Hypothermia in the elderly

The syndrome you least expect

Even in moderate climates, geriatric patients are vulnerable to accidental hypothermia — and the condition mimics many other disorders.

By RE Carlson, MD and PJ Hering, MD

If someone told you that he or she had just diagnosed a patient with "accidental hypothermia," you might immediately picture an alcoholic who had passed out in a snowbank during a drunken stupor and sustained frostbite of the extremities and a decreased core temperature.

Until fairly recently, alcoholics were the group at greatest risk in the US for accidential hypothermia; because alcohol is a vasodilator, central nervous system depressant, and anaesthetic, it can cause hypoglycemia and increase the risk of trauma and exposure.

Today, however, the hypothermic patient is more likely to be that elderly teetotaling, fixed-income patient on medications for hypertension, diabetes, or emotional problems whom you just saw in your office a few weeks ago for a routine follow-up visit.

Prolonged exposure to temperatures as high as 16C to 18C (60F to 63F) can trigger hypothermia in geriatric patients. [1]

You needn't practise in an area with an extremely cold climate to see accidental hypothermia in the elderly.

Often, it occurs in people who simply live in chilly apartments or do not use heaters in order to save money.

Normal thermoregulation

To understand how accidental hypothermia occurs, it's helpful to briefly review the body's heat-regulating system.

We can think of the body as a layered structure with a core protected by a superficial zone and an intermediate zone, which modulate heat loss and gain.

The superficial zone is composed of the skin, subcutaneous tissue, and thermoreceptors; these structures are the most important agents in heat exchange.

Skin temperature varies with blood flow, ambient air temperature, humidity, and air velocity and may drop to nearly environmental levels in an effort to conserve heat.

The intermediate zone is composed of the skeletal-muscle mass, which ordinarily contributes little to heat production.

When the core is in danger, however, the muscles can produce a considerable amount of heat by shivering, which, in turn, causes muscular vasodilatation and an increase in blood flow and delivery of warmed blood to the core.

Integration of these various components of the body's response to cold exposure is coordinated by the preoptic anterior hypothalamus **(Figure 1).** The neural network, acting via the autonomic nervous system, rapidly controls heat dissipation.

The neuroendocrine aspects of heat production (nonshivering thermogenesis) involve a more delayed increase in heat production through the pituitary, thyroid, and adrenal mechanisms.

Hypothermia occurs when the system fails to function properly or when environmental conditions render it inadequate.

The most obvious consequence is a decreased metabolic rate; it drops to 50 percent of normal when core temperature reaches 28C (82F), diminishing oxygen consumption and CO_2 production.

How hypothermia occurs

Accidental hypothermia is defined as a spontaneous decrease of the core temperature (rectal, oesophageal, or tympanic membrane) below 35C (95F), usually but not always in a cold environment, presenting as an acute problem without primary disease of the temperature-regulatory centre.

Among the reasons for the increased incidence of the disorder among the elderly are inactivity; mal-

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nutrition; medications that impair central nervous system responses and peripheral vasomotor responses; decreased basal metabolic rate, body fat, and skeletal-muscle mass; the presence of circulatory or hormonal disorders; and reduced mental capacity.

In addition, elderly people may lack the appropriate physiological responses to cold.

Compared with younger people, they may be unable to control their heat loss effectively by peripheral vasoconstriction. Often, they are less able to sense the cold than they were when they were younger.[2]

An episode of hypothermia may itself damage the patient's thermoregulatory mechanism for as long as three years afterward and may make him even more vulnerable to subsequent episodes of hypothermia.[2, 3]

The mortality from significant hypothermia in the elderly is about 80 percent, compared with only 10 percent in younger patients, an astounding difference.

One of the reasons is that temperature is usually recorded with standard clinical mercury thermometers, which do not register low levels of body temperature, even when shaken down.

The proper diagnosis of hypothermia requires a thermometer that will read at least as low as 24C (75F). Electronic digital thermometers have a far wider range than mercury thermometers.

When to suspect hypothermia

As most of us were taught in medical school, you can't diagnose what you don't think of; therefore, consider the possibility of accidental hypothermia in the differential diagnosis of elderly people who have any of a number of presentations, even when environmental conditions are not extreme.

Dizziness, momentary blackouts, or changes in mentation may be the only initial symptoms of accidental hypothermia in geriatric patients.

Surprisingly, most elderly victims of accidential hypothermia neither shiver nor turn pale.

These symptoms may be followed by fatigue, weakness, slow movements, incoordination, apathy, and generalised confusion. As the core temperature decreases, judgement declines, hallucinations often occur, and the patient may become extremely combative and resist all attempts at medical aid. If the core temperature drops below 27C (80,6F), the patient will go into a deep coma.

