Chapter 16

TREATMENT OF ACCIDENTAL HYPOTHERMIA

DANIEL F. DANZL, MD[*]; AND EVAN L. LLOYD, MD[†]

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MEDICAL OUTCOME AND DISPOSITION OF CASUALTIES

SUMMARY

[*]*Professor and Chair, Department of Emergency Medicine, University of Louisville, School of Medicine, Louisville, Kentucky 40292*
[†]*72 Belgrave Road, Edinburgh EH-12-6NQ, Scotland*

INTRODUCTORY OVERVIEW AND EPIDEMIOLOGY

Accidental hypothermia is geographically and seasonally pervasive, and can develop in virtually any military setting.[1–6] The treatment of hypothermia requires a flexible approach and familiarity with all available rewarming modalities. Hippocrates, Aristotle, and Galen each suggested a variety of remedies.[7] Not surprisingly, the cold has had a major impact on military history.[8,9]

Accidental hypothermia is defined as a core temperature below 35°C. At this temperature the body becomes progressively unable to generate sufficient heat to function efficiently.[10] In trauma, hypothermia has a deleterious effect on survival; the mortality rate approaches 40% if the core temperature is below 34°C. Hypothermia also contributes to the coagulopathies that accompany massive transfusion.[11]

Many variables contribute to the development of accidental hypothermia in the military service member.[12] Exposure, age, health, nutrition, or prescribed medications can decrease heat production, increase heat loss, or interfere with thermostability.[13,14] The healthy individual's compensatory responses to heat loss via conduction, convection, radiation, and evaporation are often overwhelmed by the exposure.[15] Military campaigns routinely present the potential for protracted exposure to the elements.

The term *cold stress* applies to any degree of environmental cold that causes the physiological thermoregulatory mechanisms to be activated. The severity of cold stress is not related to the absolute temperature alone but is also affected by air movement (wind or drafts) and moisture (humidity, rain, or dampness); a body will lose less heat at –10°C in still air than at +10°C with a 20 mph wind (see Figure 12-1 in Chapter 12, Human Psychological Performance in Cold Environments).[16] The sensation of cold is related to the lowered average skin temperature, but humans are more sensitive to change—and rate of change—in temperature than to any absolute value.

Routes of Heat Loss

The human body normally maintains a steady core temperature by balancing heat production to the rate of heat loss. The body, being warmer than the surrounding environment, loses heat through the normal physical mechanisms of conduction, convection, radiation, and evaporation, and the standard laws of physics apply. Radiation heat loss is maximal when the body is unclothed and erect and least when curled up and insulated. The amount lost by conduction depends on the temperature difference between two surfaces in direct contact. Conduction is the major route for heat loss during immersion in very cold water; even on land, wet clothing increases conductive loss. Evaporative heat loss occurs from the skin through insensible moisture loss and active sweating, through evaporation from wet clothing, and from the respiratory tract during warming and humidifying the inspired air. Large quantities of heat are required to convert a liquid into its gaseous phase (ie, the latent heat of evaporation). Convective heat loss is increased by limb movement and shivering, because the currents produced by the pendulum effect remove the warmed layer of air or water next to the skin; this effect is aggravated by a bellows effect of clothing. Both convective and evaporative heat losses are increased in windy conditions: the wind chill.

Regulation of Body Temperature

Body temperature is controlled through a central mechanism in the preoptic anterior hypothalamus (POAH) in the brain, which is not a simple ON/OFF device, although its function is similar to that of a thermostat. It functions more like the proverbial "black box," with a complex system of neurons cross-linking the sensory input and the affected output.[17] The thermostat-like POAH is activated by impulses from central receptors, which respond to changes in the temperature of the blood, and from peripheral receptors, which are located mainly in the skin. There are also spinal thermostatic reflexes, although these alone are insufficient to control body temperature. The POAH regulates the temperature of the body by adjusting heat production and heat loss, but the setting of the "thermostat" itself may be altered.[18]

The body responds to cold by constriction of the peripheral vessels mainly via the sympathetic nervous system and also through direct action of the cold environment on the blood vessels. Vasoconstriction is very effective in reducing heat loss by limiting blood flow to the periphery. This increases the depth of shell insulation and reduces the temperature differential between the skin and the environment. In fact, vasoconstriction can result in the outer 2 cm (1 in.) of the body's thermal conductivity being equivalent to that of cork.[18] Vasoconstriction, however, increases the risk of local cold injury. There is also a countercurrent exchange of heat

between the arteries and veins in the distal half of the limbs. Below a temperature of 10°C to 12°C, the peripheral vasoconstriction fails and alternating vasodilation and vasoconstriction occur. Actually, there may be very little increase in the volume of blood circulating in the skin during vasodilation[19] and, therefore, the insulating effect of the vasoconstriction is preserved. The head has minimal vasoconstrictor activity, and the rate of heat loss through the head increases in a linear manner between environmental temperatures of +32°C and –20°C. At –4°C, the resting heat loss from the head may equal half the total heat production.[16]

Heat production rises when muscle metabolism and tone increase. Any increase in heat production is always accompanied by a rise in oxygen consumption, and shivering may double or triple oxygen consumption. Deliberate activity also increases heat production 10- to 15-fold during hard physical exercise. In the cold, additional heat may be needed to maintain normothermia. Therefore, the oxygen consumption at any given any level of exercise will be higher in a cold environment than in a warm one.[20] This is seen clinically when angina develops during a particular level of activity in the cold but not at normal temperatures. Physical activity and shivering are not economical in the thermoregulatory sense because they are accompanied by an increased blood supply to the muscles, and therefore also by increased heat loss. In certain circumstances only 48% of the extra heat generated is retained in the body.

If hypoxia is present (eg, at high altitude), there will be a decrease in the total potential heat production and shivering may be inhibited.[21] Similarly, there is a limit to the maximum oxygen utilization. If an individual undertakes vigorous exercise in severe cold, the maximal oxygen uptake may be insufficient to provide for the high demand of both the exercise and the severe cold stress. As a result, unexpected and unsuspected hypothermia can develop despite vigorous muscular exercise. Finally, in individuals who are exhausted or suffering from malnutrition, heat production cannot increase because of the lack of substrate (fuel) for metabolism.[16]

Even at complete rest at a comfortable temperature, the vital functions of the body continue to generate heat. Reduced to a minimum, this is called the *basal metabolic heat*. This basal heat production increases if the body temperature rises, and it falls in hypothermia. During sleep the cerebral "thermostat" is reset to a new low level. Vasoconstriction is reduced with an immediate rise in skin temperature, while the metabolic rate is reduced.[16] Although ingesting ethanol produces a number of effects that increase the risk of hypothermia, the greatest danger reflects the decreased awareness of cold and increased bravado. Excellent physical fitness results in an increase in the maximum oxygen uptake; fit individuals can work and sleep better and are more comfortable in the cold.[20]

There are racial variations in the response to cold and, at the extremes of age, an increased risk of hypothermia. Many medical disorders predispose to hypothermia,[18] and a range of drugs, including anesthetics, increase the risk through impairing vasoconstriction or depressing metabolism. Even the mild degree of mental stress such as arithmetic increases the heat loss, as does nausea, vomiting, fainting, trauma, and hemorrhage.[16]

CHARACTERISTICS OF HYPOTHERMIA

Hypothermia is defined as subnormal body temperature, but thermally, the body can be divided into two zones: the core and the shell (see Figure 17-3 in Chapter 17, Cold Water Immersion). The *core* consists of the deeper tissues of the body including all the vital organs such as the heart and brain; the *shell* the remainder, including the skin. The core temperature is stable over a remarkable range of environmental thermal stressors. The depth and temperature of the shell, on the other hand, vary considerably according to the external environment, the degree of protection, and the activity of the individual. In extreme conditions, however, the tissues in the shell are thermally expendable. To allow for the diurnal variation of one to two Centigrade degrees, a person is considered to be in a state of hypothermia if the core temperature is below 35°C.[22] Obviously, medical officers should not view this threshold with the attitude that hypothermia does not exist when the core temperature is 35.5°C, and therefore the patient is safe, whereas a core temperature of 34.5°C is diagnostic of hypothermia and the patient is in danger. More fatalities occur with the many nonhypothermic, cold-related illnesses than occur as a result of primary hypothermia.[16,23] The American Heart Association classifies core temperatures of 34°C to 36°C as mild hypothermia.[24] But this classification also has limitations, as some normal individuals' core temperature varies diurnally from 35.5°C to 36°C (Table 16-1).[22]

A single measurement of core temperature is often used to classify hypothermia as mild, moder-

TABLE 16-1

SIGNS AND SYMPTOMS AT DIFFERENT LEVELS OF HYPOTHERMIA

Core Temperature (°C)	Description
37.6	"Normal" rectal temperature
37	"Normal" oral temperature
36	Metabolic rate increases to attempt to balance heat loss. Respiratory and pulse rate increase
35	Shivering maximum. Hyperreflexia, dysarthria, delayed cerebration present
34	Patients usually responsive and with normal blood pressure
33–31	Retrograde amnesia, consciousness clouded, blood pressure difficult to obtain, pupils dilated, most shivering ceases
30–28	Progressive loss of consciousness, increased muscular rigidity, slow pulse and respiration, cardiac arrhythmia develops, ventricular fibrillation may develop if heart is irritated
27	Voluntary motion lost along with pupillary light reflexes, deep tendon and skin reflexes
26	Victims seldom conscious
25	Ventricular fibrillation may appear spontaneously
24–21	Pulmonary edema develops; 100% mortality in shipwreck victims in World War II[1]
20	Heart standstill
17	Isoelectric electroencephalogram
15.2	Lowest infant accidental hypothermic patient with recovery[2]
3.7	Lowest adult accidental hypothermic patient with recovery[3]
9	Lowest artificially cooled hypothermic patient with recovery[4]
4	Monkeys revived successfully[5]
1 to –7	Rats and hamsters revived successfully[6]

Data sources: (1) Molnar GW. Survival of hypothermia by men immersed in the ocean. *JAMA*. 1946;131:1046–1050. (2) Nozaki RN, Ishabashi K, Adachi N. Accidental profound hypothermia. *N Engl J Med.* 1986;315:1680. (3) Gilbert M, Busund R, Skagseth A, Nilsen P, Solbo J. Resuscitation from accidental hypothermia of 13.7°C with circulatory arrest. *Lancet*. 2000;335:375–376. (4) Niazi SA, Lewis FJ. Profound hypothermia in man: Report of case. *Ann Surg.* 1958;147:254–266. (5) Niazi SA, Lewis FJ. Profound hypothermia in monkey with recovery after long periods of cardiac standstill. *J Appl Physiol.* 1957;10:137–138. (6) Smith AU. Viability of supercooled and frozen mammals. *Ann N Y Acad Sci.* 1959;80:291–300.

ate, or severe.[1,24] Frequently, the recommended treatment depends on the severity level of the hypothermia. This can be analogous to basing the decision to treat anemia purely on the measurement of the hemoglobin level. In fact, there is disagreement over the temperature ranges of the different grades (mild, 36°C–34°C, and severe, < 30°C[1,24,25]; or mild, 35°C–32°C, and severe, < 28°C[26]).

