

Clinical Aspects of Hypothermia

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Abstract

Hypothermia is generally defined as a core body temperature less than 35°C (95°F). It is one of the most common environmental emergencies encountered by emergency physicians and was documented as a special resuscitation situation in advanced cardiac life support (ACLS) and advanced trauma life support (ALTS) guidelines on cardiopulmonary resuscitation and emergency cardiac care. This condition is found in varied geographic regions and during all seasons. Although cold exposure is likely the most common cause of hypothermia in emergency department patients, there are many other predisposing factors as well. This article reviews the etiology, pathophysiology, clinical presentation, and management of hypothermia. (*Ann Disaster Med.* 2004;2 Suppl 2:S69-S79)

Key words: Hypothermia; Rewarming Methods; Resuscitation

Introduction

The human body functions optimally with a core body temperature between 36.4°C and 37.5°C (97.5 to 99.5°F) and hypothermia is generally defined as a core body temperature less than 35°C (95°F). Primary hypothermia (accidental hypothermia) refers to a spontaneous reduction of core temperature as a result of exposure to cold environments without adequate protection, while secondary hypothermia represents a complication of an underlying disorder.¹

Body temperature is closely regulated through a balance between heat production and heat dissipation. Approximately 90% of heat lost through the skin by radiation (non-particu-

late emission of heat from body), evaporation (cooling by conversion of fluid to vapor), conduction (transfer of heat by direct contact) or convection (transmission of heat by movement of heated particles), with the remainder lost via the lungs by respiration.²

Etiology

The exposure to cold increases activity in the afferent fibers from cold receptors, located peripherally on the skin and centrally along the great vessels, abdominal viscera and spinal cord, which stimulate the pre-optic nucleus of the anterior hypothalamus. Direct reflex vasoconstriction reduces blood flow to the cooling skin, and colder blood also reaches tempera-

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ture-sensitive neurons in the hypothalamus. The hypothalamus then initiates immediate responses via the autonomic nervous system that stimulate vasoconstriction of the peripheral and cutaneous blood vessels, delayed responses through the endocrine system, adaptive behavioral responses, extra-pyramidal skeletal muscle stimulation and shivering.³ These responses aim either to increase heat production or reduce heat loss.

Elderly are particularly susceptible to hypothermia because thermoregulatory ability is progressively impaired with age. They may

have a reduced ability to generate heat because of reduced lean body mass, malnutrition, immobilization, and reduced shivering in response to cold. Moreover, diminished ability to sense external temperature changes, inability to vasoconstrict appropriately, or abnormal adaptive behavioral responses may result in increased heat loss of the elderly.⁴ In addition to the age-related impairment of adaptability to a fall in temperature, many underlying conditions increase a person's susceptibility to hypothermia are listed in Table 1.²⁻⁵

The risk of mortality from hypothermia in-

Table 1. Factors that increase susceptibility to hypothermia

Mechanism	Clinical disorders
Decreased heat production	Insufficient fuel - malnutrition, hypoglycemia Neuromuscular inefficiency - extremes of age (infant or elderly), immobilization, impaired shivering Endocrinologic failure - hypopituitarism, hypoaldosteronism, hypothyroidism, myxedema,
Increased heat loss	Environmental exposure - homelessness, poverty, wilderness exposure, immersion, high altitude, trauma causing immobility, Skin disorders - burns, psoriasis, exfoliative dermatitis, Induced vasodilation - alcohol, lithium toxicity, toxins Iatrogenic - cold intravenous infusion, emergent deliveries
Impaired thermoregulation	CNS pathology - trauma, stroke, hemorrhage, subarachnoid hemorrhage, Parkinson's disease, Wernicke's encephalopathy, multiple sclerosis, neoplasms, hypothalamic dysfunction Drugs - barbiturates, benzodiazepines, opioids, phenothiazines, tricyclic antidepressants, antimanic agents, alcohol Peripheral failure - neuropathies, spinal cord transections, diabetes
Miscellaneous	Sepsis, pancreatitis, diabetic ketoacidosis, uremia, carcinomatosis, vascular insufficiency, multi-system trauma, anorexia nervosa

creases with age. People older than 75 years old are five times as likely to die from hypothermia as those younger than 75 years old.⁴ Also the presences of hypothermia in trauma patients or sepsis were associated with a higher

mortality rates.^{3,6}

Pathophysiology

Hypothermia can have marked physiologic effects on the body's vital organ systems. They

