loss induced hypothermia.

Pathophysiology

Patients suffering from hypothermia experience a marked overall decrease in tissue oxygenation, due to respiratory depression. In addition, metabolism of organic acids from the liver decreases and lactic acid from the poorly perfused skeletal muscle builds up. These factors can lead to respiratory and metabolic acidosis. This acidosis can lead to enzymatic dysfunction because enzymes and some hormones are very sensitive to both pH and temperature changes. Without these enzymes, the ability to lower the energy barrier to carry out various chemical reactions within the body may be impaired.

Hypothermia can also manifest itself in liver dysfunction. Poor hepatic perfusion results in the depletion of glycogen, which can lead to profound **hypoglycemia**. However, **hyperglycemia** — a result of both a decrease in insulin production and release as well as inhibited peripheral sensitivity to insulin — may also present as a result of severe hypothermia.

Hypothermic patients may also experience renal complications. Patients may have an increase in diuresis (cold diuresis), which can result in significant volume depletion and electrolyte imbalance. Additionally, urine carries heat away with it, further exacerbating a hypothermic state.

Cardiac physiology is affected by hypothermia in various ways. In mild hypothermia, the body will attempt to compensate by increasing the heart rate, blood pressure and peripheral vascular resistance. Blood is shunted away from the periphery to vital organs, and the metabolic rate is increased to promote heat generation. In moderate hypothermia, these compensatory mechanisms will begin to fail, and you may witness atrial fibrillation or other atrial and ventricular arrhythmias. The heart rate will

progressively decrease with a resultant drop in cardiac output. A “J-wave” or “Osborn wave” may be seen on the ECG. It appears as a small, rounded wave at the J-point of the QRS complex, can sometimes mimic ST-Elevation and is best seen in V4. In severe hypothermia, compensatory mechanisms fail completely, and susceptibility to ventricular fibrillation and asystole is extremely high due to severe tissue ischemia, alterations in electrolytes, local tissue acidosis, contractility dysfunction and myocardial cell irritability.

Clinical Presentation

Hypothermia is fairly obvious in settings that involve severe environmental exposure. However, elderly, infirm or pediatric patients exposed to less extremes of cold may not present as obviously hypothermic. The same may be true for patients who are wet. Mild or moderate hypothermia can present with misleading symptoms, such as confusion, dizziness, chills or dyspnea.

Initial symptoms may be detected by friends and/or family members prior to your arrival and those symptoms will assist you in your assessment. They can include mood change, irritability, poor judgment and apathy. Bystanders may tell you the patient was removing clothing as though he or she was too warm (called paradoxical undressing). This action is in response to prolonged cold stress and is evident in the moderately hypothermic person. They may report that the patient was performing rhythmic or repeated motions, such as rocking. The patient may present with slurred speech and ataxia mimicking a stroke, alcohol intoxication or drug use. There are also other contributory factors such as the use of alcohol, overdose, psychiatric emergencies and major trauma that can be associated with an increased risk of hypothermia.

The key to determining an indication of hypothermia is the rapid determination of the patient's core temperature. In the field, core temperature may be difficult to establish reliably. A special low-reading thermometer can be used orally or rectally, but with less reliability, and it may not reflect a core temperature. Rectal thermometers must be placed at least 15 cm deep in order to obtain

any semblance of accuracy. Temporal or infrared tympanic temperature recording devices may be more accurate in the field than the rectal thermometer. Once in the emergency department, the patient's core temperature will be measured using a low-reading temperature probe in the bladder or rectum or an esophageal probe.

Obtaining a core temperature is a valuable assessment tool to further assist the provider in determining whether the patient has an altered mental status caused by stroke, drug overdose, alcohol intoxication or mental illness.

Specific physical examination findings may vary among patients. However, upon examination, you will be able to classify the patients based on their presenting symptoms into mild, moderate or severe hypothermia.

Mild hypothermia ($32\text{--}35^{\circ}\text{C}$ or $90^{\circ}\text{--}95^{\circ}\text{F}$):

Between 34°C and 35°C , most people will begin to shiver vigorously, usually in all extremities. They may complain of muscle cramping as a result of the shivering. As their temperature drops below 34°C , they may develop altered judgment; amnesia; and slow, slurred speech. Patients may have difficulty controlling their voice qualities (varying high/low pitch and tone). Their respiratory rate may increase. At approximately 33°C , ataxia and apathy may be seen. Patients are still generally hemodynamically stable and are able to compensate for the symptoms. You may also observe hyperventilation, tachypnea, tachycardia and cold diuresis as renal concentrating ability is compromised.

Moderate hypothermia ($28\text{--}32^{\circ}\text{C}$ or $82^{\circ}\text{--}90^{\circ}\text{F}$):

The body's oxygen consumption will begin to decrease and you will see a depression of the CNS, resulting in hypoventilation and hyporeflexia (e.g., slow or absent reflexes). Paradoxical undressing may also occur. Most patients with a temperature of 32°C or lower will present in a stupor. As the core temperature reaches 31°C or below, the body loses its ability to generate heat by shivering. At 30°C , patients develop an increased risk for arrhythmias. Most commonly, atrial fibrillation and other atrial and ventricular rhythms become more likely. The patient's pulse will

continue to slow progressively, and cardiac output will be markedly reduced. As mentioned earlier, the "J-wave" or "Osborn wave" may be seen on ECG in moderate hypothermia. Between 28°C and 30°C , pupils may become dilated and minimally responsive to light, which may mimic brain death.

