Deadly Degrees
Don't Discount These Diagnoses

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KEYWORDS

• Temperature • Hypothermia • Hyperthermia • Emergency medicine

KEY POINTS

- Accurate temperature measurement is an essential component of any emergency department encounter.
- A patient's temperature has significant impact on workup as well as diagnostic implications.
- Nonenvironmental causes of temperature derangement should be considered.

INTRODUCTION

Temperature is considered one of the core vital signs, checked within minutes of presentation to the emergency department (ED) as part of the triage process. This routine assessment can have significant clinical implications, informing Emergency Severity Index and subsequent diagnostic workup. Both hyperthermia and hypothermia can indicate life-threatening conditions. Accurate temperature measurement is essential to avoid overlooking critical diagnoses.

TEMPERATURE MEASUREMENT

There are numerous methods for measuring body temperature in the clinical setting. The most accurate, but highly invasive measurement is a pulmonary artery catheter temperature. Esophageal probes, when properly placed, are close to the left ventricle and aorta, which supplies blood to the hypothalamic temperature regulation system (HTRS). Esophageal probes can cause physical discomfort, and if improperly placed, may lead to inaccurate temperature readings, as not all portions of the esophagus have the same temperature.[1] When the bladder is continuously drained, as in the case of a temperature-sensing urinary catheter, bladder temperatures can be quite accurate.[2] Rectal temperature measurements are practical, but temperatures in this

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Abbreviations	
Bpm	beats per minute
CNS	central nervous system
ECG	electrocardiogram
ED	emergency department
HTRS	hypothalamic temperature regulation system
IDSA	Infectious Disease Society of America
MI	myocardial infarction
NCIT	noncontact infrared thermometry
NMS	neuroleptic malignant syndrome
SS	serotonin syndrome
TBI	traumatic brain injury

area may take some time to equilibrate with central temperatures, as rectal vasculature is peripheral.[1] Logically, a large rectal stool burden can limit accuracy.

The most used and consistently reliable noninvasive method is oral temperature measurement with a thermometer placed in the sublingual pocket. Oral temperature measurements may, however, be inaccurate because of an inability to close the mouth, rapid breathing through the mouth, or recent consumption or hot or cold substances. Tympanic membrane temperatures should, in theory, be a highly accurate noninvasive measurement, as this location is directly supplied with blood from the internal carotid artery that also supplies the HTRS. Practically, inserting a probe into the ear is fraught with user error and limited in accuracy by cerumen. Axillary temperature measurements are not preferred, as measurements take longer and are inaccurate, typically with falsely low readings.[2] During the COVID-19 pandemic, noncontact infrared thermometry (NCIT) was popularized as a screening method for fever in clinical and nonclinical environments (eg, airports). NCIT technology should not be used to inform clinical decisions.[3]

EFFECT OF TEMPERATURE ON OTHER VITAL SIGNS

Heart Rate

The association between temperature and heart rate has been well studied. In the late 1800s, it was discovered that for each degree in Celsius above 38.3°C, there is expected to be an increase in heart rate by approximately 10 beats per minute (bpm; **Table 1**).[4]

In contrast, as patients' body temperatures decrease, there is an increasing trend toward bradycardia. Cardiac arrhythmias can be induced by either hyperthermia or hypothermia. With mild hypothermia, an initial sympathetic response occurs, resulting in tachycardia. As hypothermia worsens, the compensatory mechanisms become

Table 1
Expected heart rate for temperature

Temperature	Expected Heart Rate
38.3°C (100.9°F)	110 bpm
39.3°C (102.7°F)	120 bpm
40.3°C (104.5°F)	120 bpm
41.3°C (106.3°F)	140 bpm

If heart rate is higher than expected for temperature, this is considered relative tachycardia. If heart rate is lower than expected for temperature, this is considered relative bradycardia.

depleted, and cardiopulmonary systems slow. Relative bradycardia, a dissociation of the temperature-pulse response, can occur in certain hyperthermic conditions and is known as the Faget sign. This is nonspecific but can be sensitive as a sign of infection owing to an intracellular organism. Infectious conditions that are most associated with relative bradycardia include typhoid fever, Q fever, chlamydial infection, tick-borne infection (anaplasmosis, ehrlichiosis, babesiosis, Lyme), parasitic infections (malaria, Chagas), and spirochetal infections (leptospirosis).[4] Noninfectious causes of relative bradycardia include lymphoma, drug-induced fever, and adrenal insufficiency.[4] Central nervous system (CNS) infections, including brain abscess, can also be associated with relative bradycardia.[5]