There are numerous nonspecific physiological effects of exposure to cold (Exhibit 16-1); three categories, however, are of particular relevance to the safe management of casualties with hypothermia: (1) energy reserves, (2) fluid balance, and (3) vascular responses[27]:

1. Energy reserves. The body responds to cold by increasing heat output and therefore the energy reserves are utilized. With rapid cooling as in cold water, the energy reserves are relatively undepleted and, once removed from the cold, the person will rewarm. With less severe cold, the body temperature will only fall when the energy reserves are exhausted. These individuals, with their reduced heat-generating capacity, may continue to cool, and die, even in a mildly cold environment.
2. Fluid balance. Cold-induced vasoconstriction shunts blood from the peripheral vas-

EXHIBIT 16-1

NONSPECIFIC EFFECTS OF COLD EXPOSURE

- Muscular
  - Muscle and tendon tears
  - Shivering
- Cardiovascular
  - Angina on decreased exertion
  - Rise in blood pressure; increases risk of
    - Stroke
    - Myocardial infarction
    - Heart failure
- Respiratory
  - Asthma
  - Rhinorrhea on return to warm room
- Peripheral Nervous System
  - Loss of manual dexterity
  - Loss of sensitivity
- Central Nervous System
  - Impaired coordination
  - Reduced visual acuity
  - Reduced alertness
  - Slowed reflexes
  - Increased mistakes
  - Misinterpretations of visual and auditory sensory input
  - Hallucinations
- Miscellaneous systemic physiological derangements such as coagulopathies, acid–base imbalance, and decompression sickness

Adapted with permission from Lloyd EL. ABC of sports medicine: Temperature and performance, I: Cold. *Br Med J*. 1994;309:534.

culature into the deep capacitance veins. The body attempts to reachieve equilibration of this relative central overload by means of a diuresis.[26,28] Water immersion, even thermoneutral, also causes a marked increase in diuresis.[29] As the core temperature falls, urinary output increases again owing to the loss of the ability of the distal renal tubules to reabsorb water[25] and to a decrease in sensitivity to antidiuretic hormone.[26]

Respiratory moisture loss is increased by exercise, especially in cold, dry air (eg, in the polar regions and at high altitude). Cold air is also dry and evaporation is rapid, and diaphoresis of 1 to 2 liters per day may be unnoticed. Even with total body dehydration, exercise causes an increase in the intravascular fluid volume,[30] thus increasing the cold diuresis and worsening any dehydration.

During exposure to cold there is also a shift of fluid from the intravascular space into the extracellular and then into the intracellular space.[31] The volume of the shift depends on the duration of the cold exposure. The body has not lost fluid, but the fluid is no longer immediately available to the circulation. This shift reverses during rewarming, and the circulating volume can rise to 130% above the normothermic volume.[28] This is dependent on the potential volume of fluid available, which in turn is related to the duration of cold exposure and the rate of rewarming. The fluid status of any hypothermic individual will depend on the relative importance of these responses.

3. Vascular responses. Vasoconstriction occurs during cooling, which reduces the volume of the vascular bed in active use. After the casualty is removed from the cold, the continuous cutaneous cold-stimulation ceases. The vasoconstriction then relaxes, thus increasing the active volume of the peripheral vascular bed. This volume is further increased by active surface warmth.

   Immersion in water produces a hydrostatic squeeze with effects similar to vasoconstriction. Removal from water will also increase the volume of the active vascular bed by removing the hydrostatic squeeze.[32,33] If there is insufficient available fluid to fill the increased active vascular capacity (eg, through dehydration owing to diuresis or fluid shifts), the central venous pressure will fall.

   In very prolonged exposure to mild cold, vasoconstriction, and therefore fluid loss from cold-induced diuresis, will be minimal. Loss of fluid from the vascular space due to fluid shifts will have been replaced by fluid intake, and any rapid return of fluid to the intravascular space during rewarming may result in fluid overload and pulmonary or cerebral edema, or both.

Types of Hypothermia

Using the above physiological parameters, it is possible to describe four different types of hypothermia: acute, subacute, subchronic, and submersion.[16,27]

Acute (Immersion) Hypothermia

In acute or immersion hypothermia, the core temperature drops despite maximal heat production. Hypothermia occurs before the body becomes exhausted, and the person will have very little difficulty in rewarming following removal from the severe environment. Because of the timescale, the shifts of body fluid will be minimal. Drowning, the commonest cause of death in water, may follow loss of consciousness due to hypothermia (Figure 16-1).

Hypothermia has been implicated in 20% of scuba diving fatalities, and hypothermia in cavers often involves immersion in cold water. Deep diving (below 150 m) with the use of oxyhelium gas breathing mixtures may also cause immersion hypothermia even in a dry diving chamber, because of the tremendous respiratory heat loss that occurs under these conditions due to the heat transfer capacity of the compressed gas. Another scenario is an injured climber, lying on the snow, unable to move owing to injuries, which also inhibit shivering and vasoconstriction.

Subacute (Exhaustion) Hypothermia

In subacute or exhaustion hypothermia, the cold is less severe and the heat production can maintain body temperature until exhaustion occurs and the supply of heat fails. Cooling only occurs when the energy reserves are exhausted. Therefore, spontaneous rewarming is less certain and cooling (including the

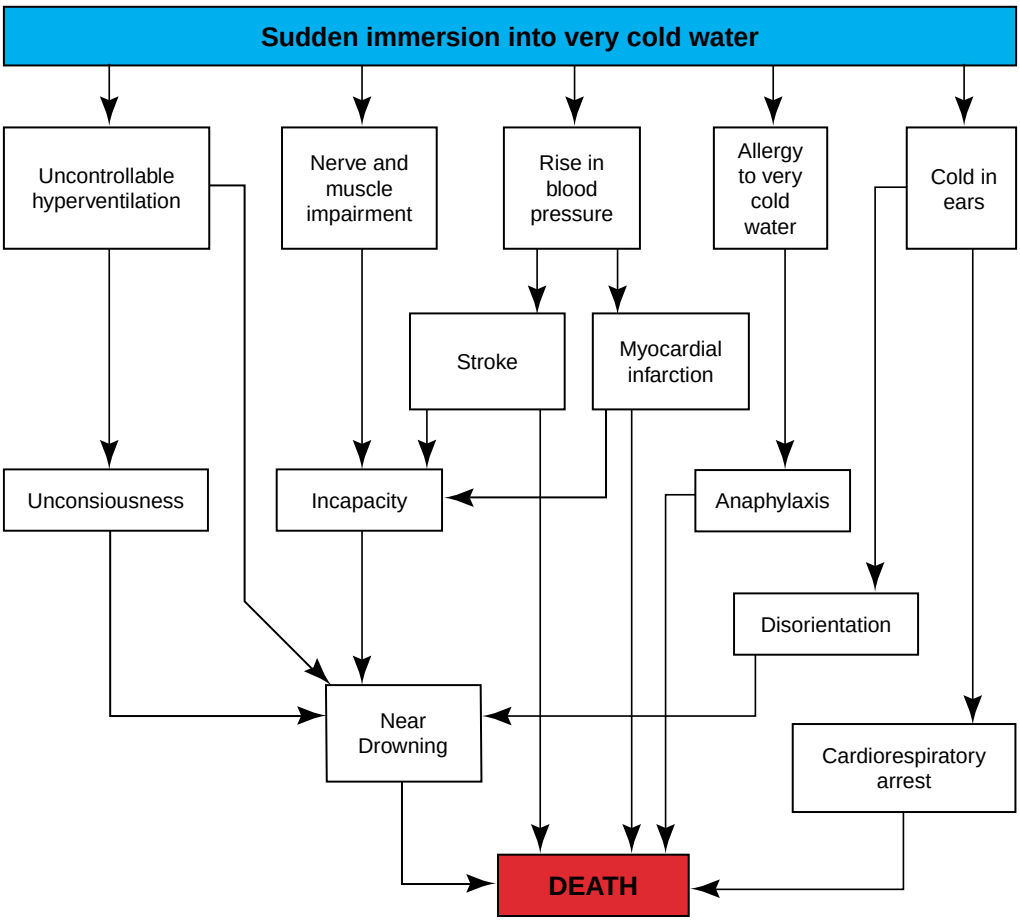

Fig. 16-1. Sudden immersion into very cold water initiates processes that can lead to death. Adapted with permission from Lloyd EL. ABC of sports medicine: Temperature and performance in cold. *Br Med J.* 1994;309:532

core) may continue even with very little continuing heat loss at the shell. Thermal protection must consider every avenue of heat loss and gain, because even small quantities of additional available heat may make the difference between life and death.

There will also have been fluid changes from diuresis and intercompartmental shifts, producing a net loss of circulating fluid volume. Removal from the cold will result in an increase in the active vascular bed with no increase in the circulating fluid volume. This combination will produce a relative hypovolemia and a drop in blood pressure, sometimes severe. This fall in blood pressure is often seen soon after a patient is admitted to the hospital.[34,35]

Subacute hypothermia is most commonly found among climbers and others exposed to a combination of moderate cold with wind and rain, as is common in the Scottish hills. In many of the deaths due to physical injury, the effects of cold exposure probably contributed to the fatal outcome. Death may also occur in endurance activities (not only in winter), and if someone falls overboard in relatively warm water.

Subchronic (Urban) Hypothermia

In subchronic or "urban" hypothermia, the cold, although relatively mild, is usually prolonged. The core temperature remains "normal" (35°C or above) possibly for weeks before a precipitant, such as an injury, results in hypothermia. This can result in vast intercompartmental fluid shifts. Any loss from the vascular space, however, has often been replaced through fluid intake, and vasoconstriction may not have occurred because the cold was relatively mild. Rewarming causes a reversal of the fluid shifts, the volume depending on the rate of rewarming. If the recirculation of the sequestered fluid exceeds the excreting ability of the kidneys, cerebral or pulmonary edema will occur.

Active rewarming of casualties with this form of hypothermia requires intensive therapy. The rate of rewarming must be conservative to avoid triggering cerebral or pulmonary edema,[36,37] a complication most common in the elderly living in poor housing or in those with malnutrition. When induced or iatrogenic hypothermia has been maintained over prolonged periods during hospital procedures, the complications that occur during rewarming are also probably due to these fluid shifts.[37]

Submersion Hypothermia

The fourth type of accidental hypothermia is called submersion hypothermia.[26,27] There are now a considerable number of reports in which patients have survived without oxygen for up to 60 minutes and yet have been successfully resuscitated without brain damage. A common factor is that all were totally submerged in ice-cold water. The younger the victim, the better the chance of survival. Children have a larger surface area-to-body mass ratio and will therefore cool faster than adults. Also, the head, with its very poor vasoconstrictor activity, is an important route for heat loss; and the younger the child, the larger the head in proportion to the rest of the body. Very rapid cooling could therefore be expected if the body is totally submerged, and this was proved in incidents in which time of submersion, time of rescue, and rescue temperatures are known.[16]

Signs and Symptoms

A cold environment, coupled with exhaustion, is a common cause of hypothermia in military settings. When the historical circumstances suggest significant exposure, the diagnosis is often simple. The presentation, however, may be quite subtle and deceptive. Cold tolerance is not uniform, and the depletion of energy stores and the type and degree of wetness of the clothing worn may vary significantly among a cohort exposed to identical climatic conditions (Exhibit 16-2).[38]

During military maneuvers, service members may simply appear uncooperative, uncoordinated, moody, or apathetic. Psychiatric symptomatology is common. Some individuals who remain functional under temperate circumstances decompensate in the cold.[39] Some alterations in mental status can endanger others. For example, the individual in a leadership role may exhibit impaired judgment, anxiety, perseveration, neurosis, or psychosis. Unusual risk taking and a peculiar flat affect have also been observed.[15]

Vague symptoms of mild hypothermia may also include hunger, nausea, confusion, dizziness, chills, pruritus, or dyspnea.[40,41] Signs such as slurred speech and ataxia often resemble those of a cerebrovascular accident, hypoglycemia, exhaustion, and heat illness. Some individuals have a decreased ability to sense cold and thus fail to seek a heat source or to take appropriate adaptive action. For example, the maladaptive phenomenon of paradoxical undressing is not uncommon. Rather than donning extra clothing, the hypothermic victim removes clothes and makes no effort to conserve heat or to move to a heat source.