Table 2. Physiologic changes and clinical manifestations associated with hypothermia

Systems	Mild hypothermia	Moderate hypothermia	Severe hypothermia
Cardiovascular	↑heart rate, ↑cardiac output, ↑systemic vascular resistance, ↑blood pressure	↓heart rate, ↓cardiac output, ↓blood pressure,	↓blood pressure, ↓cardiac output, ↓heart rate
	- prolong PR and QT intervals - sinus rhythm predominate	- J (Osborn) wave, - Junctional rhythm, atrial and ventricular arrhythmia	- Ventricular arrhythmias, - heart block, - pulseless electrical activity, - Asystole at <24°C
Neurologic	- Confusion, amnesia, dysarthria, - ataxia, - hyperreflexia ↓cerebral metabolism	- lethargy, - pupils dilate - hallucinations - hyporeflexia - EEG abnormalities	- coma - loss of cerebrovascular regulation, - loss of ocular reflex - areflexia - decline in EEG activity and silent at < 26°C
Respiratory	↑respiratory rate, ↑minute ventilation, - bronchorrhea	↓respiratory rate, ↓minute ventilation, ↓oxygen consumption and CO ₂ production, - atelectasis - loss of cough reflex and airway protection	- pulmonary edema, - acute respiratory distress syndrome, - apnea at < 24°C
Metabolic/endocrine	↑metabolic rate, ↑catecholamines, - hyperglycemia	↓metabolic rate, - hyper/hypoglycemia	- progressive ↓to 20% of basal metabolic rate - hyper/hypoglycemia
Renal/electrolytes	- cold diuresis, - bladder atony	- cold diuresis - hyperkalemia, - lactic acidosis	↓renal perfusion, ↓glomerular filtration rate, - oliguria - hyperkalemia - lactic acidosis
Hematologic	↑hematocrit (hemoconcentration), ↓platelet count and function, ↓white blood cell count, ↓enzyme function in coagulation cascade, - coagulopathy - disseminated intravascular coagulation		
Gastrointestinal	- ileus, pancreatitis, gastric stress ulcers, hepatic dysfunction		
Musculoskeletal	- hypertonia	- rigidity	- rhabdomyolysis, - 'pseudo-rigor mortis'
Thermoregulatory	- Shivering intact	- Shivering lost, rapid cooling	

are varied with the severity of hypothermia, which is classified based on the core body temperature as mild (35°C - 32.2°C), moderate ($<32.2^{\circ}\text{C}$ - 28°C), and severe ($<28^{\circ}\text{C}$).² Because the prognosis associated with hypothermia in the trauma patient is so poor, a separate classification of hypothermia has been developed for the trauma patient that is classified as mild (36°C - 34°C), moderate ($<34^{\circ}\text{C}$ - 32°C), and severe (<32).^{6,7} Increasing severity of hypothermia produces a predictable pattern of organ dysfunction and associated clinical manifestations are summarized in Table 2.

Cardiovascular Manifestations

The initial effect of hypothermia is a sympathetic response that causes tachycardia, peripheral vasoconstriction, and a consequent increase in cardiac output, blood pressure and myocardial oxygen consumption. As the temperature fall to moderate hypothermia, a progressive bradycardia will be developed and decreased spontaneous depolarization of the pacemaker cells makes atropine ineffective. Mean arterial pressure and myocardial contractility and cardiac output fall dramatically at lower temperatures.

Hypothermia is associated with various atrial and ventricular dysrhythmias. At mild hypothermia, sinus rhythm predominates.⁸ Decreased AV conduction velocity often causes sinus bradycardia. With progressive hypothermia, junctional rhythms and atrial arrhythmia may occur. More than 50% of patients with moderate hypothermia develop atrial fibrillation with a slow ventricular response.⁹ As the core temperature fall below 30°C , there is increased myocardial irritability and ectopic ventricular beat are common. Patients are at high risk for development of ventricular fibrilla-

tion that is refractory to electrical cardioversion. At temperature less than 24°C , the risk of asystole increases significantly.