Relative tachycardia is when the heart rate is greater than 10 bpm for each degree in Celsius above 38.3°C. The pathophysiology for this is incompletely understood but thought to be due to excessive catecholamine surge. Relative tachycardia has been recognized as a clinical indicator for myocarditis, although it remains nonspecific.[6] It is also considered a poor prognostic factor in patients, particularly those who are critically ill.[7,8]

Oxygen Saturation

The inability to obtain an accurate, pulsatile, pulse oximetry reading on the distal digits when evaluating an undifferentiated critically ill patient could suggest reduced core temperature. Hypothermia causes vasoconstriction and can cause poor distal perfusion, reducing the accuracy of pulse oximetry. Using the earlobe or forehead site, opposed to fingertip, may allow for more accurate readings.[9–11]

Blood Pressure

Exposure to cold temperatures most commonly results in vasoconstriction, which can cause an increase in blood pressure. Extreme hypothermia can lead to hypotension, arrhythmia, and cardiovascular collapse. Interestingly, the blood pressure of patients with cardiovascular disease is more affected by a temperature decrease.[12,13]

Respiratory Rate

Respiratory rate tends to increase during hyperthermia and fever; however, this association is not as clearly established as that between heart rate and temperature.[14,15] This correlation is less commonly observed in children.[16]

EFFECT OF TEMPERATURE ON DIAGNOSTIC TESTS

Most diagnostic tests are not altered by body temperature. Blood-gas measurements should be corrected for temperature considering the partial pressure of gases (including oxygen and carbon dioxide) is proportional to a given temperature and atmospheric pressure. Typically, blood-gas analyzers heat or cool a patient's blood sample to conduct an analysis at 37°C. The analyzer will then report this standardized result, as well as a calculation based on the patient's reported temperature. At any given constant atmospheric pressure, it is expected that a hypothermic patient would have less oxygen and carbon dioxide dissolved in the blood than a normothermic patient. Consequently, pH is expected to be higher, and oxygen and carbon dioxide levels are expected to be lower. The opposite holds true for the hyperthermic patient.[17]

Electrocardiogram (ECG) parameters also change based on body temperature. Shivering artifact may affect ECG interpretation. In extreme hypothermia, upward deflections at the end of the QRS complex called "J waves/Osborn waves" may be

appreciated (Fig. 1).[18] In extreme hyperthermia, hyperkalemia can occur with corresponding ECG changes. A Brugada pattern may become apparent on an ECG when a patient is febrile and may resolve when afebrile.[19] The Brugada pattern is characterized by a 2-mm J-point elevation and a 1-mm ST-segment elevation in at least 2 precordial leads. The ST changes may be a coved (type 1) or saddleback (type 2) appearance (see Fig. 1). This pattern, in the setting of presyncope or syncope, may suggest a life-threatening sodium-channelopathy, and timely diagnosis may be a critical point for intervention in the prevention of sudden cardiac death.[20]

DISCUSSION

Once a temperature abnormality is detected, critical workup and management decision depend on determining the underlying cause, whether this is due to environmental exposure (often considered primary hypothermia/hyperthermia) or an underlying disease process (often labeled secondary hypothermia/hyperthermia). Unfortunately, this determination is not always straightforward, and there is some overlap between primary and secondary causes of temperature derangement. Collateral history, and additional patient risk factors, should be considered when helping to determine underlying cause of temperature change.