Check the organ systems

Beyond the fact that hypothermia may be missed through inadequate temperature readings, patients may be subjected to over-zealous or even injurious medical care.

Since the disorder can affect literally every organ system in the body, it may mimic such diseases as hypothyroidism, hypopituitarism, hypoadrenalism, senile dementia, and cardiovascular shock.

Because low body temperature is associated with many of these conditions, the hypothermia goes unnoticed and untreated.

Thus, in making a differential diagnosis it is important to evaluate the organ systems with an understanding of how a hypothermic state affects them.

Central nervous system. Cerebral blood flow decreases seven percent for each degree centigrade drop in core temperature. This and alterations in the microcirculation resulting from increased blood viscosity are the causes of the typical abnormal or decreased mentation seen in hypothermic patients.

Dizziness, blackouts, confusion, euphoria, or even overt psychosis may develop as the core temperature drops.

Fixed, dilated pupils suggest a temperature level below 30C (86F). Hyporeflexia progresses as the core temperature drops, and a classic "hung up" reflex develops that can be easily confused with hypothyroidism.

The patient is usually areflexic at 25C (77F) or below. A flat EEG indicates a level below 20C (68F). In an emergency room setting, these findings and the lack or palpable peripheral pulses may make the patient appear dead.

The one universal axiom of hypothermia that needs to be stressed is that *no one is dead until he is warm and dead.* Patients have fully recovered after core temperatures as low as 17C (60F).

As hypothermia develops, the patient may become irrational, experience a paradoxical surge of heat from the body core to the surface, and remove some or all of his clothing.

When an elderly person is found in a state of undress, people may suspect that he or she is a victim of foul play.[4] If the patient is a woman, police may jump to the conclusion that she is a rape/murder victim and refuse to allow anyone, including medical personnel, to examine the body.

If there is even the remotest chance that accidental hypothermia is involved, aggressive action must be taken, no matter what the authorities say.

Several anecdotal reports tell of doctors being arrested for interfering with a murder investigation, with the "murder victim" being saved after appropriate medical therapy.[4]

Cardiovascular system. Look for a decline in heart rate and cardiac output as indications of hypothermia. Although the blood pressure rises initially, it gradually falls with core hypothermia.

Clinically significant hypotension is usually present by 25C (78F).

Be especially alert to hemodynamic effects of cold on myocardial conduction. At 30C (86F), a definite progression of ECG abnormalities begins, with a diminished sinus rate, T-wave inversion, Q-T interval prolongation, and the development of the pathognomonic J, or "Osborn", wave **(Figure 2).**

Atrial fibrillation or flutter is usually present by 28C (82F), and below 28C junctional rhythm, ventricular fibrillation, and asystole all become likely possibilities.

Careful management of patients with such abnormalities is critical to avoid triggering ventricular fibrillation or cardiac asystole.[5]

Respiratory system. Depression of the respiratory centre becomes significant only when the core temperature is very low, but decreased mentation, a diminished cough reflex, and cold-induced bronchorrhea lead to aspiration, difficulty clearing secretions, and secondary pneumonias.