Electrocardiographic (ECG) findings are nonspecific but include prolonged PR, QRS, and QT intervals, and a classic "J" (Osborn) wave. J wave appears as a positive deflection in the terminal portion of the QRS complex, usually best seen in the lateral precordial leads and tends to increase in amplitude with falling temperature.⁹ J wave is not diagnostic of hypothermia, but can also seen in subarachnoid hemorrhage, other cerebral injuries and myocardial ischemia.³

Central Nervous System Manifestations

Mild hypothermia may be associated with confusion, dysarthria, impaired judgement and memory. As the temperature falls farther, progressive depression of consciousness and ultimately coma develop below 30°C . The cerebrovascular autoregulation is maintained until 25°C and cerebral blood flow and metabolism decrease 7% for each degree decline in temperature.³

The electroencephalogram (EEG) becomes abnormal below 34°C and silent below 26°C .¹⁰ Ataxia and loss of fine motor control are seen in mild hypothermia; hyporeflexia, an extensor plantar response and pupillary sluggishness occurred in moderate hypothermia; rigidity, loss of pupillary reflex and ocular reflex and areflexia appeared as the temperature falls below 28°C . Therefore, one has to be very careful not to declare a hypothermic patient brain dead before rewarming the patient and re-evaluating the condition thereafter.

Respiratory Manifestations

In mild hypothermia, there is an initial tachypnea, followed by a reduction in minute volume and oxygen consumption; bronchospasm and bronchorrhea may occur. In moderate hypothermia, respiratory rates fall and the associated hypoventilation and carbon dioxide retention produce hypoxia and respiratory acidosis. There is also loss of the protective airway reflexes because of the impairment of ciliary function, and this predisposes to aspiration pneumonia.¹¹ Local gas exchange is not affected by hypothermia, but there is an increase in pulmonary vascular resistance and a degree of ventilation-perfusion mismatch in the lungs. In severe hypothermia, progressive hypoventilation, apnea, pulmonary edema and acute respiratory distress syndrome may also occur.³

Metabolic and Endocrine Manifestations

Total body metabolism reduces with increasing hypothermia, as measured by a fall in oxygen consumption, which is about 6% for every degree Celsius fall in temperature.¹² Pituitary, adrenal and thyroid function are thought to be normal but should be measured to exclude them as the underlying causes. In the initial of hypothermia, sympathetic activity is increased, with raised plasma norepinephrine and free fatty acid levels, and the catecholamine-induced glycogenolysis and gluconeogenesis contribute to the hyperglycemia.

In addition, peripheral uptake of insulin at the tissues is impaired and insulin release is inhibited by increased corticosteroid level and direct cooling effect on the islets of Langerhans, result in hyperglycemia.³ When hypothermia is

long lasting, glycogen stores may be depleted and hypoglycemia will develop.

Renal Manifestation

In patient with mild hypothermia, cold diuresis occurs that may be the results of increase in renal blood flow consequent on peripheral vasoconstriction, loss of distal tubular ability to reabsorb water and resistance to the action of vasopressin (ADH). In severe hypothermia, glomerular filtration rate falls as cardiac output and hence renal blood flow fall and oliguria may develop. About 40% of patient will develop acute renal failure in severe hypothermia, especially in patients with acute tubular necrosis secondary to rhabdomyolysis.¹³

Plasma sodium, calcium, magnesium and chloride concentrations do not change significantly above 25°C. Hyperkalemia may be seen in severe hypothermia and often associated with metabolic acidosis, rhabdomyolysis, renal failure and cell death. Therefore, it is an indicator of poor prognosis.³

Hematologic Manifestations

With fall in core temperature, the increased vascular permeability and vasoconstriction result in the loss of plasma to extravascular compartments, leading to blood viscosity and hematocrit (hemoconcentration) increase. The hematocrit increases by about 2% for every 1°C decline in temperature.¹⁴ Hypothermia impairs coagulation by directly inhibiting the enzymatic reactions of both intrinsic and extrinsic pathways of the clotting cascades.¹⁵ A small drop in temperature to 34°C can decrease up to 40% of the enzymatic activity of the coagulation factors. The prothrombin and partial prothromboplastin time can be deceptively nor-

mal if measured at 37°C but can be significantly prolonged if measured at lower temperature. Therefore rewarming, rather than administration of exogenous clotting factors, is the appropriate management.