Age-related physiologic limitations can increase risk of environmental exposure. Newborns and infants have limited mobility to escape temperature extremes, have a high body surface area, and have a low amount of fat.[21] Older adults are less able to regulate body temperature by sweating and more likely to be on a medication or have a medical comorbidity that interferes with temperature control.[22] Substance use can increase risk for environmental exposure by impairing judgment to seek shelter, but also by impairing intrinsic body thermoregulatory functions, such as muscle contraction, shivering, and vasoconstriction/vasodilation.[23] Additional risk factors for environmental exposure include chronic psychiatric disease, socioeconomic factors, comorbid medical conditions, and recreational hobbies.[24]

HYPERTHERMIA

Fever is generally defined as a temperature greater than 38°C; however, there are some exceptions. Decreased body fat percentage and lower temperature setpoints make achieving an elevated temperature less likely in older adults. The Infectious Disease Society of America (IDSA) defines fever for older adults (greater than the age of 65) to be a single oral temperature greater than 37.8° , repeated oral temperatures greater than 37.2°C, repeated rectal temperature greater than 37.5°C, or an increase from baseline temperature greater than 1.1°C. There is consensus between the IDSA and National Comprehensive Cancer Network that neutropenic patients are considered febrile when they have a one-time temperature of greater than 38.3°C or sustained temperature of greater than 38°C for at least 1 hour.[25] Elevated temperature

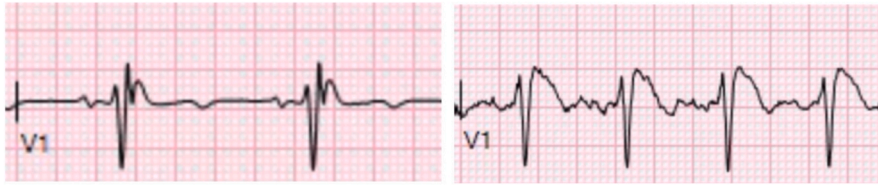

Fig. 1. Osborn wave (*left*) ECG pattern that can be seen in hypothermia. Brugada type 1 (*right*) ECG pattern that can be seen in hyperthermia.

states can be subcategorized into disease-mediated hyperthermia, environmental hyperthermia, and medication-associated hyperthermia (**Box 1**).

DISEASE-MEDIATED HYPERTHERMIA

Infection and Sepsis

One of the most common causes of disease-mediated hyperthermia is infection. Many febrile patients seen in the ED setting will simply have a viral illness. If no antibiotics or antiviral agents are indicated, supportive care measures should be implemented. Antipyretics can be considered.

For febrile patients with an infection and concern for sepsis who can participate in history and physical examination, diagnostics should be tailored to the patient's individual presentation. If the patient is unable to participate because of their baseline health status or their current illness, and sepsis is suspected, a broad diagnostic approach should be taken to include evaluation of the source. If the specific source is evident, antimicrobial agents should be targeted accordingly.

Jarisch-Herxheimer Reaction

First characterized in the treatment of patients with syphilis, this inflammatory reaction is also appreciated in the treatment of multiple spirochete infections, including Lyme

Box 1
Nonenvironmental causes of hyperthermia

Noninfectious inflammatory conditions
- Adrenal insufficiency
- Deep vein thrombosis
- Gout
- Fat embolism
- Hematoma resorption
- Intracranial hemorrhage
- Ischemic cerebrovascular accident
- Jarish-Herxheimer reaction
- Pancreatitis
- Paroxysmal sympathetic hyperactivity
- Pericarditis
- Pheochromocytoma
- Pulmonary embolism
- Postmyocardial infarction fever
- Thyrotoxicosis
- Transfusion reactions
  - Acute hemolytic transfusion reaction
  - Delayed hemolytic transfusion reaction
  - Febrile nonhemolytic transfusion reaction
  - Transfusion-related acute lung injury
- Transplanted organ rejection
  - Acute transplanted organ rejection
  - Chronic transplanted organ rejection
- Tumor lysis syndrome

Medication-associated hyperthermia
- Alcohol withdrawal
- Drug fever
- MDMA intoxication
- Neuroleptic malignant syndrome
- Serotonin syndrome
- Sympathomimetic intoxication

disease, leptospirosis, and relapsing fever. This reaction occurs when patients with spirochete infections are treated with antibiotics, including penicillin, cephalosporins, tetracyclines, and macrolides. This reaction is due to cytokine surge, complement and kinin activation, and accelerated phagocytosis of spirochetes by polymorphonuclear leukocytes. Patients frequently present with fever, chills, headache, and myalgias. If a rash is present, the rash may intensify in appearance. Pregnant patients may experience uterine contractions. It is important to note that patients may not have a known spirochete infection when this reaction occurs, as it may develop while being treated for an unrelated infection. Logically, this reaction may masquerade as an inadequately treated infection, allergy, or adverse medication effect.[26]