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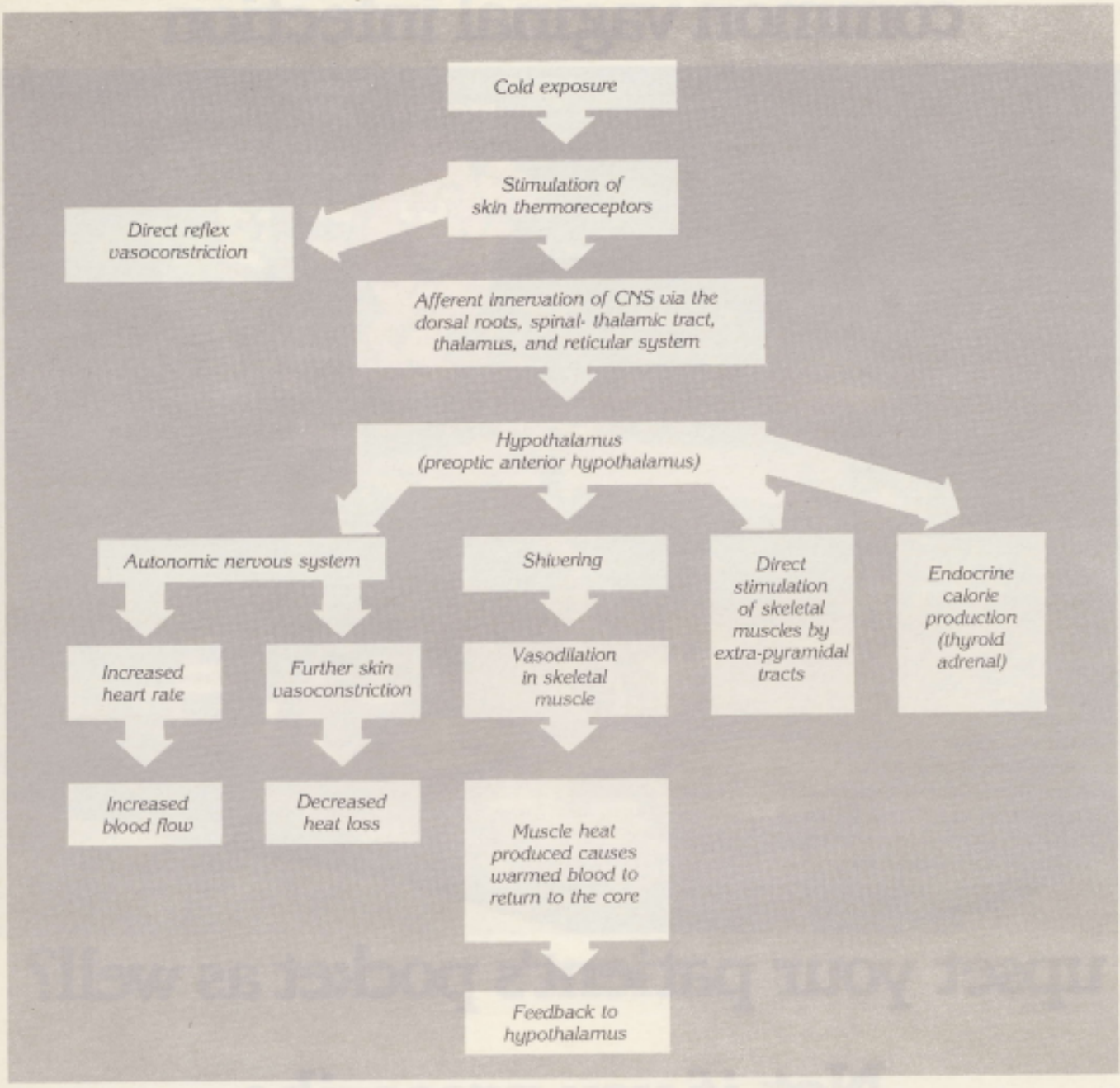

Figure 1 *How the body keeps warm*

The oxyhemoglobin disassociation curve shifts to the left with an increased affinity for oxygen, but this is usually not clinically significant because of the decrease in oxygen requirements with hypothermia.

In hypothermia, respiratory arrest usually indicates that the patient's core temperature has fallen to 24C (75F).

Hypothermia markedly affects the interpretation of routine blood gases, and blood-gas determinations must be adjusted to reflect the effective tissue level at a given core temperature.

Samples for blood gases are routinely put on ice and in the laboratory are rewarmed to 37C (98,6F) to be run through the blood-gas analyzer.

If you fail to warn the lab that a patient's core temperature is, say, 30C (86F), the blood-gas analysis will be reported as pH, 7,20, PCO_2, 60 percent, and PO_2, 90 percent, instead of 7,30, 44, and 60, respectively.

A diagnosis of excessive CO_2 retention and over-all acidosis, based on the unadjusted data, could result in inappropriate and excessive treatment.

Alkalosis, like acidosis, markedly favours the development of ventricular tachycardia. The treatment of an apparent severe acidosis with excessive amounts of bicarbonate could easily produce alkalosis, with catastrophic results.[5]

Acid-base equilibrium. The acid-base status of a patient with hypothermia depends to a great extent on the patient's age and the presence of other concomitant illnesses. Most hypothermic patients show acidosis secondary to CO_2 retention from respiratory failure, or metabolic microcirculatory failure.

If shivering has occurred it may also cause a great increase in the production of lactic acid; the accumula-

tion may be aggravated by decreased metabolic clearance of the lactic acid by the liver.

In addition, circulatory failure may prevent organic acids from being mobilised toward the appropriate buffer systems. This may help to explain why patients are only mildly acidotic while they are hypothermic and why they tend to get much more severe acidosis during the rewarming process as the products of anaerobic metabolism return to the general circulation.

Hematologic effects. Hypothermia brings an increased hematocrit as a result of plasma loss and splenic contraction. A significant increase in blood viscosity occurs at 27C (80F) or less.

The leukocyte and platelet counts are depressed as a result of splenic, hepatic, and intravascular sequestration.

Particularly during rewarming, disseminated intravascular coagulation is a major complication in some patients, especially the elderly.

Gastrointestinal tract. Gastrointestinal motility decreases below 34C (94F), and ileus is universal in significant hypothermia. Hepatic detoxification and conjugation are also severely depressed with hypothermia, and drugs, especially those given during the hypothermia, remain in the system for prolonged periods.

