Accidental Hypothermia in the Elderly

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DR. FRANK S. CELESTINO (*Assistant Professor, Department of Family Medicine*): Today's Grand Rounds will focus on a problem among the elderly population with which many clinicians are unfamiliar: hypothermia. Several recent reviews[1–11] have stressed the presentation, pathophysiology, and management of hypothermia and its complex clinical sequelae. We recently encountered a very elderly patient with profound depression of body temperature and a multitude of associated complications. Before hearing this patient's presentation, I thought it would be useful to establish some working definitions.[1–6]

CLASSIFICATION AND TERMINOLOGY

Clinical hypothermia is defined as a decrease from man's average 37 °C homeothermic core body temperature to less than 35 °C (95 °F). *Subclinical hypothermia* implies a central temperature of 36.5 to 35 °C without obvious signs and symptoms, a situation seen almost exclusively in the very old. The lowest reading on standard clinical thermometers is 34 to 35 °C,[8,10] a possible cause of underdiagnosis of hypothermia. Although emergency rooms should be stocked with specially scaled thermometers to detect lower temperatures, a 1982 survey of hospital-based emergency rooms in New York State[12] revealed that only 20 percent had such thermometers available.

Accidental hypothermia refers to an unintended decrease in core body temperature without primary disease of the central nervous system's thermoregulatory center. Golden[13] subdivides accidental hypothermia into primary and secondary categories. The *primary* form involves normal thermoregulatory mechanisms but overwhelming cold stress, as in cold water immersion or mountaineering accidents. The *secondary* type, which usually affects the frail elderly population, involves only mild to moderate cold stress but abnormal thermoregulation caused by drug use or disease-related alterations in heat production or loss.

Others[1,5,14] have proposed subtypes of accidental hypothermia based on the rate of heat loss: *acute* (in 1 hour or less, *subacute* (over several hours), or *gradual* (slow heat loss over days to weeks). The last type, seen primarily in the very old, is associated with decreased thermogenesis and abnormal heat conservation brought on by chronic debilitation or superimposed acute illness.

Many authorities[1,5,14] characterize hypothermia as mild (core temperature 32 to 35 °C), moderate (28 to 32 °C), or severe (<28 °C). These categories are conceptually helpful in terms of predicting clinical pathology (Table 1).[1–7,11]

CASE PRESENTATION

DR. COLLEEN MIRAGLIA (*Chief Resident in Family Medicine*): Mrs. M.S. was an 87-year-old widowed woman who was being followed for well-controlled essential hypertension, euthyroid goiter, and atherosclerotic heart disease, which was manifested by compensated congestive heart failure and sick sinus syndrome and managed with a permanent pacemaker. Medications included furosemide, a potassium supplement, and a low dose of aspirin. She had been living in a foster care home with three other elderly women.

During the five days prior to admission, Mrs. M.S. expressed multiple complaints to her caretakers. Her complaints included nausea, mild diarrhea, abdominal cramps, and progressive swelling, pain, and coldness of her lower extremities, the left side being worse than the right. On the day prior to admission, she was weak, though she was still able to sit in a chair, feed herself, and take medications.

On the morning of admission, the patient became more confused and lethargic. She was found by the rescue squad at 11:00 AM, responsive only to deep pain. Vital signs were normal except for a rectal temperature of 31.6 °C

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TABLE 1. CLINICAL SIGNS AND SYMPTOMS AT DIFFERENT LEVELS OF HYPOTHERMIA*

