Accidental Hypothermia

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Accidental hypothermia, defined as an unintentional drop in core body temperature to less than 35°C (95°F), is commonly encountered in emergency departments. In the United States, approximately 700 deaths are attributed to accidental hypothermia each year,[1–3] with approximately half of all hypothermia-related deaths occurring in the elderly. A rise in the incidence of hypothermia has been attributed to increased participation in outdoor activities as well as social problems related to alcoholism, mental illness, and homelessness.[4]

Hypothermia is classified based on core body temperature as mild (32°C–35°C), moderate (28°C–32°C), and severe (< 28°C). Management depends on the degree of hypothermia, with severe hypothermia requiring more aggressive treatment (eg, active internal rewarming). Mild hypothermia is not associated with significant mortality; however, the mortality rate associated with moderate to severe hypothermia is 30% to 80%.[5] This article reviews the pathophysiology, etiology, clinical manifestations, and management of accidental hypothermia.

TAKE HOME POINTS

- Hypothermia is defined as core body temperature less than 35°C.
- Hypothermia is classified as mild (32°C–35°C), moderate (28°C–32°C), and severe (< 28°C) based on the degree of hypothermia, with clinical signs and symptoms guiding management.
- Osborn waves, a positive deflection at the ST segment, may be seen on electrocardiography, but this finding is not a pathognomonic feature of hypothermia.
- Management involves evaluating the patient for a possible cause of hypothermia and rewarming.
- Passive rewarming is a slow process and should be used in patients with mild hypothermia.
- Active rewarming is faster and more efficient and should be used in patients with moderate or severe hypothermia or in patients with cardiovascular compromise.
- A patient should be pronounced dead only when "warm and dead."

THERMOREGULATION

Temperature regulation involves loss, conservation, and production of heat and varies in a diurnal fashion within a range of 0.5°C. Body temperature also varies with menstrual cycle by 1°C.[6] Temperature is regulated centrally by the hypothalamus in coordination with peripheral cold and heat receptors. Under normal circumstances, alterations in ambient temperature initiate accommodative changes that maintain core body temperature within a narrow range (0.2°C–0.4°C). Deviations in temperature from a set point are sensed by the hypothalamus, which triggers the initiation of mechanisms that produce, conserve, or dissipate heat. Behavioral responses (eg, moving to a warmer environment, layering of clothes) as well as heat production through shivering and increased basal metabolic rate due to hormonal activity involving thyroxin, cortisol, and epinephrine contribute to changes in core body temperature. The temperature thresholds for sweating and vasodilation, vasoconstriction, nonshivering thermogenesis, and shivering are 37°C, 36.8°C, 36°C, and 35.5°C, respectively.[6] Radiation and conduction contribute to 70% of heat loss. Other methods of heat loss include convection, evaporation of sweat, and respiration.[1,7]

ETIOLOGY

Hypothermia is the end result of various factors related to disturbances in temperature regulation (**Table 1**).[1,3,8,9] Decreased basal metabolic rate as seen in

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Table 1. Etiology of Accidental Hypothermia

Hypoadrenalism
Hypothyroidism
Hypopituitarism
Hypoglycemia
Diabetic ketoacidosis
Extremes of age (lack of behavioral response and impaired shivering)
Skin disorders (burns)
Drugs (benzodiazepines, phenothiazine, barbiturates, ethanol)
Neurologic (head trauma, stroke, tumors)
Sepsis
Environmental exposure (cold water immersion, inadequate indoor heating)
Iatrogenic (fluid resuscitation, perioperative)

hypopituitarism, hypothyroidism, and hypoadrenalism can lead to hypothermia. The presence of a neurologic disorder can lead to altered temperature thresholds, which may result in changes in the behavioral response to hypothermia. Inappropriate behavioral response can occur in patients with head trauma, cerebrovascular accident, and intracranial tumors.