The cold increases the preshivering muscle tone.

EXHIBIT 16-2

FACTORS PREDISPOSING TO HYPOTHERMIA IN THE MILITARY SETTING

Decreased Heat Production
- Insufficient fuel
- Hypoglycemia
- Malnutrition
- Extreme exertion
- Neuromuscular inefficiency
- Impaired shivering
- Inactivity
- Lack of adaptation
- Endocrinological failure
- Hypopituitarism
- Hypoadrenalism
- Hypothyroidism

Increased Heat Loss
- The environment, including wind-chill effects
- Immersion in water
- Radiation, convection, evaporation
- Induced vasodilation
- Effects from medications
- Toxins
- Burns, including severe sunburn
- Iatrogenic problems, including cold infusions and medical treatment for heatstroke

Impaired Thermoregulation
- Peripheral failure
- Neuropathies
- Acute spinal cord transection
- Diabetes
- Depressed central nervous system function
- Central nervous system trauma
- Cerebral vascular accident
- Toxins
- Metabolic derangements
- Subarachnoid hemorrhage
- Effects from medications
- Decreased heat production

This is ultimately manifested as muscular rigidity and paravertebral spasm. In military settings, losing the effective coordinated use of the hands can be devastating. In situations of enforced immobility, extremities may develop compartment syndromes after perfusion is reestablished in frostbitten (ie, frozen) extremities.

Neurological manifestations vary widely.[42] There is a progressive decrease in the level of consciousness that is proportional to the degree of hypothermia. [43,44] Some patients, however, are verbally responsive and display intact reflexes at 27°C to 25°C, core temperature levels generally considered to be severely hypothermic. The rest of the neuromuscular examination may suggest the diagnosis of hypothermia.[45] The patient's posture ranges from stiff to "pseudo-rigor mortis" to opisthotonos. Reflexes are usually hyperactive down to a core temperature of 32°C, then become hypoactive until they disappear around 26°C.

With luck, the history coupled with the constellation of suggestive physical findings will suggest the diagnosis. The most common signs of hypothermia are listed in Exhibit 16-3. These can only be a very general guide, as most of the signs have been established during experimental immersion and they are unlikely to be the same, or to occur at the same temperature, in the other types of hypothermia. Some of the early signs (eg, tachycardia and tachypnea) are likely to be of little value in the field, because they may also be produced by exertion or fear. Also, individuals show a great range of responses. For example, shivering is reported to cease at a core temperature between 32°C and 30°C.[29,46] However, mountain rescue teams note that many of their casualties do not shiver even with a core temperature of 35°C,[47] whereas shivering has been recorded below 29°C.[48] Similarly, neurological manifestations vary widely.[42] There is a progressive decrease in the level of consciousness[43,44] with consciousness being lost between 33°C[49] and 27°C.[50] Some patients, however, are verbally responsive and display intact reflexes at 26°C[7] and 24.3°C,[16] core temperature levels generally considered to be severely hypothermic. One of the earliest signs of hypothermia is a change in personality or behav-

EXHIBIT 16-3

PRESENTING SIGNS OF HYPOTHERMIA

Head, Eye, Ear, Nose, Throat
- Mydriasis
- Decreased corneal reflexes
- Extraocular muscle abnormalities
- Erythropsia
- Flushing
- Facial edema
- Epistaxis
- Rhinorrhea
- Strabismus

Cardiovascular System
- Initial tachycardia
- Subsequent tachycardia
- Dysrhythmias
- Decreased heart tones
- Hepatojugular reflux
- Jugular venous distention
- Hypotension

Respiratory System
- Initial tachypnea
- Adventitious sounds
- Bronchorrhea
- Progressive hypoventilation
- Apnea

Gastrointestinal System
- Ileus
- Constipation
- Abdominal distention or rigidity
- Poor rectal tone
- Gastric dilatation

Genitourinary System
- Anuria
- Polyuria
- Testicular torsion

Neurological Systems
- Depressed level of consciousness
- Ataxia
- Hypesthesia
- Dysarthria
- Antinociception
- Amnesia
- Initial hyperreflexia
- Anesthesia
- Hyporeflexia
- Areflexia
- Central pontine myelinosis

Psychiatric Signs
- Impaired judgment
- Perseveration
- Mood changes
- Peculiar "flat" affect
- Altered mental status
- Paradoxical undressing
- Neuroses
- Psychoses
- Suicide
- Organic brain syndrome

Musculoskeletal System
- Increased muscle tone
- Shivering
- Rigidity/pseudo–rigor mortis
- Paravertebral spasm
- Opisthotonos
- Compartment syndrome

Dermatological Conditions
- Erythema
- Pernio
- Pallor
- Frostnip
- Cyanosis
- Frostbite
- Icterus
- Popsicle panniculitis
- Sclerema
- Cold urticaria
- Ecchymosis
- Necrosis
- Edema
- Gangrene

Adapted with permission from Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies.* 3rd ed. St Louis, Mo: Mosby; 1995: 63.

ior, but unfortunately, the victim is likely to be the last person to notice the change.

Pathophysiology

Cardiovascular System

Despite the physiological fact that hypothermia protects the brain from the effects of anoxia, the clinical experience is that survival in hypothermia is almost completely dependent on having sufficient cardiac function and output to maintain adequate perfusion of the heart and brain; therefore, cardiac function has more relevance to survival than brain temperature. Predictable cardiovascular responses occur during hypothermia. A progressive bradycardia develops after the initial tachycardia. The pulse usually decreases by half at 28°C. If the tachycardia is inconsistent with the core body temperature, the medical officer might consider other possible conditions such as hypoglycemia or hypovolemia from trauma or dehydration.[14]

The electrocardiographic (ECG) features of hypothermia are quite distinctive.[51] The Osborn (J) wave is seen at the junction of the QRS complex and the ST segment (Figure 16-2).[52] However, a J wave is present in only about 80% of patients with hypothermia and may also be present in patients with sepsis and lesions of the central nervous system. Although the J wave may be helpful in diagnosis, it is not prognostic. It may appear at any temperature below 32°C. The size of the J wave increases with temperature depression but is not related to arterial pH.[53]

Some J waveform abnormalities can simulate a myocardial injury current. Hypothermic ECG changes are not yet programmable for computer interpretation. As a result, reliance on computer interpretations of a field 12-lead ECG can result in the misdiagnosis of a myocardial infarction. Thrombolytic therapy under hypothermic conditions is unstudied but would probably exacerbate preexistent coagulopathies.[54]

All atrial and ventricular dysrhythmias are extremely common in moderate and severe hypothermia. Reentrant dysrhythmias result from the decreased conduction velocity coupled with an increased myocardial conduction time and a decreased absolute refractory period. Independent electrical foci also precipitate dysrhythmias. Cardiac cycle prolongation occurs because the conduction system is more sensitive than the myocardium to the cold.[55]

The fluctuations of pH, electrolytes, and available oxygen and nutrients also alter conduction. The PR

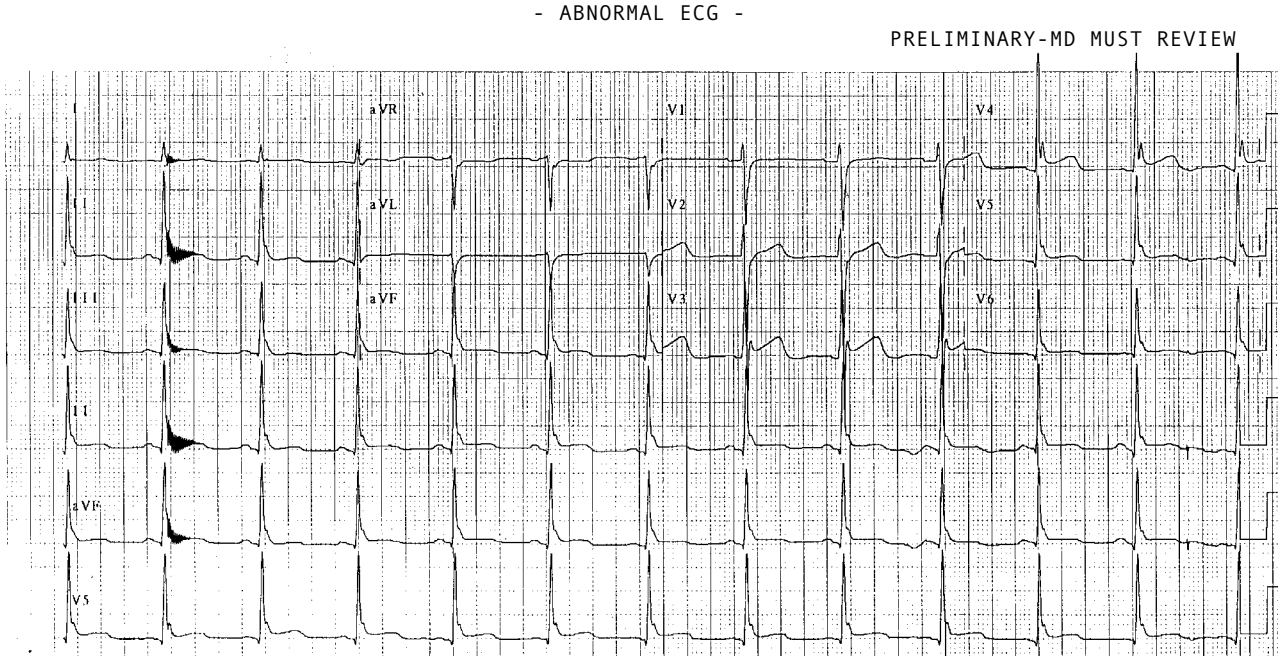

Fig. 16-2. A 12-lead electrocardiogram displaying excellent J waves (also called Osborn waves) on V4. The patient was a 32-year-old man whose core temperature at the time this electrocardiogram was made was 31°C. Note that although the computer recognized the reading as abnormal, it was not set to diagnose hypothermia.

interval, then the QRS interval, and most characteristically, the QT interval, are all prolonged. Thermal muscular tone may obscure P waves or produce artifacts even in the absence of obvious shivering.

There is a false assumption that a person who is removed from the cold stress is safe, but deaths still occur after rescue. Continuous cooling frequently causes the heart to become directly asystolic, without antecedent ventricular fibrillation (VF). The development of VF in the field is also a major concern. In many cases VF is probably an iatrogenic result of treatment attempts. Other putative explanations include tissue hypoxia, physical jostling, exertion, electrophysiological or acid-base disturbances, and autonomic dysfunction. The cold-induced increase in blood viscosity is accompanied by coronary vasoconstriction that can also exacerbate cardiac hypoxia.[26,56]

The ventricular arrhythmia threshold decreases in hypothermia, and VF and asystole occur spontaneously when the core temperature falls below 25°C. VF can also result from an independent focus or via a reentrant phenomenon. When the heart is cold, there is a large dispersion of repolarization, which facilitates the development of a conduction delay.[57] Additionally, the action potential is prolonged, which increases the temporal dispersion (ie, various cells recover at different rates) of the recovery of excitability.[58]

The term *core temperature afterdrop* refers to a further decline in the core temperature after removal from the cold.[59,60] A number of processes contribute to afterdrop, including simple equilibration across a temperature gradient and circulatory changes.[19,61] Countercurrent cooling of the blood that is perfusing the cold extremity tissues causes the temperature of the core to decline until the gradient is eliminated.