Noninfectious Inflammatory Conditions

There are various non-infectious, inflammatory conditions that may present with fever, such as gout, pneumonitis, pancreatitis, and pericarditis. Other conditions that fall into this category include tumor lysis syndrome and fat embolism. Another incompletely understood condition, paroxysmal sympathetic hyperactivity ("neurostorming"), has been described as a cause of fever in patients with traumatic brain injury (TBI). This can occur within a week of TBI, but can also happen during the rehabilitation phase, and therefore, can be seen in the ED if patients are transferred for evaluation.[27] Most of these conditions can have one or more underlying cause. Globally, these conditions are traditionally treated with supportive care measures and multimodal pain management, which typically include antipyretics and anti-inflammatory medications.

Hematologic/Ischemic Conditions

Disorders of bleeding, clotting, and tissue ischemia have long been known to be noninfectious causes of fevers. Intracranial hemorrhage and ischemic cerebrovascular accident are common culprits, particularly if the location of the injury abuts the hypothalamic temperature control center. Local inflammatory signals can cause an increase in underlying temperature set points.[28] Hemorrhage or hematoma formation outside of the brain, such as in the abdomen or soft tissues, can also be the source of a fever, although the explanation for the exact proinflammatory cascade behind this is not well defined in literature.[28,29] Deep vein thrombosis and pulmonary embolism are also known causes of fever. There is likely a proinflammatory component, but fever may also be due to the clot causing local tissue ischemia or infarction.[30] Patients with myocardial infarction (MI) tend to have an increase in temperature by $\sim$1°C by the 4- to –8-hour mark post-MI. Fever typically wanes within 5 days. This response is due to local infiltration of inflammatory cells leading to pyrogenic cytokine release. The degree of fever has been associated with greater myocardial infarct size and less salvaged myocardium.[31]

Endocrinopathies

There are 3 endocrinopathies associated with fever, as follows: thyrotoxicosis, adrenal insufficiency, and pheochromocytoma. Excess T3 and T4 release in thyrotoxicosis leads to increase basal metabolic rate of body cells and a direct increase in body temperature. Thyroid storm is a rapid, acute worsening of thyrotoxicosis resulting in end-organ damage, often in the context of a trigger, such as another acute illness, surgery, pregnancy, or medication effect. Fever is a nearly universal feature of thyroid storm, and temperatures may exceed 40°C. Associated signs and symptoms include tachycardia, atrial fibrillation, high output heart failure, gastrointestinal dysfunction (eg, vomiting, diarrhea, jaundice), and CNS disturbances (eg, agitation, psychosis, seizures). One important pitfall is administering nonsteroidal anti-inflammatory drugs

or salicylates to treat fever in thyroid storm. These medications should be strictly avoided as they can displace T3 and T4 from thyroglobulin, thereby worsening the patient's underlying condition. Preferentially use acetaminophen and external cooling measures and consider treating agitation to prevent worsening hyperthermia from hyperactivity.[32]

Adrenal insufficiency is notoriously challenging to diagnose because of its nonspecific presenting symptoms, including intermittent fevers. Other symptoms may occur, such as gastrointestinal upset and nonspecific constitutional symptoms (eg, myalgias, fatigue, weight loss, dizziness). Primary adrenal insufficiency is typically due to autoimmune destruction of the adrenal parenchyma, but may also be a result of some congenital, infectious, infiltrative, vascular, and iatrogenic pathologic conditions. Secondary adrenal insufficiency, resulting from poor production of adrenocorticotropic hormone, is typically caused by exogenous steroid use or pituitary macroadenomas, but can also have various other causes as well. In addition to nonspecific symptoms, physical examination may not reveal characteristic findings aside from skin hyperpigmentation in primary adrenal insufficiency. Otherwise, fever and hypotension may be the only other indicators that there is a life-threatening underlying pathologic condition.[33]

Pheochromocytoma can present with severe fever (>40°C) owing to excessive catecholamine production and surge that can lead to multiorgan failure. Temperatures can get severely elevated likely because of the excess catecholamines as well as tumor production of interleukin-6. Prompt recognition and management with phentolamine, an alpha blocker, can help minimize mortality. Pheochromocytoma should be considered during true hypertensive crisis with multiorgan dysfunction, and an exceptionally high fever may be one of the diagnostic clues.[34]