Hypothermia also can be caused by environmental factors, medications, and alcohol or can occur along with skin disorders. In the elderly, hypothermia can occur in the setting of an ambient cold environment, such as air conditioning or ice baths, as a result of impaired shivering mechanisms associated with older age. Other factors that place elderly patients at increased risk for hypothermia include a decreased ability to detect temperature changes as well as a decreased ability to generate heat because of reduced lean body mass and impaired mobility.[10] Immersion in cold water also can cause hypothermia, as the heat conductivity of water is 30 times that of air. Medications, such as phenothiazine and sedatives, can decrease the temperature threshold of the hypothalamus and cause impaired behavioral response, leading to hypothermia.[11] Alcohol causes vasodilation of cutaneous blood vessels and impairs the behavioral response to stimuli, increasing the risk for hypothermia. Skin disorders, such as burns, exfoliative disease, and severe psoriasis, may lead to loss of skin (a natural insulator), thereby resulting in hypothermia.[12] When hypothermia is associated with trauma (either blunt or penetrating), the outcome is significantly worse.[13] Iatrogenic causes of hypothermia can be attributed to medications mentioned above, resuscitative intravascular fluids, infusion of blood products, and perioperative anesthetics. It is essential to keep in mind that blood products are preserved at 39.2°F (4°C).[14]

Table 2. Clinical Manifestations of Hypothermia

Mild hypothermia (32°C–35°C)
Confusion
Dysarthria
Shivering
Tachycardia
Hypertension
Bronchorrhea
Cold diuresis
Atrial fibrillation
Electrolyte imbalances
Hyperglycemia
Moderate hypothermia (28°C–32°C)
Decreasing level of consciousness
Dilated pupils
Hallucinations
Loss of shivering mechanism
Bradycardia
Cold diuresis
Osborn waves (J waves) on electrocardiography
Ventricular arrhythmias
Severe hypothermia (< 28°C)
Coma
Loss of reflexes
Loss of activity on electroencephalography
Bradycardia
Oliguria
Asystole at < 20°C
Apnea at < 24°C
Pulmonary edema
Osborn waves (J waves) on electrocardiography

Adapted with permission from Kempainen RR, Brunette DD. The evaluation and management of accidental hypothermia. Respir Care 2004;49:195.

CLINICAL MANIFESTATIONS

The clinical manifestations of hypothermia are a spectrum of body responses to loss of heat and decreasing temperature (**Table 2**). Initially, the body tries to combat hypothermia by conserving heat and increasing catecholamines, thereby producing responses such as tachycardia, tachypnea, and increased blood pressure, cardiac output, and minute ventilation. Although these body responses may maintain core body temperature within the normal range, they result in increased oxygen consumption and shivering. As the patient continues to lose heat, these responses tend to cause fatigue; the patient becomes confused, bradycardic, hypotensive, and cardiac output and metabolic rate decrease.

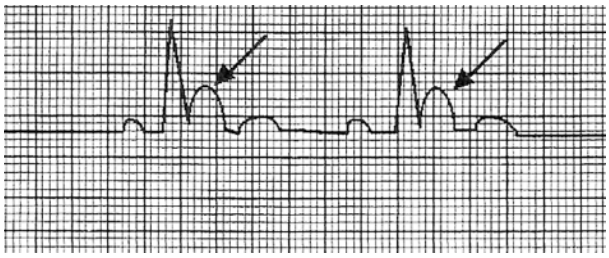

Figure. An electrocardiogram demonstrating Osborn waves, representing a positive deflection at the ST segment. (Reprinted from Thornton D, Farmer JC. Hypothermia and hyperthermia. In: Parillo JE, Dellinger RP, editors. Critical care medicine: principles of diagnosis and management in the adult. 2nd ed. St. Louis: Mosby; 2002: 1529. Copyright 2002, with permission from Elsevier.)