Active external rewarming of the extremities can obliterate peripheral vasoconstriction and reverse arteriovenous shunting.[62,63] The size of the afterdrop varies depending not only on the temperature differential between shell and core but also on the site measured and rewarming method used.[16] This is most vividly demonstrated by Hayward.[64] He measured his own esophageal, rectal, tympanic, and cardiac temperatures (via flotation tip catheter) after cooling in 10°C water on three different days. During spontaneous rewarming there was a normal size and duration of the afterdrop in the rectum but a negligible drop in the pulmonary artery. Following warm bath immersion-rewarming, the rectal afterdrop was reduced but there was an increased afterdrop in the pulmonary artery, although it was still relatively small. More importantly, warm bath immersion-rewarming caused a 30% fall in mean arterial pressure coupled with a 50% decline in peripheral vascular resistance. Similarly, Harnett[65] observes the largest core temperature afterdrops when subjects are rewarmed with plumbed garments and heating pads.

Core temperature afterdrop is a clinically relevant consideration when treating patients with a large temperature gradient between the core and the periphery. This is common following chronic exposure. Major afterdrops will also occur in severely hypothermic service members if their frozen extremities are thawed in warm water prior to thermal stabilization. After extinguishing peripheral vasoconstriction, the sudden central return of cold, hyperkalemic, acidotic blood may overtax the thermally depressed heart. The danger may be more from this biochemical insult than from temperature drop.

There is disagreement about the clinical significance of afterdrop. It is true that the lower the cardiac temperature, the more susceptible it is to VF; however, death following rescue may be due to other factors in addition to afterdrop.[27]

Central Nervous System

The numbing cold is a progressive depressant of the central nervous system. Like the heart, the brain has a critical period of tolerance to hypothermia. There are temperature-dependent neural enzyme systems that are unable to function at temperatures that are well tolerated by the kidney.[66] The cerebrovascular autoregulation remains intact until the core temperature falls below 25°C. This protective autoregulation maintains the beneficial disproportionate redistribution of blood flow to the brain.

Respiratory System

Cold stress leading to mild hypothermia initially stimulates respiration. This is followed by a progressive decrease in the respiratory minute volume (RMV) that is proportional to the decreasing metabolism. The normal stimuli for respiratory control are also altered in severe hypothermia. Carbon dioxide production decreases 50% with a fall in temperature of eight Centigrade degrees.[10,67] As a result, overzealous assisted ventilation will induce a respiratory alkalosis sufficiently severe to cause ventricular irritability.

Numerous pathophysiological factors adversely affect the respiratory system. These include viscous bronchorrhea, decreased ciliary motility, and noncardiogenic pulmonary edema.[68]

Renal System

As noted earlier, simple exposure to cold induces a diuresis despite the state of hydration. This is a major concern during prolonged expeditions in the cold. The initial peripheral vasoconstriction results in an increased amount of blood in the "central" circulation. This stimulates a diuresis. Hypothermia depresses renal blood flow, reducing it by 50% at 27°C to 30°C. The kidneys then excrete a large amount of dilute urine, termed the *cold diuresis*. This cold diuresis is essentially glomerular filtrate that does not efficiently clear nitrogenous waste products. Cold diuresis decreases the blood volume and results in progressive hemoconcentration.[69]

REWARMING OPTIONS

Rewarming is the common goal in the management of the hypothermic patient. The various options should be considered, and rewarming should be done, at any site where victims of hypothermia are found–from the field to the most elaborately equipped critical care facility. Therefore, it is necessary to discuss all the various options–spontaneous, active external, and active core–for rewarming before discussing the practical choices to be made, whether the setting is primitive or sophisticated.

There are many reports of successful rewarming using many different methods, singly or in combination. However, most reports are of individual cases managed in an emergency department or intensive care unit. A review of the literature[16] and experimental work[26] suggests that all methods of rewarming are effective and safe provided the patient is under intensive care monitoring. The best method therefore depends on the environment, and what is available.

The key initial treatment decision is to identify any factors that mandate active rewarming (Exhibit 16-4). Below 30°C to 32°C, humans are functionally poikilothermic. There is no shivering thermogenesis. When the core temperature exceeds 32°C, the major source of heat production is shivering thermogenesis unless there is complete glycogen depletion.[70,71]

The direct transfer of exogenous heat to the patient is considered active rewarming. It can be accomplished by a variety of external or internal techniques.

EXHIBIT 16-4

INDICATIONS FOR ACTIVE REWARMING

- Poikilothermia (core temperature below 32.2°C)
- Traumatic or toxicological vasodilation
- Inadequate rate of rewarming
- Cardiovascular instability
- Endocrinological or metabolic fuel insufficiency
- Impaired thermoregulation

Adapted with permission from Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach P, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St Louis, Mo: Mosby; 1995: 73.

Spontaneous Rewarming

Spontaneous rewarming (also called passive rewarming) minimizes the normal processes of heat dissipation from evaporation, convection, and radiation. This technique simply involves covering the patient with an insulating material in a favorable atmospheric condition.[72] Ideally, the ambient temperature should exceed 21°C with the air stationary, which allows less heat to be lost due to conduction, convection, and radiation. This method of rewarming is noninvasive and is the treatment of choice for most previously healthy patients with mild hypothermia. The patient must be sufficiently healthy and nourished to generate enough metabolic heat to maintain an acceptable rate of spontaneous rewarming. This rewarming option should be initiated unless there are indications for active rewarming.[73,74]

Spontaneous rewarming is the option selected by rescuers as soon as the hypothermic victim is found. To allow the casualty to rewarm spontaneously from endogenous heat production, further heat loss is prevented by enclosing him or her in a casualty bag or sleeping bag, or by using any available material.[16] Rescuers should keep in mind that the head is a major source of radiative heat loss (the original nightcap was not Kentucky bourbon but a hat worn to bed). Wet clothing should be removed only after the patient has reached a warm, dry, sheltered environment; removal of wet clothing in the field can produce very rapid heat loss.[75] If shelter is not available, then extra layers of insulation should be applied on top of the victim's clothing, even if the

clothing is wet.[76] The space blanket (metallized plastic sheeting) is often recommended as part of the insulating package, but this was shown theoretically and experimentally to be no better than a similar thickness of polyethylene, which is much less costly.[16] The victim should also be insulated from the ground with branches, leaves, or spare clothing.[75] Windproof and waterproof outer protection should be provided (eg, a polythene sheet or tent or, in the field, in a snow hole or behind a large boulder). Once this protective cocoon is in place, any disturbance risks further heat loss and a marked delay in rewarming. A fine point of the technique is that the hands and feet should be kept cool, with the casualty's hands at the sides and not on the abdomen. Warm hands and feet reduce the stimulus for heat production and will allow reduction of vasoconstrictor tone, thus increasing heat loss and increasing the risk of vasomotor collapse.[75]

Even in a warm environment the casualty should be kept insulated to prevent the surface warmth from causing a further increase in vascular dilation and a catastrophic drop in blood pressure. There is a very high mortality rate among casualties–especially if they are profoundly hypothermic–who are left exposed in a warm room.[34,77] If the casualty is shivering, then rewarming will be fairly rapid. Shivering may be dangerous, however, especially in the presence of hypovolemia such as that following trauma,[11] because shivering increases oxygen consumption and increases peripheral blood flow with the risk of hypotension.

It is not known if, without shivering thermogenesis, there is a core temperature below which spontaneous rewarming cannot occur. One casualty, however, is documented to have rewarmed spontaneously from an estimated core temperature of 18°C.[78] Medical officers should remember that depression of metabolic heat by drugs, illness, and injury may cause a lower core temperature.[16,33,36] The potential heat production will also vary depending on the victim's age and the etiology of hypothermia. Nevertheless, some metabolic heat is always being produced and therefore, theoretically, provided heat loss is totally prevented, the victim should inevitably rewarm whatever the core temperature. The difficulty is in providing perfect insulation. The route of heat loss most often overlooked is that of breathing[16] (discussed below). Prevention of this loss has converted a static core temperature (ie, poikilothermia) into a rising core temperature.[34]

Even in the hospital, apparently similar patients vary greatly in their metabolic heat production, as shown by the variable rate of spontaneous rewarming.[34,36] If the environmental cold is severe, the insulation is poor or incomplete, the metabolism is depressed through drugs or low body temperature, or if there is complete glycogen depletion,[70,71] then endogenous heat production may be insufficient to compensate for the continued heat loss and the victim may fail to rewarm–or may in fact continue to cool.

Active External Rewarming

Optimal candidates for active external rewarming are previously healthy patients with acute hypothermia. A variety of methods can be used to conduct heat directly to the skin. Options include the use of plumbed garments (in which warm fluids are recirculated through embedded tubing), hot water bottles, heating pads, commercial heating beds, blankets, and radiant sources.[16] Forced-air warming mattresses and blanket systems efficiently transfer heat. One concern in the use of the following techniques is that vasoconstricted, hypoperfused skin is susceptible to thermal injury.[16,62,79–81] Immersion in water at 40°C is another option for this patient population, in whom minimal pathophysiological circulatory changes have occurred. Unstable patients or those with external injuries are not candidates for immersion. Disadvantages of immersion in a warm water bath include the inability to monitor or resuscitate the patient in water and the difficulty of performing cardiopulmonary resuscitation (CPR) on a floating body.

Some reports[3,15,82] have linked active external rewarming with sudden vasodilation accompanied by shock. External rewarming can increase the requirement for bicarbonate and crystalloid administration during resuscitation. Peripheral metabolic demands are also increased. The ventricular arrhythmia threshold decreases because of the myocardial thermal gradients.

Classically, if active external rewarming is chosen, the heat source should be applied only to the thorax, with the extremities left vasoconstricted. Application of heat to the extremities may increase the cardiovascular load by increasing the metabolic requirements of the peripheral musculature. The depressed cardiovascular system may not be able to meet the increased demands, and cardiovascular collapse can occur. Less favorable results are seen with active external rewarming if the heat is applied to the periphery, particularly in patients with chronic hypothermia. In acute hypothermia, though, this factor may not be clinically relevant.

Combining truncal active external rewarming with core rewarming has been successful. Some

authors believe that providing heated humidified oxygen and warmed intravenous fluids, in addition to active external rewarming, may anticipate and avert hypoxia, metabolic acidosis, core temperature afterdrop, and hypotension.[83]

Surface heating is often used because the rescuers believe that they must do something active to help the victim. With surface warming, however, the warmer superficial tissues have an increased oxygen demand (a rise of 10 Centigrade degrees in tissue temperature produces a 100% increase in oxygen demand[18]). Unfortunately, in cold blood the dissociation curve for oxyhemoglobin shifts to the left, resulting in a firmer binding between oxygen and hemoglobin, which makes less oxygen available for the tissues.[18] On the other hand, severe hypothermia results in both respiratory and metabolic acidoses that shift the dissociation curve toward the right. Another consideration is that oxygen is more soluble in plasma at low temperatures, and at a body temperature of about 10°C the partial pressure of the dissolved oxygen should be adequate for tissue demands even in the absence of hemoglobin.[84] In addition, superficial perfusion is impaired in hypothermia. The combination of warm tissues and impaired perfusion with cold blood may produce hypoxia of the superficial tissues and be the cause of the acidosis seen during surface warming.