Blood Transfusion Reactions

Fever can be the result of various transfusion reactions, both during transfusion and up to weeks later. The most common and benign cause of fever during, or shortly after, a transfusion is the febrile nonhemolytic transfusion reaction. This is characterized by an increase in temperature of 1°C from the pretransfusion temperature. This is thought to be due to a cytokine response to antibodies directed against the white blood cells in the blood product. Patients can be treated with antipyretics. If the patient is unwell and develops rigors, and vital sign abnormalities are consistent with sepsis, bacterial contamination in the blood products should be suspected. The transfusion should be stopped, and the patient should be treated with antibiotics. Acute hemolytic transfusion reactions can develop up to 24 hours after a transfusion. In addition to low-grade temperatures, patients may develop chest or back pain, dyspnea, renal dysfunction, as well as evidence of hemolysis and abnormal coagulation, such as bleeding from intravenous sites. If this condition is suspected, the transfusion should immediately be stopped, and a sample of the patient's blood should be sent for evaluation of ABO incompatibility. The patient should be treated with supportive care. Transfusion-related acute lung injury typically occurs within 6 hours of transfusion and is characterized by noncardiac pulmonary edema and may be accompanied by fever among other vital sign changes. This is treated by optimization of respiratory support, and volume status is typically self-resolved. Delayed hemolytic reactions present days to weeks after a transfusion with low-grade fevers and evidence of hemolysis. Serum analysis for direct antibody testing should be sent, and the patient should be treated with supportive care.[35]

Transplant Rejection

Unfortunately, organ transplant recipients may present to an ED with concern for rejection. Acute rejection occurs within the first 6 months after transplant and is

mediated by activated lymphocytes or antidonor antibodies produced after transplantation. Chronic rejection can occur months to years after transplantation and is antibody and cell mediated. Renal and liver transplant recipients are most likely to develop fever in the context of rejection. Renal transplant recipients may simply present after an acute increase in serum creatinine is appreciated on routine laboratory testing, or may be acutely unwell with fever, hypertension, malaise, decreased urine production, and tenderness of the site of the transplanted organ. When rejection is suspected, serum creatinine, electrolyte studies, allograft ultrasonography with renal dopplers, and an inpatient allograft biopsy is recommended. Liver transplant recipients may present with fever, malaise, and abdominal pain, which may be due to hepatosplenomegaly or ascites. Hepatic function panel, coagulation studies, and an ultrasound with vascular studies of the allograft, as well as an inpatient biopsy are indicated. Prompt consultation with the patient's organ transplant team will help guide considerations for disposition and transfer.[36]

ENVIRONMENTAL HYPERTHERMIA

Climate change is putting larger swaths of the population at risk to develop an environmentally mediated heat-related illness. The most life-threatening is heat stroke, defined as a temperature greater than 40° C with associated CNS disturbance (eg, delirium, seizures, or coma). Frequently, in addition to CNS disturbance, individuals with heat stroke develop multiorgan dysfunction, including renal impairment, hepatic dysfunction, cardiomyopathy, acute respiratory distress syndrome, and disseminated intravascular coagulation.[37] The key tenant in the treatment of heat stroke is removal from the exposure and immediate cooling to 38°C to 38.5°C. The most practical and rapidly effective cooling mechanism is ice-water immersion. Additional techniques include placing ice packs in highly vascular areas, such as the neck, groin, and axilla, and evaporative cooling techniques, such as continuous water spray over patients and using fans. More invasive techniques, such as intravenous cooling catheters and extracorporeal membrane oxygenation, can be implemented.[37,38]