This is another frequent cause of error in treating hypothermia.

Kidneys. Urine flow increases with a dropping core temperature (cold diuresis), probably because of depressed oxidative tubular activity.

Reduced sodium and water resorption results; with a shift of fluids to the extracellular spaces, it renders the hypothermic patient hypovolemic intravascularly.

Endocrine system. Because hypothermia inhibits both the release of insulin from the pancreas and the peripheral use of glucose, it causes hyperglycaemia. The condition usually disappears with rewarming, and any attempt to reverse it with insulin is extremely dangerous, since it may result in profound hypoglycaemia.

Remember, shivering may deplete much of the body's stored glycogen, a situation that can also lead to hypoglycemia if insulin is given.

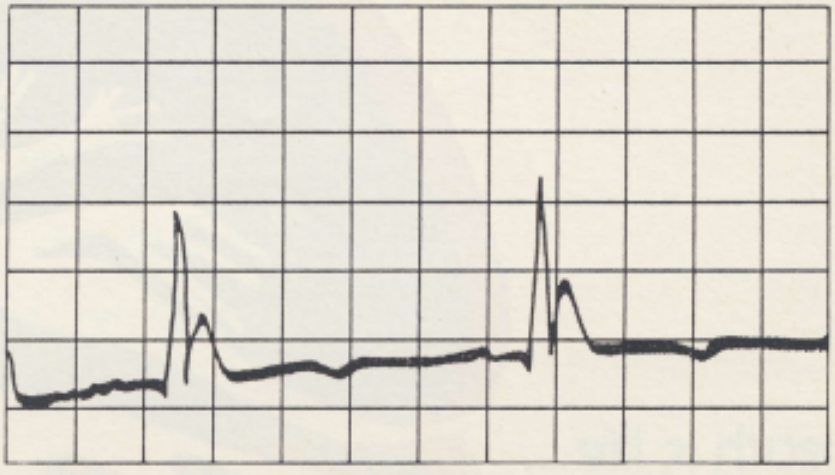

Figure 2. *The characteristic J or "Osborn" wave of hypothermia closely follows the QRS. It may be mistaken for a T wave with a narrow QT interval if the true T wave is obscured. The slightly rounded peak distinguishes it from the R' of right-bundle-branch block.*

Rewarming the patient

The choice of a specific warming method may depend on the duration and degree of hypothermia, the availability of resources, the time necessary to mobilise these resources, and contraindications that may be present in individual cases.

In general, most experts feel that rapid core rewarming is the most effective way to increase survival.[5, 6] Peritoneal dialysis, first used in 1967, has been extremely effective.[7]

Extracorporeal rewarming, by means of either femoral-femoral shunt or cardio-pulmonary bypass, has also been used successfully.[8, 9]

A relatively new technique, inhalation of heated aerosol, can be used in the field or office situation. Although the data are somewhat limited at this time, the proponents of heated aerosol claim that it is just as effective as the other methods.[10, 11]

You should anticipate postwarming complications such as ischemic damage to various parts of the body, pneumonia, adult respiratory distress syndrome, disseminated intravascular coagulation, acute tubular necrosis, and sepsis so that you can treat them appropriately if they develop. □

References

1 Altus, P, Hickman, JW, Pina, I, et al: Hypothermia in the sunny South. South Med J 73:1491, 1980.

2 What's warm enough for the young may kill the old (editorial). JAMA 239:180, 1978.

3 60F can induce accidential hypothermia in the aged. Internal Medicine News and Diagnosis News 14:19, February 6, 1981.

4 Gunby, P: Cold facts concerning hypothermia. JAMA 243:1403, 1980.

5 Reuler, JB: Hypothermia: Pathophysiology clinical settings, and management. Ann Intern Med 89:519, 1978.

6 Welton, DE, Mattox, KL, Miller, RR, et al: Treatment of profound hypothermia. JAMA 240:2291, 1978.

7 Reuler, JB, and Parker, RA: Peritoneal dialysis in the management of hypothermia. JAMA 240:2289, 1978.

8 Dorsey, JS: Venoarterial bypass in hypothermia (letter). JAMA 244:1900, 1980.

9 Towne, WD, Geiss, WP, Yanes, HO, et al: Intractable ventricular fibrillation associated with profound accidental hypothermia — successful treatment with partial cardiopulmonary bypass. N Engl J Med 287:1135, 1972.

10 Miller, JW, Danzl, DF, and Thomas, DM: Urban accidental hypothermia — 135 cases. Ann Emerg Med 9:456, 1980.

11 Edsall, DW: Treatment of hypothermia (letter). JAMA 244:1902, 1980.