Temperature °F	Temperature °C	Sign or Symptom
99.6	37.6	"Normal" rectal temperature
98.6	37.0	"Normal" oral temperature
96.8	36.0	Sensation of cold, mild confusion, shivering begins
95	35.0	Cold skin, slurred speech, stumbling, incoordination, prominent shivering
93.2	34.0	Increasing confusion, amnesia, incoordination; near maximal shivering; blood pressure normal; decreased bowel sounds; deep tendon reflexes and pupillary responses exaggerated; increased cardiac output; tachycardia; hyperventilation; electrocardiogram (ECG) normal
88.6–91.4	32.0–33.0	Decreasing blood pressure and cardiac output, stupor, minimal shivering, urine flow twice normal (cold diuresis), hypovolemia, hemoconcentration, respiratory alkalosis, deep tendon reflexes normal
86.0–89.6	30.0–32.0	Cardiac output only 50 percent; low blood pressure, cardiac arrythmias (atrial fibrillation, bradycardia, atrioventricular block); pupils dilated; hypoventilation, acidosis, hypoxia and cyanosis; shivering nearly absent; muscular rigidity (geling); hyperglycemia; few bowel sounds; progressive loss of consciousness; ECG Osborn (J) wave first seen
82.4–86.0	28.0–30.0	Severe hypoventilation, marked bradycardia, arrhythmias worsen, ventricular fibrillation possible, nearly absent deep tendon reflexes and bowel sounds, usually unconscious, worsening hypotension, increased salivation
77.0–82.4	25.0–28.0	Voluntary motion lost, patient may appear dead; unconsciousness; extreme muscular rigidity simulating rigor mortis; deep tendon, pupillary, and oculocephalic reflexes often absent; ECG and electroencephalogram (EEG) show minimal activity; spontaneous ventricular fibrillation possible; shock; protective vasoconstriction lost; no heart sounds
69.8–75.2	21.0–24.0	Patient appears dead, pulmonary edema, severe risk of ventricular fibrillation, apnea, minimal cardiac activity
68.0–69.8	20.0–21.0	Cardiac standstill
64.4–68.0	18.0–20.0	Isoelective EEG

** Adapted from Harnett RM et al.*[15]

(89 °F). Glucose determination by Dextrostix at the home was 6.7 mmol/L (120 mg/dL).

FAMILY PRACTICE RESIDENT: Was there any history of exposure or information on the indoor temperature at the foster home?

DR. MIRAGLIA: It was January. The patient had not been outdoors for six days and reportedly had not been found uncovered at any time. The indoor temperature was reported to be 24 to 25 °C (75 to 77 °F). This temperature could represent enough cold stress in an elderly patient with acute illness and disturbed thermoregulation to induce hypothermia.[1,6]

In the emergency room the patient's rectal temperature again was 31.6 °C (89 °F) with blood pressure 160/90 mmHg, a pacemaker-controlled pulse of 72 beats per minute, and shallow respirations of 32/min. Initial physical examination was notable for a goiter, carotid bruits, clear lungs, no jugular venous distention, an intermittent S_3 gallop, markedly decreased bowel sounds, and a moderately distended and diffusely tender abdomen. Stool was positive for occult blood. Extremities were mottled and very cold with a dusky left leg. There was edema (2+) of the legs with trace palpable pulses. The patient responded to pain only with incoherent mumbling and movement of all extremities.

Initial laboratory values showed hemoglobin 8.13 mmol/L (13.1 g/dL), hematocrit 0.42 (42.2 percent), leukocyte count 8.5×10^9/L (8,500/mm^3) with normal differential, and platelets of 107×10^9/L (107×10^3/mm^3). Prothrombin time was 23 seconds, and partial thromboplastin time was 49 seconds. Fibrinogen was markedly decreased at 0.58 g/L (58 mg/dL) with high titers of fibrin degradation products. Total creatine kinase equaled 35.51 μkat/L (2,130 U/L). Sodium was 141 mmol/L (141 mEq/L), potassium 8.4 mmol/L (8.4 mEq/L), chloride 114 mmol/L (114 mEq/L), total carbon dioxide 7 mmol/L (7 mEq/L), blood urea nitrogen 28.2 mmol/L (79 mg/dL), creatinine 292 μmol/L (3.3 mg/dL), and glucose 6.3 mmol/L (113 mg/dL). Temperature-corrected arterial blood gas on room air showed a pH of 7.40, a carbon dioxide pressure (PCO_2) of 11 mmHg, and an oxygen pressure (PO_2) of 87 mmHg. Urinalysis revealed a specific

gravity of 1.020. The dipstick was positive for blood and protein, and there were 3 to 5 white blood cells per high-power field, 6 to 8 red blood cells, and many bacteria. Urine myoglobin was 730 g/L (73 g/dL). Results of urine drug screening tests for acetaminophen, salicylates, and alcohol were negative. A chest roentgenogram showed moderate cardiomegaly but no heart failure or infiltrate. An electrocardiogram revealed the patient's paced rhythm and peaked T waves, but no acute ischemia or infarction was seen.

FAMILY PRACTICE RESIDENT: How do you correct arterial blood gases for body temperature?