Electrocardiographic (ECG) changes seen in patients with mild hypothermia initially include tachycardia, suppression of ventricular ectopy, and prominence of atrial arrhythmias. As hypothermia progresses (ie, moderate hypothermia), bradycardia proceeds to asystole. Asystole has been suggested to be the primary cause of cardiopulmonary arrest due to hypothermia.[10] Additionally, ventricular fibrillation may occur as a result of cardiac stimulation through rewarming, hypocapnia, alkalosis, or physical manipulation.[10] Although not pathognomonic, the ECG in moderate or severe hypothermia may show a J wave or Osborn wave (**Figure**), which represents a positive deflection at the ST segment.[10,15] These changes are best seen in the lateral leads with increasing amplitude as hypothermia worsens. J wave/Osborn waves may also be seen in cases of subarachnoid hemorrhage and intracranial injuries as well as in myocardial ischemia in the absence of hypothermia. Other ECG changes include PR, QRS, and QT prolongation as well as T-wave inversion.[7]

Hypothermia-related peripheral vasoconstriction causes cold diuresis, which in turn leads to increased kidney perfusion and the loss of the kidney's ability to concentrate fluids, ultimately resulting in hyperosmolar hypernatremia and oliguria with ensuing hypovolemia.[10] As the kidney starts to fail, acid-base disturbance causes respiratory alkalosis, which progresses to metabolic acidosis and associated hyperkalemia.

As the metabolic rate decreases, hypothermia causes reductions in oxygen consumption and carbon dioxide production. A left shift of the oxyhemoglobin dissociation curve with subsequent oxygen delivery impairment and tissue hypoxia is also seen; however, the acidosis and decreased rate of tissue metabolism due to hypothermia compensate for this effect.[10]

In trauma patients, hypothermia is associated with coagulopathy,[5,13,16] which is a result of impaired platelet function and thrombocytopenia caused by extravascular sequestration of platelets. Furthermore, as body temperature decreases, coagulation cascade enzymes tend to lose their function. Hypothermia-related coagulopathy cannot be measured by available laboratory tests, as these are performed after heating blood to room temperature. Clinically, coagulopathy might manifest with either bleeding or a hypercoagulable state and thrombus formation.

Interestingly, in severe hypothermia, paradoxical undressing may be observed. Although the cause of this phenomenon is not clearly understood, it is thought that cutaneous vascular disturbances result in a false sensation of warmth.[17] Undressing leads to further heat loss and progression of hypothermia. Also, in severe hypothermia, the level of consciousness progressively deteriorates to coma and loss of electroencephalographic activity, which may lead to terminal primitive behaviors (eg, the hide-and-die syndrome). This may explain why some patients who die from hypothermia are found in hidden positions.[17]

EVALUATION OF THE PATIENT

Managing hypothermic patients can be a challenge because of the absence of a reliable history in most cases. Initially, core body temperature should not be measured with a standard thermometer, which cannot measure temperatures lower than 34.4°C (94°F).[1] Pulmonary artery probe is the most reliable method for measuring core temperature in patients with hypothermia, but it is invasive. Other methods include tympanic membrane probes, rectal probes, and bladder probes. After the diagnosis of hypothermia is made, work-up is individualized based on the suspected cause of hypothermia and the presenting symptoms. Initial laboratory tests should include complete blood count, blood glucose, serum electrolytes, blood urea nitrogen, serum creatinine, arterial blood gas studies, ECG, alcohol level, and toxicology screening, especially in the absence of a reliable history.[3] Other tests to be considered are evaluation for sepsis, endocrine studies, and a coagulation profile. Blood tests might need to be repeated at intervals. Although laboratory results vary with changing temperature, patients with hypothermia should be managed based on results obtained at 37°C, as this is the temperature at which blood testing is conducted in medical laboratories.[16,18]

MANAGEMENT

Management should be guided by the cause and degree of hypothermia and coexisting conditions. Initially, patients should be rewarmed while looking for a potential cause. If the etiology cannot be determined from

Table 3. Methods for Rewarming Hypothermic Patients

Method	Uses/Advantages	Disadvantages
Passive external rewarming (eg, blankets, humidified heated oxygen by mask)	Noninvasive Useful for mild hypothermia in a previously healthy person Intense monitoring is not needed	A slow process
Active external rewarming (eg, forced warmed air, radiant heat, electric/plumbed heat blankets)	Useful in mild to moderate hypothermia with cardiovascular stability Can be combined with passive external rewarming	May cause iatrogenic body surface burns Core temperature afterdrop may develop
Active internal rewarming (eg, heated humidified air, warmed intravenous fluids, peritoneal dialysis, closed thoracic lavage, extracorporeal blood rewarming)	Most effective modality Fastest modality Can be used in patients with stable or compromised cardiovascular system	Can be invasive Patient needs to be intensively monitored