Shivering will aggravate any metabolic acidosis. Warmth on the skin will depress shivering and reduce oxygen consumption but at the expense of reduced heat production.[85] Interestingly, radiant heat applied to the blush area of the head and neck will inhibit shivering and reduce oxygen consumption[86] without markedly impairing vasoconstriction. If there is no circulation (or very little) through the skin, as may be the case with cardiac depression or arrest, surface warming is ineffective and may cause burning even at "baby bath" temperatures.[16]

The hot bath is the fastest method of rewarming a mildly hypothermic person, but that technique has many disadvantages and limitations even when used with modern, "safe" guidelines. The main benefit occurs only within 20 minutes after the victim has been removed from the cold. This technique should be used only for mildly hypothermic casualties who are conscious, shivering, and uninjured, and who can get into the bath with minimal assistance.[76]

The current recommendations are that the temperature of the bath should approximate but not exceed 40°C, that is, "the elbow comfort temperature."[60(p1238)] The 40°C temperature should be maintained by constant stirring, and by adding hot water as necessary. This technique requires large quantities of hot water–more than the ordinary domestic hot water supply even if the hot water system is active when the rescue team reaches the nearest house. Heavy outer clothing should be gently removed before the casualty is immersed to the neck. Assistance should be given with removing the rest of the clothing once the casualty is comfortably settled in the bath. Almost immediately on immersion shivering will stop but this is not an indication for removing the casualty. The casualty should be helped out of the bath when he or she is adequately rewarmed (ie, pink all over), dried, covered with blankets, and kept lying flat. Remove the casualty from the bath if diaphoresis develops.

For the sake of completeness, body-to-body rewarming is included here but is *not* recommended. A rescuer, stripping to underwear,[11] gets into the same sleeping bag as the victim. Most sleeping bags will admit only one body, and transport of two persons would be almost impossible. The physiological effects of body-to-body rewarming are similar to those of mild surface warming.[87] Although it is part of rescue fantasies, this technique is of no practical value.

Radiant heat, in the form of an open fire, is dangerous and can be lethal. During the retreat from Moscow in 1812, Napoleon's surgeon, Baron Dominique-Jean Larrey, noticed that hypothermic solders died if they were close to the camp fires.[88] A radiant heat cradle[16] has also been used successfully, but this device requires electricity and the patients are then rewarmed in a hospital intensive care unit. Heating pads and hot water bottles placed at the neck, axilla, and groin can also be used. Plumbed garments are effective but are rare and expensive, and restrict access to the patient. Other methods of surface heating are likely to be unavailable or dangerous on the battlefield.

Active Core Rewarming

With active core, or central, rewarming, heat is supplied to the core of the body first, and rewarming proceeds outward, from the core to the shell. The core organs, which constitute approximately 8% of the total body weight, contribute 56% of the heat production in basal metabolism at normothermia, and a higher percentage in hypothermia. This is because the muscles and superficial tissues have cooled more than the core and are therefore producing a lower percentage of the total body heat production. As the temperature of a tissue rises, the heat generated also rises rapidly. Therefore, by concentrating the heat gain in the core, the thermal

benefits will be significantly greater than calculations alone would suggest.[16]

Numerous alternatives have been explored to achieve active rewarming of the core, in which the heat is delivered internally. These techniques may minimize rewarming collapse in victims whose core temperature is below 30°C.

Airway Warming

The use of heated, humidified air and oxygen has been studied extensively both in the field and in the healthcare facility. The amount of heat delivered is small; the main benefit is from the preservation of heat and humidity, which otherwise are lost during breathing. This method is effective only when combined with insulation of the rest of the body. Rewarming via the airway is indicated as an adjunct in all cases of moderate hypothermia.[16,34,89] The main advantages of delivering heated, humidified oxygen to the victim include its noninvasiveness, assurance of adequate oxygenation, and avoidance of core temperature afterdrop. Some of the additional benefits are the stimulation of pulmonary cilia, a decrease in pulmonary secretion viscosity, and a reduction of the cold-induced bronchorrhea.[90] Pulmonic absorption occurs without adverse effects on surfactant or increased pulmonary congestion.

A sufficient RMV and complete humidification are necessary for maximal heat delivery.[91] The heat transfer from the inhalation of water-saturated heated inhalant equals the number of liters ventilated per minute multiplied by the heat yielded as water vapor condenses and cools to the current core temperature. Ventilation with warm, dry air provides negligible heat because of the low thermal conductivity of dry air. The medical officer should anticipate a rewarming rate of 1 to 2.5 Centigrade degrees per hour, depending on the delivery technique used (an endotracheal tube is more rapid than a mask).[15] As the ventilatory rates increase by 10 L/min at 42°C, the core temperature rises an extra 0.3 Centigrade degrees per hour.[92]

The victim with a core temperature of 28°C, whose normal basal metabolic rate would yield 70 kcal/h at 37°C, endogenously generates only 30 kcal/h. Water vapor transports the majority of the heat, and the latent heat of vaporized water is 540 kcal/g if condensation occurs in the lung. At this core temperature, the rate of rewarming by heated ventilation will equal that of the endogenous heat production.[93]

The efficiency and influence of heated mask ventilation (ie, the casualty receives heated air or oxygen via a mask) continues to be debated.[94–96] However, only one study[94] has compared airway warming with passive rewarming in the same patients during a single admission. Airway warming accelerated rewarming to a statistically significant degree, compared with passive rewarming, whether it occurred before and/or after the period of airway warming. There is a thermal countercurrent heat exchanger in the cerebrovascular bed of humans known as the rete mirabile. This heat exchanger may preferentially rewarm the brainstem. Another warming option is the use of heated air or oxygen delivered via a face mask under continuous positive airway pressure.[97]

Another benefit of airway warming is the maintenance of sufficient oxygenation in moderate and severely hypothermic victims. In patients on cardiopulmonary bypass cooled to 28°C to 30°C, the "functional" value of hemoglobin is 4.2 g per 10 g of hemoglobin at normothermia.[98] That is, during hypothermia, the capacity of hemoglobin to unload oxygen to the tissue is low.

Complete airway protection averts aspiration. Hypothermia is associated with ileus, bronchorrhea, and depressed protective airway reflexes. Although the airway warming technique provides less heat than some of the other forms of active core rewarming, it is safe, noninvasive, and practical.

During spontaneous or assisted ventilation with heated ventilation, there is the flexibility to alter the fraction of inspired oxygen (FIO_2), monitor airway pressure, and deliver continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP). Heated inhalation also suppresses the amplitude of shivering, which is advantageous in patients with severe hypothermia because of the decreased metabolic demands of the periphery.[90,99]

The technique for patients with spontaneous respirations requires a heated cascade nebulizer. An immersion heater can be connected to a hose with a warming wire. Because patients with a depressed level of consciousness will not complain of pain if the pharynx is burned, the temperature of the inspired air must be checked frequently with an inline temperature probe.[100] The gas temperature is maintained at 42°C to 45°C. Most heater modules require modification to allow these temperatures to be achieved, and they should be so labeled to avoid routine use.[101]

Airway warming is similar to spontaneous rewarming in that the main thermal input comes from the body's own metabolic heat production.[102,103] Even with perfect surface insulation the victim still loses heat through breathing. Airway warming is therefore only of value as an adjunct when the

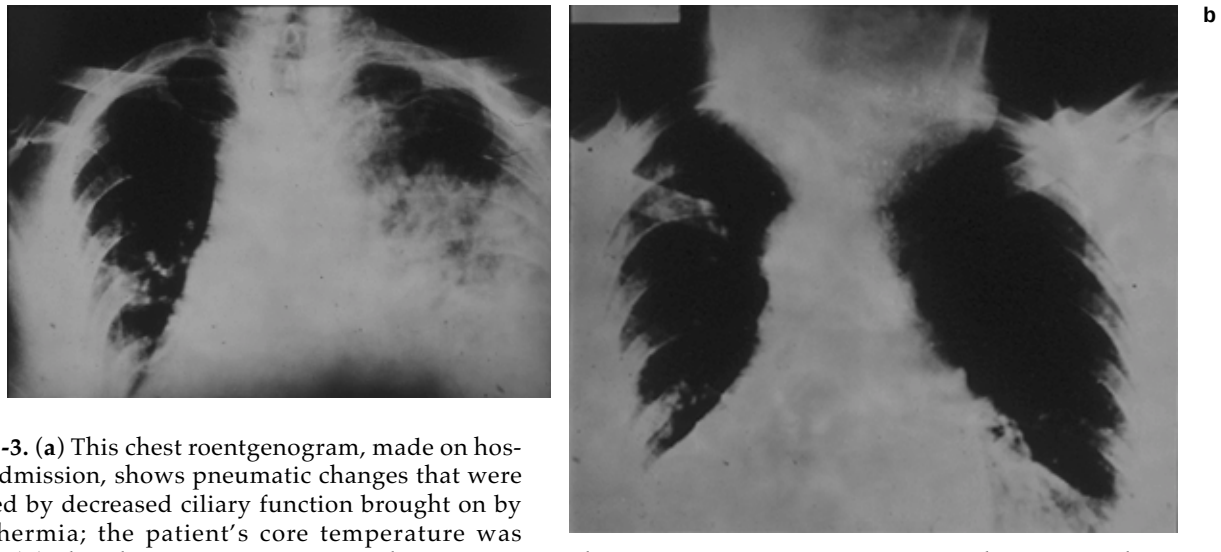

Fig. 16-3. (**a**) This chest roentgenogram, made on hospital admission, shows pneumatic changes that were induced by decreased ciliary function brought on by hypothermia; the patient's core temperature was 32.5°C. (**b**) This chest roentgenogram of the same patient shows marked improvement on reaching normothermia after 12 hours of airway warming treatment. The pneumonia-like pulmonary changes resolved purely with rewarming. taken together, these roentgenograms demonstrate the important point that clinical management decisions should not be taken while the patient is hypothermic. Reprinted with permission from Lloyd EL. *Hypothermia and Cold Stress.* Hampshire, Enbgland: Chapman & Hall; 1986:53.

whole body is insulated. Airway warming and the use of airway warming devices are now widely recommended[24,76,100,101,104-108] in the management of accidental hypothermia (Figure 16-3).

In the field, the aim of airway warming is to produce warm, moist air (which should not be above 45°C to avoid thermal burns to the face and pharynx). A variety of designs of potentially portable

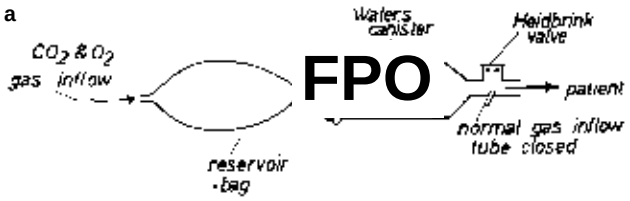

Fig. 16-4. (a) Simple circuit for providing airway warming from an anesthetic machine. The patient may be connected by face mask or endotracheal tube as appropriate, and ventilation assisted if required. INSTRUCTIONS FOR USE: The carbon dioxide is allowed in as a flow rate of 3 to 5 L/min, and oxygen at 0.5 to 1 L/min. Once the heat in the Waters canister has reached the desired level, the carbon dioxide is discontinued and only the oxygen continued. The desired temperature feels comfortably hot to the bare hand on the outside of the metal Waters canister. Additional carbon dioxide is added as the temperature of the canister falls. **(b)** The system is portable and rugged enough to be carried to various environments. here, the portable airway warming device, showing its insulated canister and breathing tube, is on Mount Everest; the Khumbu icefall is seen in the background. Diagram (a): Reprinted with permission from Lloyd EL, Conliffe N, Orgel H, Walker P. Accidental hypothermia: An apparatus for central re-warming as a first-aid measure. *Scott Med J.* 1972;17:89. Photograph (b): Reprinted with permission from Lloyd EL. *Hypothermia and Cold Stress.* Hampshire, England: Chapman & Hall; 1986:224