DR. MIRAGLIA: Although nomograms exist for easy interpretation, for each degree of centrigrade drop in temperature, the pH increases .015, the PCO_2 decreases by 4.4 percent, and the PO_2 drops by 6 percent.[2,7]

The admitting diagnoses for Mrs. M.S. included hypothermia with possible occult sepsis from urinary tract infection, disseminated intravascular coagulation, hyperkalemia, probable left leg tissue necrosis, rhabdomyolysis, metabolic acidosis, renal insufficiency, and possible occult myocardial infarction.

Initial resuscitative measures in the field and emergency room included administration of naloxone, thiamine, high-dose glucose, potassium-binding resins, intravenous fluids, and broad-spectrum antibiotics. The patient was rewarmed with a heat lamp, warm humidified oxygen by mask, a heating blanket, and warmed intravenous fluids. During the first hour of treatment, her rectal temperature dropped further to 29.5 °C (85 °F) before rebounding to 31.6 °C (89 °F) after two hours. The temperature did not stabilize at 37 °C until 16 hours after admission.

During the first 36 hours of admission, Mrs. M.S. underwent emergency exploratory laparotomy for signs of an acute abdomen. She received an above-the-knee amputation for her left leg necrosis, and was treated with emergency hemodialysis, invasive hemodynamic monitoring (through a Swan-Ganz catheter), and mechanical ventilation for respiratory tract failure. The laparotomy revealed no source for the physical findings, and the pathology report of the leg indicated severe occlusive atherosclerotic arterial disease with an extensive clot in the proximal popliteal and femoral arteries.

Over the next few days the patient required repeated dialysis and multiple transfusions of blood products to combat hemolysis and disseminated intravascular coagulation. She remained responsive only to pain. Bronchopneumonia and necrosis of the right leg became evident. We held a family conference to discuss further therapy.

DR. GLENN R. VAN NOORD (*Assistant Professor, Department of Family Medicine*): As Mrs. M.S.'s attending physician, I recognized that the family members were increasingly dismayed about the patient's technology-supported status. After hours of agonizing discussion, they requested a continuation of the ventilator, antibiotics, and pressor agents, but specifically opposed further dialysis, right leg amputation, additional blood transfusions, and cardiopulmonary resuscitation. Under these guidelines, the patient died during the next week.

EPIDEMIOLOGY

DR. CELESTINO: The hazard that the cold presents to elderly people, especially the very old, has been recognized clinically for only 25 years. Assessing the true impact of this problem is hindered by underdiagnosis and the lack of uniformity in the coding of death certificates, which so often reflect associated clinical conditions or complications.[1,14] Despite the inadequacies in reporting, there are data[16] documenting an increasing number of hypothermia-related deaths and significantly increased mortality rates for the elderly compared with younger individuals. Risk of hypothermia is highest for the very old (over 75 years), and disease-specific mortality rates for men are three to five times those of women at all ages.

It is possible that we are missing diagnosing patients with occult hypothermia in our hospitals and clinics. One British study[17] revealed that nearly 4 percent of elderly inpatients unexpectedly had core temperatures less than or equal to 35 °C, while in another hospital report,[18] oral temperature readings revealed a 34 percent incidence. Other researchers[14,19,20] have found that between 0.6 to 15 percent of elderly outpatients were hypothermic. In contrast, a recent small study[21] showed no evidence of clinical hypothermia in 97 elderly outpatients.

PATHOPHYSIOLOGY AND PREDISPOSING CONDITIONS

DR. CELESTINO: Under normal circumstances,[8] cold exposure activates skin thermoreceptors that send signals to the hypothalmus, which in turn increases heat production by stimulating shivering, voluntary activity, and catecholamine output. Heat loss is minimized by increasing cutaneous vasoconstriction and piloerection. Under continued cold stress, augmented thyroid and adrenal output contribute to nonshivering thermogenesis. All of these mechanisms act to counterbalance the physical forces responsible for heat loss—conduction, convection, radiation, and evaporation.

Aging affects this thermoregulatory system in a variety of ways. In Table 2[5,6,10] this altered physiology and the constricted hemeostasis so characteristic of the frail elderly individual are highlighted.

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TABLE 2. ALTERED PHYSIOLOGIC MECHANISMS IN ELDERLY PATIENTS PREDISPOSING TO HYPOTHERMIA

1. Diminished ability to increase respiratory quotient and heat production (basal metabolic rate) in face of cold stress
2. Inadequate vasoconstrictor response
3. Decreased resting peripheral blood flow
4. Decreased mobility (trauma, musculoskeletal disease, stroke, gait disturbance, etc)
5. Diminished muscle mass
6. Impaired autonomic nervous system function and reflexes
7. Impaired recognition of lowered environmental temperature
8. Diminished shivering response
9. Lower resting basal metabolic rate
10. Desynchronization of the diurnal cycle controlling heat loss and production
11. Decreased subcutaneous fat (insulating layer) in the very old

Conditions that predispose to hypothermia are listed in Table 3. Many of these conditions are found frequently in the very old and may act in concert to impair central and peripheral thermoregulation and to adversely affect heat production or loss.