the patient's presentation, the standard measures for coma patients can be applied (ie, basic airway, breathing, and circulation assessment). In addition, a dextrose solution infusion with thiamine and, if drug overdose is suspected, medications such as naloxone (for opium overdose) or flumezanil (for benzodiazepine overdose) should be considered. Complications can be prevented by avoiding procedures such as nasogastric tube insertion and endotracheal intubation when possible, as these procedures can precipitate ventricular fibrillation. When required, securing the airways and endotracheal intubation are performed using the same guidelines as in nonhypothermic patients. Oxygen therapy may be considered empirically.[7,14]

Doppler ultrasonography should be used to check pulses. If this is unavailable, palpate for pulses for 35 to 45 seconds before announcing cardiac arrest as recommended by American Heart Association guidelines.[19] If pulses are present but blood pressure is low, fluid resuscitation is preferred over pressors. Cardiac arrest in hypothermic patients, whether due to asystole or ventricular fibrillation, might be resistant to medical therapy or defibrillation until the patient has been rewarmed.[14] Nonetheless, advanced cardiac life support protocols should be followed initially and, if no response is noted, they should be continued until the patient's core body temperature is above 32°C. Resuscitative efforts have been successful with temperatures as low as 13.7°C.[20] Furthermore, survivors of hypothermia tend not to have the usual adverse hypoxic neurologic sequelae associated with prolonged resuscitation.[18]

Rewarming

Rewarming can be achieved by different means (**Table 3**), the choice of which varies based on predisposing factors, degree of hypothermia, and cardiovascular instability.[1,7,8,21–23] Passive external rewarming is used in patients with mild hypothermia without cardiovascular instability. This is a slow process that involves adequate insulation and provision of a warm environment. Passive external rewarming (eg, blankets, humidified heated oxygen by mask) is successful when thermoregulatory mechanisms and endocrine functions are intact.

Active external rewarming combined with passive external rewarming is preferred in patients with mild to moderate hypothermia without cardiovascular compromise. Heat is applied directly to the skin by way of forced warmed air, radiant heat, or electric/plumbed heat blankets. Of these, forced air is the most effective.[11] The efficiency of radiant heat or electric/plumbed heat blankets varies, as skin perfusion decreases from 200 mL/min/m^2 to 4 mL/min/m^2 in hypothermic patients due to vasoconstriction.[10] Another limitation of using heating blankets is that they do not cover all areas of the body, and therefore heat is exchanged through less than 25% of the total body surface area.

Active internal rewarming is the most effective and the fastest modality and is used in cases of moderate to severe hypothermia. In patients without cardiovascular compromise, heated humidified air, warmed intravenous fluids, peritoneal dialysis, and closed thoracic lavage can be used. In patients with cardiovascular compromise, extracorporeal blood rewarming is the modality of choice. This modality raises body temperature by 1°C to 2°C every 5 minutes and can be achieved by using either femoro-femoral bypass or the standard aorto-right atrial bypass. Femoro-femoral bypass is preferred, as it can be established in the emergency department and does not interfere with chest compressions if resuscitation is needed.[21]

During rewarming, some complications should be

anticipated. During external rewarming, rapid resolution of peripheral vasoconstriction may cause the patient's core temperature to drop (ie, core temperature afterdrop). This effect can be minimized by using minimally invasive active internal rewarming prior to active external rewarming. Another complication related to peripheral rewarming is rewarming shock, which should be managed with fluid resuscitation. Finally, rewarming acidosis occurs as blood flushes lactic acid from hypoxemic peripheries.