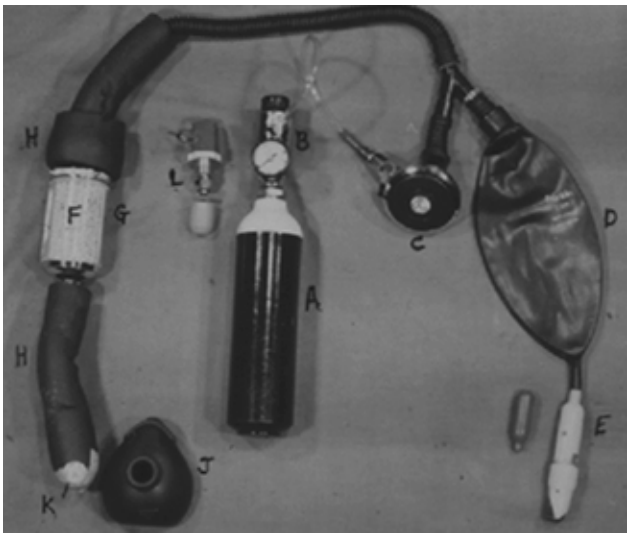

Fig. 16-5. Lightweight Lloyd portable airway warming equipment. (**A**) Oxygen cylinder. (**B**) First-stage reducing valve and gauge. (**C**) Demand-reducing valve with manual override to allow 2-L reservoir bag (**D**) to be filled if ventilation assistance is required. The demand valve may be replaced by a button valve. (**E**) Corkette (Sparklet corkmaster) with the distal portion of the needle removed and inserted into the tail of the reservoir bag. Spare sparklet alongside. (**F**) Soda lime. (**G**) Pediatric Waters canister. (**H**) Insulation: neoprene foam tubing. (**J**) Face mask. (**K**) Thermometer registering mean air temperature at mask inflow. (**L**) Adaptor for refilling small oxygen cylinder from a large cylinder.
INSTRUCTIONS FOR USE: Empty one sparklet cylinder into the system by using the Corkette (E). Open the valve (B) on the oxygen cylinder (A). Apply the face mask (J) to the patient. If appropriate, the face mask may be replaced by an endotracheal tube. The reservoir bag (D) should be inflated by depressing the center of the demand valve (C) or the alternative button valve. Thereafter the system will work on demand or by intermittent refilling of the reservoir bag. The thermometer (K) should be observed. This will rise steadily in the Corkette (E) and depress the lever to allow carbon dioxide to flow for 3 sec. This should be repeated whenever the airflow temperature falls to 35°C. Where possible, the Waters canister (G) should be vertical rather than horizontal to reduce the risk of gases channeling along the side of the canister. The gauge on the oxygen cylinder should be checked regularly and the cylinder should be refilled from a large cylinder after use. The soda lime (F) should be replaced in the Waters canister after use, ensuring, by tapping and shaking, that the canister is completely full, to reduce the risk of gases channeling along the side if the canister is horizontal. The equipment, which weighs 3 kg (7lb), can be carried in any convenient container. The device should provide warmed, moist oxygen for 2 h before the soda lime needs to be replaced. Reprinted with permission from Lloyd EC. *Hypothermia and Cold Stress*. Hampshire, England: Chapman & Hall; 1986: 202.

equipment[109] include electrically operated hospital humidifiers (not nebulizers), gas-heated humidifiers, and a design that utilizes the chemical reaction between soda lime and carbon dioxide (Figure 16-4). This latter system was carried on Mount Everest by the successful Bonnington-led expedition and, when tested, worked satisfactorily at 20,000 feet.[16] It is also used by cave rescue teams in the United Kingdom to treat casualties underground, and is being evaluated and developed by the Swiss Air Rescue service. A small version weighs 3 kg (Figure 16-5). A simple first aid measure is to use a heat and moisture exchanger (Figure 16-6) with mask and tubing covered by clothing (even a loose scarf over the nose and mouth can be used provided the airway is not compromised), so the victim can breathe the prewarmed boundary layer of air close to the skin.[109] This equipment is inexpensive, weighs only a few ounces, and should become part of climbers' first aid equipment.

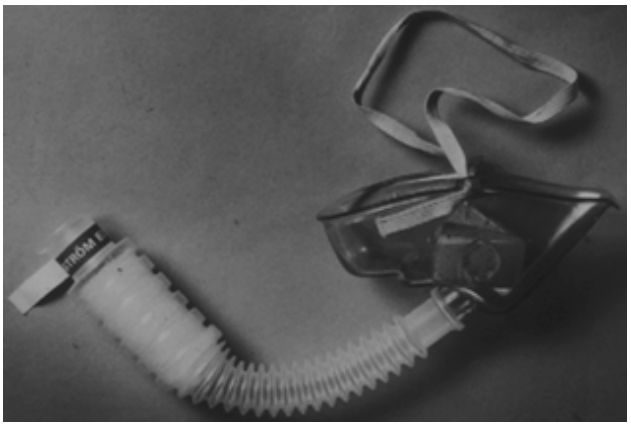

Fig. 16-6. Condenser humidifier with face mask attached. This airway rewarming equipment can be used in the emergency treatment of accidental hypothermia. The end of the humidifier should be placed under the clothing next to the skin, and the whole device, including the mask, should be covered (eg, with a scarf). Reprinted with permission from Lloyd EL. Equipment for airway warming in the treatment of accidental hypothermia. *J Wilderness Med.* 1991;2:347

Peritoneal Dialysis

Peritoneal dialysis delivers dialysate heated to 40°C

to 45°C into the peritoneal cavity. Heat is conducted directly to the intraperitoneal structures. Heat is also conducted through the posterior parietal peritoneum to the solid viscera and through the hemidiaphragms to the heart and lungs. But adhesions from previous abdominal surgery not only minimize heat exchange, they can also increase the complication rate.

A double catheter system with suction at the outflow will increase the rate of rewarming. The standard clinically attainable exchange rate is 6 L/h.[82,110] Two liters of isotonic dialysate are infused, retained for 20 to 30 minutes, and then aspirated. Rewarming rates average one to three Centigrade degrees per hour, depending on flow rates and dwell times.[111] Peritoneal dialysis will exacerbate preexistent hypokalemia; therefore, potassium supplementation of the dialysate is often essential.[112] There are some advantages unique to this rewarming technique:

- Detoxification may be an incidental benefit of peritoneal dialysis by accelerating the removal of myoglobin that was caused by exertion-induced rhabdomyolysis.
- The direct hepatic rewarming stimulates the detoxifying and conversion enzymes.
- An occult hemoperitoneum may also be discovered in the traumatized hypothermic victim if ultrasonography or computed tomography scanning is not available.

Peritoneal lavage rewarming should not be routinely selected for stable patients. Accelerating the rate of rewarming with an invasive technique is not warranted in a stable patient who is not severely hypothermic. Medical officers should consider selecting peritoneal lavage in cases of severe hypothermia; its use should be combined with all available rewarming techniques for patients without spontaneous perfusion.

Heated Irrigation

Heat transfer from irrigation fluids is usually very limited because the surface area available for heat exchange is minimal. In addition, direct upper or lower gastrointestinal irrigation is less desirable than heat transfer via warm-fluid-filled gastric or colonic balloons because of the potential for fluid and electrolyte fluxes.[113–115] Patients who require heated gastrointestinal irrigation also require tracheal intubation prior to gastric lavage to prevent aspiration. If more than 200- to 300-mL aliquots are infused, some fluid will enter the duodenum. Always log the input and output while calculating the volume resuscitation. The usual rewarming rate achieved with gastric, bladder, or colon lavage is approximately one to two Centigrade degrees per hour.[1]

The clinical experience is growing with closed thoracic lavage in accidental hypothermia. Two large-bore thoracostomy tubes are inserted into one or both hemithoraces. One is placed anteriorly in the second or third intercostal space at the midclavicular line. The other is placed in the posterior axillary line at the fifth to sixth intercostal space. Then normal saline is heated to 40°C to 42°C, infused and drained through a nonrecycled sterile system,[116–118] and collected for recycling of the perfusate. This technique is facilitated if a high-flow countercurrent fluid infuser is available that heats the crystalloid infusion fluid to 40°C. Heated normal saline is delivered from 1-L or, preferably, 3-L bags. A sterilized, plastic, graduated, two-way connector is used to adapt the tubing from the warmer to any size chest tube. The effluent is collected in a thoracostomy drainage set.

The efficiency of heat transfer varies with the flow rate and the dwell times. Pleural adhesions will prevent acceptable infusion rates. Adequate drainage must be assured to prevent the development of a tension hydrothorax and mediastinal shift. The insertion of a left-sided thoracostomy tube in a patient with spontaneous mechanical cardiac activity risks the development of iatrogenic VF because of the mechanical stimulation of the heart. If the perfusing patient requires a thoracostomy tube for a traumatic hemothorax or pneumothorax, then the placement of a second tube, especially on the right side, is a good option. The clinically attainable infusion rates average 200 to 400 mL/min. This results in a rate of rewarming that at least equals that of peritoneal dialysis, with the heat transfer preferentially occurring through the mediastinal pleura.

Lavage should be reserved for severely hypothermic patients who do not respond to standard rewarming techniques. In potentially salvageable patients with cardiac arrest, thoracic lavage should be combined with all the other available rewarming modalities. When the patient is successfully rewarmed, the upper chest tube is removed and the lower one is left in place to facilitate residual drainage.[119–121]

Mediastinal irrigation and direct myocardial lavage are generally considered alternatives only in patients without spontaneous perfusion,[71] but the procedures require a thoracotomy incision, which is most commonly made in the anterolateral location. It is not necessary to open the pericardium unless there is a pericardial effusion or cardiac tamponade. The heart should be bathed for several

minutes in 1 to 2 L of an isotonic solution heated to 40°C, the solution should be removed via suction, and then the process should be repeated. Internal defibrillation should be attempted at intervals of one to two Centigrade degrees after the myocardial temperature exceeds 26°C to 28°C.[68,121] A median sternotomy does allow ventricular decompression in addition to direct defibrillation. Nevertheless, open cardiac massage of a cold, rigid, and contracted heart may not generate blood flow[122,123]; interestingly, in a similar situation, closed-chest CPR will generate blood flow. Therefore, cardiopulmonary bypass capability is essential.

Diathermy

Diathermy involves the transmission of heat by the conversion of energy.[124,125] Large amounts of heat can be delivered to deep tissues with ultrasonic and low-frequency microwave radiation. Diathermy is contraindicated if the patient has frostbite, burns, significant edema, a pacemaker, or any type of metallic implant. In the absence of circulation, diathermy may cause burning of the core tissues.

Dosimetry guidelines and ideal application sites are being developed. Truncal application is being investigated in animals and humans. This may prove to be a valuable modality in the field. Potential problems include the power supply and the potential for navigational and electronic interference.[126–129]

Extracorporeal Blood Rewarming

The four common methods used to directly rewarm blood are (1) cardiopulmonary bypass, (2) arteriovenous rewarming, (3) venovenous rewarming, and (4) hemodialysis. However, rapid acceleration of the rate of rewarming per se does not necessarily improve survival rates. Some of the complications of rapid rewarming in severe hypothermia include disseminated intravascular coagulation, pulmonary edema, hemolysis, and acute tubular necrosis. Extracorporeal blood rewarming is unlikely ever to succeed if core temperature is below 5°C; medical officers should abandon any of these attempts if frozen or clotted intravascular contents are identified.