Hypothermia directly affects platelet function by decreasing thromboxane B2 production and platelet surface molecules expression. Thrombocytopenia may also result from sequestration in the liver and spleen, bone marrow suppression or disseminated intravascular coagulation.¹⁶ Fibrinolysis is also enhanced in hypothermic animals as a result of impaired inhibitors of clot lysis, such as plasminogen activator inhibitor or alpha-2-antiplasmin.¹⁷

Hypothermia can result in granulocytopenia. In animal and vitro studies, neutrophil migration and bacterial phagocytosis are impaired, predisposing to infection. These effects have not been demonstrated in human.³

Gastrointestinal Manifestations

Temperature below 34°C will slow intestinal motility. An ileus will be developed when temperature falls below 28°C. Therefore a nasogastric tube should be placed to reduce the risk of aspiration. The absorption of drugs given orally or by nasogastric tube will also be impaired in this situation.³ Animal study has shown that hypothermia increases gastric acid production and reduces duodenal bicarbonate secretion, predisposing to the mucosal damage in both the stomach and duodenum.¹⁸ Autopsy studies have found gastric erosions and submucosal hemorrhage to be common but not clinically significant.

Hepatic impairments from reduced cardiac output and the decreased clearance of lactic acid contributes to metabolic acidosis. The

liver functions of detoxification and conjugation are also impaired in hypothermia, causing reduced clearance of many drugs and toxins. Pancreatitis may also be developed, being found at autopsy in 20-30% of cases.³

Musculoskeletal Manifestations

Shivering in mild hypothermia can increase total metabolism and basal metabolic rate two to five times normal.¹ The shivering activity is lost in moderate and severe hypothermia. Synovial fluid becomes more viscous at lower temperature, so stiffness of muscles and joints will be developed in moderate hypothermia. In severe hypothermia, muscle and joint stiffness may simulate rigor mortis.

Managements

Initial stabilization

Moderate to severe hypothermia is a medical emergency necessitating maintenance of airway, breathing and circulation. The core body temperature should be monitored by esophageal, bladder or rectal routes.⁵

Standard ACLS and ATLS protocols should be initiated, including spine immobilization if trauma is suspected. Supplemental oxygen should be given empirically. Endotracheal intubation is indicated if apnea, coma, or loss of airway reflexes occurred. Orotracheal intubation is preferred because hypothermic patients are coagulopathic and prone to traumatic nasal bleeding via nasal route. Neuromuscular blocking agents should be avoided because they are ineffective when core temperature below 30°C. Impaired renal, hepatic and plasma enzyme function make metabolism and clearance unpredictable.²

Cardiopulmonary resuscitation (CPR)

should be instituted if the patient is not breathing or pulse is absent. Defibrillation should be attempted for ventricular fibrillation or ventricular fibrillation or pulseless ventricular tachycardia, although attempts may be unsuccessful at temperature below 30°C. If initial defibrillation is unsuccessful, initiate rewarming and reattempt defibrillation every 1-2°C increase in core temperature. In general, all resuscitation medications should be withheld until core temperature higher than 30°C. Most hypothermia induced dysrhythmias convert spontaneously with rewarming and transvenous pacing is not recommended, as it may precipitate ventricular dysrhythmias. The use of vasopressor agents in moderate or severe hypothermic patient with hypotension should also be avoided owing to these agents have minimal effect on already constricted vessels and may induce dysrhythmias.

Patient with moderate and severe hypothermia are volume depleted and required intravenous fluids throughout the warming process. Crystalloids should be warmed to 40-42°C in a microwave or commercial fluid warmers to avoid exacerbating heat loss. Lactated Ringer's solution should be avoided because of decreased ability of the liver to metabolize lactate.¹ Arterial catheters provide continuous blood pressure and arterial blood gas monitoring. Pulmonary artery catheterization and central venous pressure monitoring through internal jugular or subclavian arteries catheterization should also be avoided owing to potential dysrhythmias and the risk of vascular perforation.

Moreover nasogastric or orogastric tube should be placed to relieve gastric distention and ileus. A urinary catheter is also essential to monitor urine output and assess volume resus-

citation efforts. Wet clothes, contributing to conductive heat loss, should be removed and the skin should be kept dry and covered with insulating materials. Empirical antibiotics are appropriate for patients with suspected aspiration or sepsis. The patient should also be handled gently because movement and manipulation may precipitate dysrhythmias.

Rewarming methods. Rewarming is the primary treatment for hypothermia. It may be passive external, active external or active internal and which methods should be chosen depend on the patient's condition, hypothermia severity or institutional expertise and capability.