Terminating Resuscitative Efforts

Some believe that patients who appear to be dead after prolonged exposure to cold temperatures should not be considered dead until they are rewarmed to a near-normal core temperature.[23,24] Rewarming efforts should be continued until the core temperature is at least 32°C. Efforts may be discontinued if the patient continues to show no effective cardiac rhythm and remains unresponsive to all resuscitative measures.[25] However, the decision to terminate resuscitative measures should be based on the unique circumstances of each case.[25]

CONCLUSION

Clinicians should be aware of the clinical signs and symptoms of hypothermia. Documentation of core body temperature using special thermometers is required to diagnose and appropriately classify hypothermia. Active internal rewarming is the most effective method for treating hypothermic patients, but it is invasive. Therefore, passive rewarming and active external rewarming methods should be used in less severe cases of hypothermia in the absence of cardiovascular instability. With proper management, a good outcome can be achieved, even in cases of severe hypothermia. **HP**

Test your knowledge and comprehension of this article with the *Clinical Review Quiz* on page 22.

REFERENCES

1. McCullough L, Arora S. Diagnosis and treatment of hypothermia. Am Fam Physician 2004;70:2325–32.
2. Hayward JS, Eckerson JD, Kemna D. Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. Resuscitation 1984;11:21–33.
3. Hypothermia-related deaths—Philadelphia, 2001, and United States, 1999. Centers for Disease Control and Prevention (CDC). MMWR Morb Mortal Wkly Rep 2003; 52:86–7.
4. Aslam AF, Aslam AK, Vasavada BC, et al. Hypothermia: evaluation, electrocardiographic manifestations, and management. Am J Med 2006;119:297–301.
5. Hildebrand F, Giannoudis PV, van Griensven M, et al. Pathophysiologic changes and effects of hypothermia on outcome in elective surgery and trauma patients. Am J Surg 2004;187:363–71.
6. Ganong WF. Review of medical physiology. 22nd ed. New York: McGraw-Hill Medical; 2005:253–5.
7. Wilkey SA. Accidental hypothermia: a case report and focused review. Am J Clin Med 2004;1:4–11.
8. Danzl DF, Pozos RS. Accidental hypothermia. N Engl J Med 1994;331:1756–60.
9. Taguchi A, Kurz A. Thermal management of the patient: where does the patient lose and/or gain temperature? Curr Opin Anaesthesiol 2005;18:632–9.
10. Mallet ML. Pathophysiology of accidental hypothermia. QJM 2002;95:775–85.
11. Sessler DI. Mild perioperative hypothermia. N Engl J Med 1997;336:1730–7.
12. Fos LA, Magonet GM, Guidry AF, Judson K. Case study: hypothermia secondary to erythroderma: FP-2. South Med J 2005;98:S51.
13. Soreide E, Smith CE. Hypothermia in trauma victims—friend or foe? Indian J Crit Care Med 2004;8:116–9.
14. Kempainen RR, Brunette DD. The evaluation and management of accidental hypothermia. Respir Care 2004; 49:192–205.
15. Saadlla H. Images in clinical medicine. Electrocardiographic changes in extreme hypothermia. N Engl J Med 2004;351:e4.
16. Gentilello LM, Pierson DJ. Trauma critical care. Am J Respir Crit Care Med 2001;163(3 Pt 1):604–7.
17. Rothschild MA. Lethal hypothermia: paradoxical undressing and hide-and-die syndrome can produce obscure death scene. Forensic Pathol Rev 2004;1:263–72.
18. Walpoth BH, Walpoth-Aslan BN, Mattle HP, et al. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. N Engl J Med 1997;337:1500–5.
19. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 10.4: hypothermia. Circulation 2005;112 (Suppl 1):I–211.
20. Gilbert M, Busund R, Skagseth A, et al. Resuscitation from accidental hypothermia of 13.7 degrees C with circulatory arrest. Lancet 2000;355:375–6.
21. Lazar HL. The treatment of hypothermia. N Engl J Med 1997;337:1545–7.
22. Sessler DI. Complications and treatment of mild hypothermia. Anesthesiology 2001;95:531–43.
23. Steinman AM. Cardiopulmonary resuscitation and hypothermia. Circulation 1986;74(6 Pt 2):IV29–32.
24. Southwick FS, Dalglish PH Jr. Recovery after prolonged asystolic cardiac arrest in profound hypothermia. A case report and literature review. JAMA 1980;243:1250–3.
25. Weinberg AD. Hypothermia. Ann Emerg Med 1993;22 (2 Pt 2):370–7.