Cardiopulmonary Bypass. The major advantage of cardiopulmonary bypass (CPB) in perfusing patients is that flow can be preserved if mechanical cardiac activity is lost during rewarming.[130–137] The appropriate candidates for CPB are patients who do not respond to less-invasive rewarming techniques, those with completely frozen extremities, and those with rhabdomyolysis that is accompanied by electrolyte disturbances. The simplest circuit includes percutaneous or cutdown cannulation of the femoral artery and vein in adults, or the iliac vessels in small children. During the first hour of CPB, an enhanced physiological fibrinolysis occurs. The development of heparin-coated perfusion equipment is decreasing the need for heparinization in CPB, which makes its use as a rewarming technique safer in trauma patients (in whom heparin is otherwise contraindicated).[138–141]

Heated and oxygenated blood is returned to the patient. This results in femoral flow rates of around 2 to 3 L/min of oxygenated blood at 38°C to 40°C. This procedure can elevate the core temperature one to two Centigrade degrees every 3 to 5 minutes. In a major review, the mean CPB temperature increase was found to be 9.5°C/h.[132] Flow rates of up to 6 L/min can be achieved with commercially available systems. During bypass the physician should expect vascular endothelial leaks that might necessitate massive volume replacement.[142]

The optimal bypass rewarming rate and temperature gradient are not yet resolved. Most investigators prefer gradients of 5 to 10 Centigrade degrees between the temperature of the perfusate and the core temperature.[143,144] The CPB technique can result in complications including vascular damage, hemolysis, air embolism, and disseminated intravascular coagulation. It may be necessary to aggressively augment the intravascular volume to maintain adequate flow rates.

Arteriovenous and Venovenous Rewarming. Continuous arteriovenous rewarming is another option when the patient's spontaneous blood pressure is at least 60 mm Hg. Femoral arterial and venous catheters are percutaneously inserted and connected to the inflow and outflow ports of a countercurrent fluid warmer. This eliminates the need for a pump, perfusionist, or systemic anticoagulation.[144]

Another variation of the extracorporeal recirculation approach in perfusing patients is venovenous rewarming. Blood is removed through a central venous catheter, heated to 40°C, and returned either through a second central, or a peripheral venous, catheter. This technique commonly achieves flow rates of 150 to 400 mL/min, although it does not provide oxygenation or full circulatory support.[145–148]

Hemodialysis. Hemodialysis is another practical rewarming technique when using two-way flow catheters that allow percutaneous cannulation of a single vessel.[141] After central venous cannulation, exchange cycle volumes of 200 to 500 mL/min are possible.

FIELD STABILIZATION AND MANAGEMENT

Field treatment of hypothermia is of "the art of the possible." The prime directive is to prevent further heat loss. Field conditions commonly mandate that the hypothermic victim first be rescued, then examined, and finally insulated prior to transport (Exhibit 16-5).[149,150] Rescuers or field medical personnel should document the duration of exposure, circumstances of discovery, associated injuries, frostbite, and predisposing conditions. Initial management should emphasize both prevention of further heat loss and rewarming the core before the shell. Rigid rewarming protocols are ill-advised, given the variability of the multifactorial physiological, environmental, and tactical conditions. The history of exposure is often critical. Chronic exposure presents a far greater challenge to safe rewarming than does acute exposure. If the service member is unresponsive and not shivering, medical officers should presume that the hypothermia is severe and chronic. At temperatures below 32°C, anticipate an irritable myocardium, greater dehydration, and a larger temperature gradient between the peripheral shell and the core.[151]

Field Stabilization

It is generally impractical to accurately measure the core temperature in the field. Always assume that the patient who is not fully alert and oriented to person, place, time, and (most importantly) situation may be severely hypothermic. Estimates of severity based simply on the level of consciousness, behavior, or the presence of shivering are often misleading. Conscious patients may be rewarmed in the field if evacuation will be delayed and the appropriate rewarming equipment is available. It may be necessary to "go to ground" during certain military maneuvers until conditions are suitable for gentle evacuation.

Do not allow the casualty to initiate significant muscular exertion. This can exacerbate core temperature afterdrop. Many alert but severely hypothermic individuals have collapsed after rescue while ambulating, exerting, or climbing into ships.[15]

Prolonged field treatment should be avoided whenever possible. The service member should be thermally stabilized. Remove any wet clothing and replace with dry, and begin passive external rewarming. This will minimize radiant, conductive, convective, and evaporative heat losses. The patient can be insulated with dry clothing, blankets, insulated pads, bubble wrap, or newspaper. Windproof and waterproof casualty evacuation bags are available in various designs; these insulated bags have special openings and zippers that allow access to the patient during transport. Although aluminum-coated foils are reported to be more efficient than woolen rescue blankets,[72,152,153] the woolen blankets should be used in addition to polyethylene bags or aluminum-coated foils.

EXHIBIT 16-5

PREPARING HYPOTHERMIC CASUALTIES FOR TRANSPORT

1. The casualty must be dry. Gently remove or cut off wet clothing and replace it with a dry uniform or insulation system. Keep the casualty horizontal, and do not allow exertion or massage of the extremities.
2. Stabilize injuries (eg, the spine; place fractures in the correct anatomical position). Open wounds should be covered before packaging.
3. Initiate intravenous infusions if feasible; bags can be placed under the casualty's buttocks or in a compressor system. Administer a fluid challenge.
4. Active rewarming should be limited to heated inhalation and truncal heat. Insulate hot water bottles in stockings or mittens and then place them in the casualty's axillae and groin.
5. The casualty should be wrapped. The wrap starts with a large plastic sheet, on which is placed an insulated sleeping pad. A layer of blankets, a sleeping bag, or bubble wrap insulating material is laid over the sleeping bag; the casualty is placed on the insulation; the heating bottles are put in place along with intravenous infusions, and the entire package is wrapped, layer over layer. The plastic is the final closure. The casualty's face should be partially covered, but a tunnel should be created to permit breathing and allow access for monitoring.

If extrication will be prolonged, it may be necessary to hydrate the victim who appears to be only mildly hypothermic. The cooling process induces a significant diuresis. It will be safer to ambulate the mildly hypothermic casualty to safety after he or she has ingested adequate quantities of warmed oral fluids. Whenever possible, attempt to immobilize the severely hypothermic casualty in a horizontal position. This will minimize the orthostatic hypertension induced by an autonomic dysfunction.

In addition to an adequate intravascular volume, the most important factor in maintaining perfusion is the degree of oxygenation maintained during severe hypothermia. Assisted mouth-to-mouth or mouth-to-nose ventilation may be difficult because of the chest stiffness and decreased pulmonary compliance (Exhibit 16-6).

Resuscitation with crystalloid volume replacement fluid is also critical. It may be difficult to locate a peripheral vessel in the vasoconstricted dehydrated patient. Focal application of heat to the antecubital fossa may be helpful. Intraosseous infusions have not been studied under hypothermic conditions, but intuitively, they are an option.

Under ideal circumstances, heated (37°C–41°C) 5% dextrose should be infused in normal saline solution.[154] If unavailable, any other crystalloid will be satisfactory. The initial fluid challenge should be at least 500 mL to 1 L. The attempts to prevent the intravenous fluids from freezing often requires improvisation. Plastic containers of intravenous fluid can be placed under the casualty's shoulders, buttocks, or back to add infusion pressure and warmth. Other options involve taping heat-producing packets to the intravenous fluid bags. Consider the use of any of the various chemical packets and phase-change crystal units that produce heat for several hours after activation. Under extreme conditions, pneumatic bag compressors are less practical than spring steel compression devices.[15]

Heated inhalational therapy is a safe active rewarming adjunct for the field treatment of profound hypothermia.[65] In addition to providing heat, inhalational therapy minimizes the respiratory heat loss that varies with the ambient air temperature, humidity, and the patient's RMV. Inhalation of air heated and humidified with any available portable device is desirable. The net heat yield from warmed air delivered through a mask can represent 10% to 30% of the hypothermic casualty's heat production.[16]

Surface rewarming may be the only practical field option, even though this can suppress shivering and impede the overall rate of core rewarming. Other rewarming options include placing warmed objects on the patient, radiant heat, or body-to-body contact. Burns are a hazard if the objects remain in contact with vasoconstricted skin for an extended period. Ideally, active external rewarming should

EXHIBIT 16-6

OXYGENATION CONSIDERATIONS DURING HYPOTHERMIA

Detrimental Factors

1. Oxygen consumption increases with rise in temperature (use caution if rapidly rewarming, as shivering also increases demand).
2. Decreased temperature shifts oxyhemoglobin dissociation curve to the left.
3. Ventilation–perfusion mismatch; atelectasis; decreased respiratory minute volume; bronchorrhea; decreased protective airway reflexes.
4. Decreased tissue perfusion from vasoconstriction; increased viscosity.
5. Functional hemoglobin concept; capability of hemoglobin to unload oxygen is lowered.
6. Decreased thoracic elasticity and pulmonary compliance.

Protective Factors

1. Reduction of oxygen consumption: 50% at 28°C, 75% at 22°C, 92% at 10°C.
2. Increased oxygen solubility in plasma.
3. Decreased pH and increased $Paco_2$; shift in oxyhemoglobin dissociation curve to right.

Adapted with permission from Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach P, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St Louis, Mo: Mosby; 1995: 57.

be limited to the trunk.[15] This allows the extremities to remain vasoconstricted. In severe hypothermia, extinguishing peripheral vasoconstriction may result in the sudden return of cold, acidotic, hyperkalemic blood to the heart. Upper truncal contact is preferable to and less hazardous than full-body contact rewarming.[65] Immersion rewarming is logistically impossible in the field, and is rarely desirable unless the casualty became cold acutely.

In the field, life support for hypothermia is far more challenging than life support under normothermic conditions. The breathing casualty may appear apneic because of the significantly depressed RMV. Overzealous ventilatory assistance can induce hypocapnic ventricular irritability and fibrillation. The indications for endotracheal intubation of hypothermic casualties in the cold field are identical to those under normothermic conditions. The heart will be protected if the patient is handled gently and adequately preoxygenated. Standard plastic endotracheal tubes require care during frigid conditions. Cold ambient air in the cuff will expand when heated and may cause the tube to kink. Similarly, the tubing of the cuff port may break easily during extremely cold conditions.[155]

It is easy to misdiagnose cardiac arrest. The palpation of peripheral pulses is very difficult in the patient with vasoconstriction and bradycardia. In severe hypothermia there is significant depression of the RMV. The patient may appear apneic. Sufficient time (at least 1 min) spent palpating and auscultating for any spontaneous pulse is essential. The extreme bradydysrhythmias may provide sufficient cardiac output to meet the depressed metabolic demands. The cold myocardium is extremely irritable, and iatrogenic ventricular fibrillation is easily precipitated with closed chest compressions. Individuals who are cold, stiff, cyanotic, with fixed pupils and inaudible heart tones, and without visible thoracic excursions have been successfully resuscitated. Resuscitation measures should not be discontinued unless attempts fail after the patient has been rewarmed to 35°C.

If a cardiac monitor and defibrillator are available in the field, the QRS complex should be maximally amplified. The adhesion of standard monitor leads to cold skin is poor; benzoin or needle electrodes improve the quality of the reading. Although most equipment has not been tested for operation below 15.5°C, defibrillation may be attempted once with 2 J/kg when there is no evidence of spontaneous cardiac perfusion.[156] A successful reestablishment of electromechanical activity has been reported at a temperature as low as 20°C.[157] If the defibrillation attempt is unsuccessful, active rewarming with available equipment should be initiated while CPR is continued en route to the healthcare facility. Defibrillation attempts are usually unsuccessful until the core temperature is well above 28°C to 30°C.[158,159] If the patient is perfusing, then gentle transport is critical. Ideally, vehicles should be driven slowly and gurneys should be carried, not rolled. In some terrain, the ideal transport option is aeromedical evacuation, which minimizes mechanical jostling of the patient.[83-85]

Practical Management Advice

At all times it is important to ensure the safety of the rescuer as well as the casualty. This is particularly important in a hostile environment (eg, in water or on a hillside). As well as being aware of the dangers of falling rocks, unstable snow, or unsafe ice, rescuers must guard against becoming hypothermic themselves, either through exhaustion or as a result of donating clothing to the casualty.