FAMILY PRACTICE RESIDENT: Was a specific cause for the patient's hypothermia ever determined?

DR. VAN NOORD: No, not with any certainty. All culture results remained negative, and the invasive hemodynamic readings were not those of sepsis.[22] Thyroid functions were normal. Despite very high peak levels of creatine kinase with MB isoenzyme positivity, no evidence of myocardial infarction developed on electrocardiogram. The creatine kinase findings are not unusual in the setting of hypothermia[3,7] and may also have been related to the patient's leg muscle necrosis. No clinical evidence of stroke ever appeared, though a computed tomography head scan was not performed. It is possible that arterial thrombosis of the left leg was the critical inciting factor, though disseminated intravascular coagulation may have accounted for this finding.

Unfortunately an autopsy was not obtained, which markedly limited our ability to determine the precipitating event. During the hospitalization we had mentioned to the family members our strong interest in an autopsy, but, despite these discussions, they ultimately did not consent.

COMPLICATIONS

DR. CELESTINO: Our patient exhibited many of the recognized complications of a hypothermic insult (Table

TABLE 3. PREDISPOSING CONDITIONS FOR HYPOTHERMIA IN THE ELDERLY

Environmental
Acute or chronic exposure (immersion, after falling, hiking accidents, immobility), poor living conditions (living alone, lower socioeconomic status)

Iatrogenic
Induced (for surgery or other treatment)

Drug induced
Phenothiazines, ethanol, barbiturates, anesthetics, phenformin, antidepressants, benzodiazepines, glutethimide, narcotic analgesics, muscle relaxants, reserpine

Central nervous system or neurologic
Cerebrovascular disease, Wernicke's encephalopathy, head trauma, neoplasms, mental illness, dementia syndromes, spinal cord injury or transection, neuromuscular disease

Nutritional
Protein calorie deficiency, cirrhosis

Infectious
Sepsis, pneumonia

Renal
Uremia

Endocrine or Metabolic
Hypothyroidism, hypopituitarism, hydroadrenalism, hypoglycemia, diabetes mellitus or ketoacidosis, carbon monoxide poisoning, anorexia nervosa, Paget's disease (extensive)

Cardiovascular
Myocardial infarction, peripheral vascular disease, congestive heart failure, pulmonary embolus

Dermatologic
Diffuse erythrodermas (psoriasis, exfoliative dermatitis, ichthyosis), severe burns

4).[6–9] Her initial abdominal distention and guaiac-positive stool were, in retrospect, related to the hypothermia rather than to a primary intraabdominal process. Despite temperatures in the 30 to 32 °C range, our patient did not manifest the characteristic J wave, or Osborn wave, on an electrocardiogram.[2,7,11] This abnormality, J point elevation and "camel humping" of the first portion of the ST interval, was thought to be pathognomonic of hypothermia, but now there is evidence that other conditions may cause this abnormality.[11]

MANAGEMENT AND PROGNOSIS

DR. CELESTINO: Accidental hypothermia is a medical emergency. The goals of management[15] are to maintain adequate cellular respiration, minimize additional heat loss, restore normal body temperature and homeostasis (fluid, electrolyte, and acid-base balance), treat any non-

TABLE 4. COMPLICATIONS OF HYPOTHERMIA

1. Bronchopneumonia, pulmonary edema, respiratory arrest, aspiration
2. Shock, congestive heart failure, myocardial infarction, cardiac arrest, ventricular fibrillation and other arrhythmias
3. Stroke, seizures, altered mental status
4. Acute renal failure (acute tubular necrosis), hematuria
5. Disseminated intravascular coagulation, hemolysis, depressed bone marrow
6. Hemorrhagic pancreatitis, gastrointestinal bleeding, ileus
7. Hypoglycemia
8. Myoglobinuria
9. Temporary adrenal insufficiency (usually in exhaustive hypothermia)
10. Sepsis

environmental cause of the hypothermia, and optimize medical problems unrelated to hypothermia. These goals are usually best acccomplished in the intensive care unit.[1–11,15,23–25]