Passive external rewarming. Passive external rewarming consists of optimizing environmental conditions while allowing the patient's own heat generating capabilities to rewarm core temperature. This includes removal of the patient from the cold environment, protection against wind chill, removal of wet clothing, and insulation of patient in a warm environment. The warming rate of this method may be 0.5-2°C per hour depending on the shivering thermogenesis of the patient.¹

Active external rewarming. This involves direct exposure of the patient to exogenous heat sources, such as immersion in a 40°C bath, warming blankets, heating pads, radiant heat and forced warmed air. The warming rate of this method may be 1-2.5°C per hour.¹⁹ Peripheral vasoconstriction makes the skin of hypothermic patients especially vulnerable to burn injuries from externally applied heat sources.

Active internal rewarming. Active core rewarming methods are indicated for any patient with severe hypothermia. Heated humidified air or oxygen, up to 45°C via endotracheal tube and the administering warm intravenous fluids

(heated to 40-42°C in a microwave or commercial fluid warmers) will raise the core temperature 1-2.5°C per hour.²⁰

Pleural cavity lavage can be performed by infusing sterile saline up to 42°C through a thoracostomy tube placed in the 2nd or 3rd anterior intercostal space in the midclavicular line. The fluid is drained via a second thoracostomy tube in the 4th, 5th or 6th intercostal space in the posterior axillary line.²¹ Alternatively, warm saline can be repeatedly infused and drained through a single chest tube, using a 15-20 minutes dwell time.² Mediastinal irrigation and myocardial lavage could be considered in patients who have severe hypothermia and no spontaneous perfusion. The warming rate of this method may be 2-4°C per hour.

Peritoneal lavage by infusing sterile saline up to 45°C through two or more catheters in the intraperitoneal space with a flow rate of 6L/h. Direct irrigation of the liver can accelerate the recovery of hepatic function and facilitate the clearance of toxins and lactic acidosis. When warm dialysate is used, it allows the removal of dialyzable toxins and treatment of concomitant renal failure or rhabdomyolysis.²² The warming rate of this method may be 2-4°C per hour.

Irrigation of the stomach, bladder or colon has limited utility because the surface area available for heat transfer is minimal. Moreover, gastric lavage may predispose to aspiration and must be discontinued during chest compression.

Extracorporeal circulatory rewarming provides infusion of warm intravenous fluid and recirculation of the patient's blood. It includes hemodialysis, arteriovenous rewarming, venovenous rewarming, and cardiopulmonary bypass and usually reserved for the critical hypothermic patient.

Cardiopulmonary bypass can provide very rapid rewarming (7-10°C per hour), circulatory support, oxygenation and can be combined with hemodialysis for the treatment of renal failure. However, it has some drawbacks such as considerable time to institute, unavailable in all institutions, and requiring systemic anticoagulation that is contraindicated in trauma patients.

Continuous arteriovenous rewarming can provides rapid rewarming (3-4°C per hour), and can be more rapidly initiated. It requires less specialized equipments and personnel to operate as compared with cardiopulmonary bypass. Also heparin-coated arterial and venous catheters are available and additional systemic anticoagulation is not necessary. However, it may require adequate blood pressure of the patient and cannot oxygenate or dialyze blood.

Hemodialysis and hemofiltration are widely available, rapid initiation and useful in the setting of renal insufficiency, electrolyte abnormalities, volume overload or following ingestion of a dialyzable toxin.²³ The warming rate may be 2-3°C per hour.

Conclusion

Hypothermia can be found in varied geographic regions and during all seasons. Prompt recognition of the clinical presentation, advanced knowledge of the pathophysiology and institution of appropriate management strategies are imperative for a successful outcome with minimal complications. The prognosis of hypothermia is related to age, preexisting illness, nutritional status, precipitating events, duration and severity of cold exposure.²⁴ All hypothermic patients should be rewarmed to at least 35°C

before declaring futility and withdrawing support. Prevention of hypothermia through patient education and provision of shelter to at-risk individuals remains an important public health strategies.

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低體溫

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摘要

低體溫的定義為中心體溫低於攝氏 35 度或華氏 95 度，它是常見的環境醫學急症之一，並且在高級心臟救命術(ACLS)及高級傷救命術(ATLS)的指引中，亦加入低體溫這特殊情景的急救及照護，低體溫可發生於任何地區及任何季節，雖然在急診室遇到的低體溫病患，大部份為環境暴露所引起，但還有一部份為其他誘發因素所致，本篇主要針對低體溫的誘發因子、病生理學，臨床症狀及處理作回顧及探討。(Ann Disaster Med. 2004;2 Suppl 2:S69-S79)

關鍵詞：低體溫；回溫的方法；復甦術

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