MEDICATION-ASSOCIATED HYPERTHERMIA

Serotonin Syndrome and Neuroleptic Malignant Syndrome

Serotonin syndrome (SS) and neuroleptic malignant syndrome (NMS) are two rare, but life-threatening reactions to psychotropic medications that may present with fever. Patients may also present with tachycardia, tachypnea, and labile blood pressures. Hyperreflexia, ocular clonus, and tremors are characteristic of SS. SS is often rapid in onset, developing over 24 hours. NMS is characterized by bradykinesia with hyporeflexia and muscular rigidity, often developing over the course of many days to weeks. SS is due to increased serotonergic activity from its precursors, decreased uptake, or decreased metabolism. Aside from antidepressants, common medications given in an ED setting, such as fentanyl, linezolid, or ondansetron, can cause or worsen SS. Immediate cessation of offending agents is paramount. Antipyretics are unlikely to be effective in treating fevers associated with SS. Cooling measures, as well as treating agitation leading to hyperactivity, may be necessary. Administration of cyproheptadine can be considered in consultation with a toxicologist for its direct antiserotonergic effects. Most patients improve rapidly. The pathophysiology of NMS is poorly understood but is likely secondary to dopamine blockade by antipsychotic medications. Like SS, the offending agent should be removed. In consultation with a toxicologist, dantrolene or bromocriptine can be administered. With proper treatment of NMS, patients should improve over 1 to 2 weeks. If there is clinical

uncertainty as to whether a patient has SS or NMS, bromocriptine should be strictly avoided, as it can cause worsening illness in SS.[39]

Drug Fever

Drug fever is defined as a fever that occurs after a medication is administered without any other underlying cause of fever. In the ED setting, it would be unusual for a definitive diagnosis of drug fever to be made, considering it is a diagnosis of exclusion by definition. The median time between onset of fever after initiating a drug is 7 to 10 days; therefore, if a patient is presenting with a fever of unknown origin and drug fever is being considered, the offending agent would be an agent that was initiated well in advance of their current visit. Antineoplastic agents, antimicrobials, and antiseizure medications are among the most common drugs associated with drug fever. Because variable classes of drugs can cause drug fever, the mechanism by which drug fever is caused is multifactorial and somewhat dependent on the class of the offending agent. Logically, drug fever caused by antimicrobial agents is likely frequently confused with antibiotic failure. Drug fever is a clinical diagnosis. Patients appear nontoxic and may not be aware that they have a fever. They may also have relative bradycardia. A minority of patients with drug fever may have a cutaneous component, including a maculopapular, urticarial, or petechial rash. If there is a cutaneous component, it may be suggestive of an impending hypersensitivity reaction, so the agent should be discontinued. Fevers should cease within 72 hours of discontinuing the offending agent.[40]

Substance Use–associated Hyperthermia

When a patient presents with a fever, consider their social history. Use of illicit substances and in particular sympathomimetic agents, such as cocaine, 3,4-Methylenedioxymethamphetamine (MDMA), and methamphetamines, can cause fever, as they act directly on the hypothalamus. MDMA also has serotonergic effects that can cause fever in a manner like SS. Withdrawal from substances like alcohol can also cause fever because of high adrenergic tone from high N-methyl-D-aspartate (NMDA) levels and low gamma-aminobutyric acid levels.

HYPOTHERMIA

Hypothermia is considered at a core body temperature $\leq$35°C, although there is no uniform definition or classification system. Temperature ranges for mild (32°C–35°C), moderate (28°C–32°C), and severe (<28°C) hypothermia are different between conventional and trauma-related hypothermia, as trauma patients are less tolerant of hypothermia. In trauma, a temperature of less than 32°C is considered severe, as data have shown a 100% mortality with temperatures at this level. As such, hypothermia makes up one-third of the "lethal triad" of trauma (along with acidosis and coagulopathy), and trauma-related hypothermia is associated with faster onset and higher mortality.[41] Hypothermia can cause numerous symptoms that depend on severity of hypothermia (**Box 2** and **Table 2**).

DISEASE-MEDIATED HYPOTHERMIA

Shock

Any shock state, where there is poor end-organ perfusion and impaired oxygen delivery needed for cellular metabolism, can cause hypothermia. Cardiogenic shock is known as "cold shock" because of poor perfusion from impaired cardiac output. Hypovolemic shock, owing to loss of circulating volume from hemorrhage or fluid

Box 2
Nonenvironmental causes of hypothermia

Dermatologic disorders
- Severe burn
- Severe dermatitis

Endocrine disorders
- Adrenal insufficiency
- Hypothyroidism

Iatrogenic

Infection/sepsis

Neurologic disorders
- CNS infection (meningitis/encephalitis)
- Multiple sclerosis
- Parkinson disease
- Stroke (hemorrhagic or ischemic)
- Trauma (TBI/spinal cord injury)
- Tumor

Nutritional disorders
- Alcoholism
- Hypoglycemia
- Malnourished
- Wernicke syndrome