Management can be divided into two categories: *general supportive care* and *specific rewarming techniques*.[1–9,15,23–25] Patients require intensive monitoring of fluids, electrolytes, acid-base balance, arterial blood gases, renal function, urine output, electrocardiogram, cardiac output, pulmonary function, and coagulation indices. Patients must be handled gently and without unnecessary stimulation to avoid precipitating ventricular fibrillation. In all but the mildest cases, therapeutic decisions are aided significantly by invasive hemodynamic monitoring. Treatment and identification of underlying illness should proceed in concert with initial resuscitative efforts. The routine use of corticosteroids, vasoactive drugs, antiarrythmics, and thyroid supplements is no longer recommended. Though controversial, many authorities[6,10,14,26] now recommend routine early administration of broad-spectrum antibiotics, even without an obvious source of infection.

When hypothermia is severe, the patient many appear to be dead, in cardiopulmonary arrest and central nervous system shutdown. Resuscitative efforts must continue until the core temperature is greater than 35 °C.[2,27] There are several reported cases of presumably dead individuals who have recovered uneventfully after two to three hours of intensive life support.[27] As has been so often stated, "The patient is not dead until he is warm and dead."

Intense controversy continues concerning the optimal method and rate of rewarming.[1–11,15,23–25] Experts hold widely divergent views regarding the efficacy and risk of alternative modalities in varied clinical settings. This lack of consensus exists because of the absence of statistically significant controlled clinical trials. Recommendations are often based on retrospective case studies and anecdotal reports of relatively small, select patient groups.

Three classes of rewarming methods have been described[1–7,11,15,23–25]: passive external rewarming, active external rewarming, and active core rewarming. Passive external rewarming refers to moving the patient to a warm environment (greater than 25 °C) and insulating him from further heat loss. Active external rewarming may involve bodily contact, hot water bottles, electric blankets, radiant warmers, warm water immersion, or blankets with circulating warm water. Active core rewarming refers to the use of hot food, warmed intravenous fluids, gastric or mediastinal irrigation, peritoneal dialysis or hemodialysis, heated humidified oxygen, or extracorporeal blood rewarming.

The optimal rate and method of rewarming should minimize afterdrop and aftershock and result in the lowest morbidity and mortality.[2,5–8,15,23–25] Afterdrop is a paradoxical drop of core temperature occurring during initial rewarming. It is thought to occur when abrupt peripheral vasodilatation releases cooled, acidotic blood that is mixed with warmer core fluids, although the exact mechanism is uncertain. Aftershock, which is more likely to occur in the elderly patient, results when sudden peripheral vasodilatation occurs in the setting of insufficient circulatory volume resulting from cold diuresis and massive fluid shifts, as in gradual hypothermia. Hypotension results from a lack of compensatory increase in cardiac output from a compromised, aged, cold myocardium. Both aftershock and afterdrop, as well as skin burns and inhibition of endogenous heat production, are felt to be potential adverse effects of active external rewarming.[11]

There appears to be no statistically sound evidence to support one method of rewarming over another. A recent, large, multicenter study[28] examining the outcome for 428 hypothermic patients verified this impression. This situation may exist because no one method is ideal for the diverse causes and circumstances leading to hypothermia, or because variance in supportive care rather than rewarming technique is responsible for the large differences in mortality found among the reported series.[6] Prognosis appears less related to the rewarming modality than it is to the severity of underlying diseases and precipitating factors.[5–7,11,28] Thus, elderly patients have a dramatically increased mortality, while young patients, even in the face of more severe insults, generally do well.

As a general recommendation it would seem that warmed intravenous fluids and warmed, humidified oxygen are reasonably accessible and safe methods for rewarming in most circumstances of mild to moderate hypothermia.[1–7,15,23–25] In severe hypothermia it may be best to initiate more aggressive core rewarming, especially in

the aged,[23,25] using the most universally available method—peritoneal dialysis with warmed fluids.

Because morbidity and mortality is so high in the aged, and in light of the controversies surrounding treatment, preventive measures assume great importance.[6] Prevention of hypothermia requires physician inquiry about living conditions, diet, exercise, drugs, and alcohol intake. Actively searching for subclinical hypothermia to identify a high-risk population may be helpful. Closer monitoring during winter months of hypothermia-related chronic diseases may be useful. Such measures assume even greater importance for recovered hypothermic patients, because data exist that indicate their special susceptibility to recurrent hypothermia.[6,11]

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