Severe hypothermia ($<28^{\circ}\text{C}$ or 82°F): At 28°C , the body becomes extremely susceptible to ventricular fibrillation and an increased depression of myocardial contractility. You may be able to observe pulmonary edema with ventilation. The patient will more than likely be comatose and hypotensive, and he or she will present muscular rigidity. In most cases, patients will also be unresponsive (with fixed pupils), apneic and pulseless.

Management of Hypothermia

Assessment and recognition are the keys to treating hypothermia; follow basic life support (BLS) principles, with attention to airway, breathing and circulation. When the patient is extremely cold but has maintained a perfusing rhythm, your responsibility is to focus on interventions that prevent further heat loss and begin to rewarm the victim. These interventions include the following:

- Prevent additional heat loss by removing wet clothing.
- Protect the patient from further environmental exposures.
- Maintain the patient in a horizontal position.
- Avoid rough handling (which may trigger arrhythmias).
- Monitor core temperature.
- Monitor ECG for cardiac arrhythmias.

For patients with moderate to severe hypothermia, management is determined by the presence or absence of a perfusing rhythm. If the hypothermic patient is not in cardiac arrest, attention should be focused on warming the patient with available methods. Handle the victim gently for all procedures; rough handling or manipulations have been reported to precipitate ventricular fibrillation.

If the patient is in cardiac arrest, BLS management should still target airway, breathing and circulation but with some



modifications. The hypothermic patient will have a slow pulse and respiratory rate that may be difficult to detect. As a result, the BLS provider should assess breathing and pulse for a period of 30 to 45 seconds to confirm respiratory arrest, cardiac arrest or bradycardia that is profound enough to require CPR. If the patient is not breathing, start rescue breathing immediately. If possible, administer warmed humidified oxygen during bag-mask ventilation. If the patient is pulseless with no detectable signs of circulation, start chest compressions immediately. If there is any doubt about whether a pulse is present, begin compressions.

The temperature at which defibrillation should first be attempted in the severely hypothermic patient and the number of defibrillation attempts that should be made have not been established. But if ventricular tachycardia (VT) or ventricular fibrillation (VF) is present, defibrillation should be attempted. If VF is detected, it should be treated with one shock then immediately followed by resumption of CPR. If the patient does not respond to one shock, further defibrillation attempts should not be attempted, and the provider should focus on continuing CPR and transporting the patient to a facility that can begin active rewarming.

For the advanced provider, if the patient is unresponsive or in arrest, endotracheal intubation is appropriate. Intubation serves two purposes in the management of hypothermia: It enables provision of effective ventilation with warm, humidified oxygen, and it can isolate the airway to reduce the likelihood of aspiration. ACLS management of cardiac arrest due to hypothermia focuses on more aggressive active core rewarming techniques as the primary treatment. The hypothermic heart may be unresponsive to cardiovascular drugs, pacemaker stimulation and defibrillation. In addition, drug metabolism is reduced as a result of disruptions caused by severe hypothermia. It is possible that cardioactive medications can accumulate to toxic levels in the peripheral circulation if given repeatedly. For these reasons, IV drugs are often withheld.

For the advanced provider, if the patient is not in arrest and is mildly or moderately

hypothermic, BLS measures should be instituted and maintained while transporting to the closest appropriate facility. If IV solutions are started on the patient, they should be warmed to a temperature of 43° C (109° F). Caution should be used in administering non-warmed IV solutions so that the core temperature does not decrease further. If IV medications are indicated, consideration should be given to the effectiveness of the medication compared to the patient's temperature. Online medical consultation may be the best resource prior to administering any medication.

As noted previously, a defibrillation attempt is appropriate if VF/VT is present. If the patient fails to respond to the initial defibrillation attempt or initial drug therapy, defer subsequent defibrillation attempts or additional boluses of medication until the core temperature rises above 30°C (86°F). Sinus bradycardia may be physiologic in severe hypothermia (i.e., appropriate to maintain sufficient oxygen delivery when hypothermia is present), and cardiac pacing is usually not indicated.

In-hospital treatment of severely hypothermic patients in cardiac arrest will be directed at rapid core rewarming. The techniques for in-hospital controlled rewarming include administration of warmed, humidified oxygen (42°C to 46°C or 108°F to 115°F); warmed IV fluids (normal saline) at 43°C (109°F); peritoneal lavage with warmed fluids; pleural lavage with warm saline through chest tubes; and/or extracorporeal blood warming with partial bypass and cardiopulmonary bypass.

In the field, resuscitation may be withheld if the patient has obvious lethal injuries or if the body is frozen so that CPR or ventilator assistance is impaired due to body stiffness or muscle rigidity.

Some clinicians believe that patients who appear dead after prolonged exposure to cold temperatures should not be considered dead until they are warmed to near normal core temperature.

Hypothermia may exert a protective effect on the brain and organs if the hypothermia develops rapidly in cardiac arrest. Sometimes it may not be possible to determine whether

arrest or hypothermia occurred first, in that case the provider should start with CPR. The provider should start measures to limit heat loss and begin rewarming.

Case Revisited

As you prepare Mr. Sanders for transport, you explain to the son that you think his father is hypothermic due to being exposed to the ambient cold temperature for an extended period of time last night. You apply an electronic temporal thermometer and it confirms your assessment. The patient's temperature is 33° C (93° F). You place Mr. Sanders supine on your stretcher and begin passive rewarming by wrapping him in blankets and moving him to the ambulance where you have instructed your partner to turn on the heater full blast to try to warm Mr. Sanders. You assess blood glucose level and determine it to be 90 mg/dl. If available, you will administer warmed, humidified oxygen and provide a smooth ride to the hospital. As an ALS provider, you administer an IV of normal saline, KVO that has been in an IV warmer that maintains the solution at a temperature of 43° C (109° F) and transport.

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