Shock
- Cardiogenic shock
- Distributive shock
- Hypovolemic shock
- Obstructive shock

Table 2
Symptoms and examination findings by body system in hypothermia[24,41]

System	Symptoms/Examination Findings
Neurologic	Altered mental status, slurred speech, loss of fine motor, loss of reflexes, pupillary dilation, eventual loss of detectable brain wave on electroencephalography
Cardiovascular	Initial tachycardia followed by bradycardia, hypotension, reduced cardiac output, arrhythmia
Pulmonary	Initial tachypnea followed by bradypnea, pulmonary edema, apnea
Renal	Cold diuresis progressing to renal failure
Gastrointestinal	Ileus, stress ulcer, diminished hepatic function with slowed hepatic clearance
Hematologic	Coagulopathy, thrombocytopenia, platelet dysfunction
Integumentary	Initial erythema progressing to pale, cool, frostbite
Musculoskeletal	Initial shivering followed by muscle/joint stiffness
Endocrine	Hyperglycemia, impaired insulin release

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loss, leads to hypothermia from vasoconstriction. Similarly, obstructive shock from impaired blood flow causes decreased perfusion and oxygen delivery, impairing metabolism. Distributive shock, known as "warm shock," causes initial vasodilation that can cause hyperthermia. Examples of distributive shock include anaphylactic shock and septic shock. Hypothermia can occur if this vasodilation remains untreated leading to excessive heat loss through the skin and subsequent hypothermia. Neurogenic shock also causes vasodilation, but also impairs shivering and muscle contraction, leading to further heat loss.[42]

Infection

Infection, particularly sepsis, can cause hypothermia rather than fever. This may be one of the most frequently diagnosed precipitants of hypothermia in the elderly, with several studies citing sepsis as the underlying cause of hypothermia in up to 80% of elderly patients.[42] Hypothermia in sepsis is a cause for concern; mortality is approximately doubled, likely because of increased risk for developing septic shock.[42]

Endocrine Disorders

Hypothyroidism, regardless of cause, can lead to hypothermia owing to decreased thyroid hormone levels. The pathway of hypothermia is multifactorial, from decreased cellular metabolism, reduction of sympathetic nervous system activity, diminished cardiac output, and even impaired ability to respond to environmental cold. Hypothyroidism can be triggered by cold exposure, and myxedema coma more commonly occurs during the wintertime. One major pitfall is failing to recognize myxedema, with a very high mortality approaching 50%, because of the assumption that hypothermia was from cold exposure alone. In the setting of hypothermia from hypothyroidism, passive rewarming is recommended instead of active rewarming. Passive rewarming uses the patient's own body heat to warm and promotes heat retention. This includes removing wet clothes, insulating the patient from the cold environment, and perhaps the use of blankets. Active rewarming can cause peripheral vasodilation, which can cause hypotension.[43,44] Adrenal insufficiency was discussed earlier as a cause of fever, but can cause hypothermia because of impaired sympathetic activity, as well as diminished metabolic function.[42]

Nutritional Disorders

Hypoglycemia reduces metabolic activity because of impaired cellular metabolism. Individuals with alcoholism, who often exhibit compromised glycogen reserves and face an elevated risk of hypoglycemia, may experience hypothermia owing to multiple contributing factors. Alcohol induces vasodilation, which can cause a feeling of warmth while causing hypothermia from heat loss. Wernicke syndrome, caused from diminished thiamine (vitamin B1), affects thermoregulation at the metabolic cellular level, as well as diminished autonomic function and direct effects on the hypothalamus. Any malnutrition state, either intentional from dieting or anorexia nervosa, or unintentional, such as cachexia from chronic disease, reduces the metabolic function of the body and impairs thermoregulatory functions. In pediatric patients with severe acute malnutrition, hypothermia is a poor prognostic indicator.[45]

Neurologic Disorders

Central thermoregulation can be affected by several neurologic conditions; however, this does overlap with some of the other causes of hypothermia. Direct insults to the HTRS (eg, stroke, trauma, infection, tumors, and hemorrhage) can cause dysregulated responses. Hypothalamic and pituitary insults can cause tertiary hypothyroidism and

adrenal insufficiency. Parkinson disease, multiple sclerosis, and Wernicke are all known to cause dysfunction of central thermoregulation.[42] Finally, spinal cord injury can cause hypothermia through several mechanisms—disruption of autonomic pathways resulting in dysreflexia, impaired muscle contraction, and loss of communication with central thermoregulatory pathways.[46]

Dermatologic Disorders

Any severe disruption in the skin's ability to regulate body temperature can lead to hypothermia. Classically, this is seen in large burns,[47] but other dermatologic diseases, such as severe psoriasis or other severe dermatitis, can also lead to heat loss through the skin from impaired blood flow and sweating.[48]

Iatrogenic

Finally, iatrogenic causes can lead to hypothermia. Certainly, hypothermia can be induced in the post–cardiac arrest state as part of targeted temperature management.[49] However, unintentional iatrogenic hypothermia can occur with blood product transfusion as well as when giving intravenous fluids.[50] Hemodialysis can cause hypothermia.[51] Finally, as mentioned earlier, newborns have limited thermoregulatory ability, so be sure to have a warmer available for any precipitous births in the ED.[21]

CARDIAC ARREST

There are 1000 deaths due to hypothermia per year in the United States.[24,52] During resuscitation, temperature check is often delayed because of many other competing priorities, leading to delay in diagnosis. One indication of a hypothermic arrest could be the induction of arrhythmia when a patient is moved, such as the initial transfer from the emergency medical service stretcher to the ED stretcher. The cold myocardium is susceptible to any additional stress, and the rough handling of severely hypothermic patients can induce cardiac arrhythmia.[24] However, cardiac arrest itself leads to hypothermia, as there is cessation of all movement and metabolic activity. The question of whether the patient is in cardiac arrest owing to hypothermia, or had a cardiac arrest and is subsequently hypothermic, may be difficult to answer. Do not rely on location where the patient was found, as patients can still suffer from a hypothermic cardiac arrest despite being found indoors. Cardiac arrest owing to hypothermia may lead to a lengthy resuscitation, as good neurologic recovery can occur despite prolonged downtime. In contrast, a cardiac arrest where the patient had died and is now hypothermic owing to delay in resuscitation may indicate futile resuscitative efforts. When in doubt, always presume hypothermia leading to cardiac arrest and resuscitate accordingly. Pupil dilation, dependent lividity, even concern for rigor mortis cannot be interpreted as a sign of nonviable resuscitation in the context of hypothermic arrest. There are 2 indications to terminate resuscitation in hypothermic arrest, as follows:

1. The patient has been rewarmed to 32°C–33°C, and return of spontaneous circulation has not been achieved;
2. The patient's measured serum potassium is greater than 12mmol/L.[24]

SUMMARY

Accurate temperature measurement in the ED is crucial. There are various invasive and noninvasive methods of temperature measurement, with different limitations.

Temperature abnormalities can impact other vital signs, including heart rate, blood pressure, respiratory rate, and oxygen saturation. These subtle nuances may help identify or obscure serious diagnoses.

Body temperature can impact diagnostic tests, such as the blood-gas and ECG. Secondary causes of hyperthermia can be categorized into infectious, inflammatory, endocrine, environmental, and drug-related causes. Secondary hypothermia can be due to endocrine disorders, neurologic disorders, nutritional disorder, dermatologic conditions, and iatrogenic factors. Having a broad differential diagnosis when temperature abnormalities are identified can have significant clinical implications when caring for patients.

CLINICS CARE POINTS

- There is overlap between environmental exposure and disease-mediated impairment of thermoregulation. Be sure to take a good history and consider alternative causes of temperature derangement.
- Two poor prognostic indicators related to sepsis are hypothermia and relative tachycardia (heart rate out of proportion to fever).
- Older adults are less likely to mount an elevated temperature. Consider lowering temperature thresholds for fever for patients $\geq$65 years old per IDSA guidelines.
- When a patient develops a new or recurrent fever despite being treated with antibiotics for an infection, expand your differential to include drug fever and Jarish-Herxheimer reaction, in addition to potential antibiotic failure or worsening infection.
- Cardiac arrest efforts can be terminated during hypothermic arrest if no Return of Spontaneous Circulation (ROSC) by 32°C to 33°C or if potassium is greater than 12 mmol/L.

DISCLOSURES

The authors have nothing to disclose.

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