If possible, a casualty should be rescued horizontally, especially from water. It is often more important, however, to get the casualty to safety quickly rather than delay to enable horizontal rescue.[19] When it is safe to do so, the casualty should be lain flat, given essential first aid for any injury, and then resuscitation can be undertaken. During subsequent transport, the casualty should be kept lying flat or slightly head down to avoid orthostatic hypertension.[24,26,76] Any unnecessary movement of the unconscious or semiconscious casualty should be avoided, as movement may precipitate VF. However, these considerations may be unrealistic during the practical realities of a difficult rescue.

The casualty's wet clothing should be removed, but only when warm shelter has been reached. If the casualty is unconscious, the wet clothes must be cut off so as to avoid unnecessary movement. Close observation must be maintained. As soon as possible, get help and transport the casualty to the hospital.

The choice of treatment in the field is governed by many factors including distance, local risk including weather, number and experience of rescuers and their physical condition, and the availability of equipment.[153] The only methods of rewarming that can be considered practical in the field are spontaneous rewarming, airway warming, and surface heating.[16,76]

Advanced Life Support

Blood flow during CPR in hypothermia differs from that during normothermia. During hypothermic conditions, some flow results from the phasic alterations in the intrathoracic pressure, and not just from direct cardiac compressions.[122,123,160] Chest wall elasticity and pulmonary compliance are decreased when cold. Therefore, more force is needed to depress the chest wall sufficiently to generate the necessary intrathoracic pressure gradients. In severe hypothermia, the heart functions as a passive conduit. The phasic alterations in the intrathoracic pressure (which are generated by the chest compressions) are applied equally to all of the cardiac chambers. Because the mitral valve remains open during the compressions, blood continuously circulates through the left side of the heart.

The clinical evidence consists of the numerous neurologically intact survivors who received hours of prolonged closed chest compressions.[123,161–164] Some, who were subsequently placed on cardiopulmonary bypass equipment, were found to have hearts "as hard as stone,"[123(p492)] (ie, the heart muscle was not frozen but contracted).

The decision to initiate CPR under combat circumstances is multifactorial. Because intermittent blood flow may provide adequate support during evacuation, do not withhold CPR only because continuous compressions cannot be assured.[165–169] Dilated pupils, apparent dependent lividity, and tissue decomposition are not reliable criteria for withholding CPR. There are widely endorsed civilian recommendations[1,15] to initiate CPR in accidental hypothermia that are, for the most part, also applicable to military casualties (see Exhibit 11-5 in Chapter 11, Human Physiological Responses to Cold Stress and Hypothermia).

The literature is replete with reports of neurologically intact survivors following prolonged CPR. Examples of casualties who ultimately survived, and their treatment, include the following:

1. A man, very cold and with no signs of life, was successfully resuscitated on the seafront using external cardiac compression and expired air resuscitation after he had been hauled out of a cold winter sea by the rope that had formed a loop round his neck.[170]
2. Standard CPR has been continued for 2.5 hours during helicopter rescue[123]; for 4 hours during transport in a snow vehicle ambulance; for 4.5 hours during helicopter transport and rewarming (but only when cardiac arrest occurred at a rectal temperature of 23°C with the rhythm varying between asystole and VF)[171]; and for 6.5 hours including air ambulance transport when asystole occurred at 23.2°C.[161]

IN-HOSPITAL MANAGEMENT

In mild hypothermia, the oral temperature obtained with a low-reading thermometer may be unreliable if the patient is uncooperative, tachypneic, or if the ambient temperature is low. Standard thermometers only measure down to 35°C. In more severe cases, medical officers should confirm and monitor hypothermia with continuous core temperate evaluation.

Most commonly, continuously monitored core temperature is measured in the rectum. The rectal temperature can lag behind fluctuations of the core temperature and is markedly influenced by the lower extremity temperatures and placement of the probe. Insertion of the probe should be to a depth of 15 cm; if the temperature seems inappropriate or does not change, the probe has probably been placed in cold feces.

The tympanic temperature correlates most directly with the actual core temperature because it should be closest to the hypothalamic temperature.[172] The reliability of commercially available infrared thermography devices remains troublesome. If the patient is tracheally intubated, an esophageal probe is an invaluable ancillary option to allow accurate measurement of rewarming trends; naturally, this reading may be factitiously elevated during heated inhalation.[173]

The vital signs should be recorded on a flow sheet. A doppler measuring device may be necessary to establish the presence of a spontaneous pulse or blood pressure.[92]

The accuracy of pulse oximetry during conditions of poor perfusion and hypothermia is uncertain.[174,175] End-tidal carbon dioxide measurements also accurately assess tissue perfusion and tracheal tube placement only under normothermic conditions. The devices that are commercially available are not functional in the presence of the humidified air that is essential for airway warming.

Initial Stabilization

The indications for endotracheal intubation dur-

ing hypothermia are identical to those under normothermic conditions. Endotracheal intubation is necessary unless the patient is alert and possesses intact protective airway reflexes.[1,4,176,177] Cold depression of ciliary activity causes accumulation of secretions, which produces frothy sputum and chest congestion.

Severely hypothermic patients are frequently trismic. As a result, blind nasotracheal intubation is often the only practical nonsurgical option. Significant epistaxis is a concern if the patient is coagulopathic. A topical vasoconstrictor may be sprayed into the nares, and an endotracheal tube 0.5 mm in size (smaller than usual) may be inserted. In adults, a 7.5-mm tube is usually ideal; a 7.0-mm tube is acceptable but more prone to thrombotic or mechanical occlusion or collapse. No induced dysrhythmias were observed in a multicenter survey in which endotracheal intubation was performed on 117 patients by multiple operators in various settings.[3] Some common factors precipitating dysrhythmias include failure to preoxygenate, mechanical jostling, acid-base changes, and electrolyte fluctuations.

Nasogastric intubation (following endotracheal intubation) is also necessary in moderate and severe hypothermia. Decreased gastric motility and gastric dilation occur frequently. Physical examination of the abdomen is unreliable because the cold can induce rectus muscle rigidity. A large percentage of patients with moderate and severe hypothermia have decreased or absent bowel sounds. Because the physical examination of the abdomen is unreliable, medical officers should check for associated ileus or pancreatitis, and for occult trauma.

In patients with moderate and severe hypothermia, indwelling bladder catheters with urometers are essential to monitor urinary output. Peripheral or central intravenous catheters should be inserted as necessary. The insertion of a central venous pressure catheter tip into the right atrium may precipitate cardiac dysrhythmias. Arterial catheters for continuous monitoring of the intraarterial blood pressure may be helpful in selected profoundly hypothermic patients. The placement of a pulmonary artery catheter, in contrast, is far more hazardous. Although the catheter is potentially useful, perforation of the cold, stiff, pulmonary artery is a concern.[178]

The immediate laboratory evaluations should include a rapid bedside glucose determination; blood sugar level; arterial blood gases uncorrected for temperature; complete blood count; electrolyte panel; serum calcium, serum magnesium, and serum amylase/lipase levels; prothrombin time (PT) and partial thromboplastin time (PTT); platelet count; and fibrinogen level. The baseline serum blood urea nitrogen (BUN) and creatinine are indicated because renal failure may occur after rewarming in patients with chronic hypothermia.

Because the history may be misleading, the radiological evaluation of poorly responsive hypothermic patients must include a screen for occult trauma. Studies should include cervical and possibly thoracic or lumbar spine roentgenography. Other studies may be indicated based on the mechanism of injury and exposure. Chest radiographs help screen for pneumonia and predict which patients may be developing pulmonary edema during rewarming. Because the abdominal examination is often unreliable, roentgenograms can detect pneumoperitoneum or hemoperitoneum that is clinically silent and totally unsuspected.

Volume Resuscitation

The circumstances will determine the need for fluid administration. Particularly in combat, the normal physiological cues for dehydration, such as thirst, are not active. Patients with moderate or severe hypothermia are usually significantly dehydrated; rapid volume expansion is critical. Patients with moderate or severe hypothermia should initially receive a fluid challenge of 500 mL to 1 L of heated 5% dextrose in normal saline. Once the laboratory values are available, supplemental potassium may be required. Lactated Ringer's solution should be avoided because the cold liver inefficiently metabolizes lactate.[16] Persistent cardiovascular instability in a young, healthy patient usually reflects insufficient volume repletion.[179,180]

Whenever feasible, intravenous fluids should be heated to 40°C to 42°C. The amount of heat provided will become significant with large-volume resuscitations.[181,182] One heating option is to microwave intravenous fluids in plastic containers.[183,184] A 1-L bag of crystalloid requires an average of 2 minutes on high power. To avoid hot spots, the fluid should be shaken prior to administration. Blood-warming packs can shorten the life of red cells, and local microwave overheating will cause hemolysis. A variety of commercial blood-warming devices are available.[185–187]

Hemoconcentration secondary to decreased plasma volume, fluid shifts, and increased vascular permeability is usually present. Hemodilution can occur and is associated with hemorrhage and parenteral crystalloid administration. Transfu-

sions of washed, packed red blood cells are occasionally necessary for hemorrhaging patients. Because it is easy to underestimate the severity of the anemia, it is important to remember to correct the hematocrit for the temperature. The viscosity of blood increases 2% per Centigrade degree drop in temperature.

The safety of pneumatic antishock garments in hypothermia is unknown. Because the peripheral vasculature is already maximally vasoconstricted, the provision of additional peripheral vascular resistance may not be possible.[188] Potential clinical concerns would include the development of an extremity compartment syndrome and rhabdomyolysis.

Resuscitation Pharmacology

The efficacy of most medications is temperature dependent. The protein binding of the drugs increases during hypothermia, whereas liver metabolism is decreased. The enterohepatic circulation and renal excretion are also altered. To achieve a therapeutic response in a hypothermic patient, dosages would have to be administered that would be toxic or lethal after rewarming.

Cardiovascular System

In general, the pharmacological manipulation of the pulse or blood pressure should be avoided. Vasoactive agents may be dysrhythmogenic and have a minimal effect on the maximally constricted peripheral vasculature.[15,176] Vasodilators extinguish peripheral vasoconstriction and can precipitate severe core temperature afterdrop or a severe drop in blood pressure.

With mild hypothermia, inotropes are usually not necessary to support the blood pressure. The autonomic nervous system appears to switch off at around 29°C, however, which suggests that some catecholamine support might be useful below that temperature.[189–193] The medical officer should consider administering low-dose (2-5 μg/kg/min) dopamine infusions in patients who are disproportionately hypotensive and who do not respond to crystalloid infusion and rewarming. If the patient also has severe frostbite, catecholamines will jeopardize the affected tissues in the extremities. Catecholamines also can exacerbate preexistent occult hypokalemia.

Virtually all atrial dysrhythmias are common below 32°C, and there should be a slow ventricular response. Atrial fibrillation is common and should be considered innocent, because it usually converts spontaneously during rewarming. Digitalization or calcium channel blockade is not warranted.[194–196] When the rhythm converts back to sinus rhythm during rewarming, mesenteric embolization is a hazard during this shock state.

Preexisting chronic ventricular ectopy is often suppressed by the cold; it reemerges during rewarming. As a result, without an accurate past cardiac history, medical officers must weigh the advantages of prophylaxis. It is generally wise to ignore transient ventricular arrhythmias that appear during rewarming. The pharmacological options are limited. The prophylactic value of bretylium or lidocaine during hypothermia has not been evaluated.

The ideal treatment of ventricular dysrhythmias is also not resolved. Bretylium tosylate, however, has reportedly been extremely effective in several studies.[197–202] Some clinical case studies report "chemical" ventricular defibrillation with bretylium in severe hypothermia.[197–199] Bretylium tosylate appears to be the drug of choice for both the prophylaxis and treatment of hypothermic VF.[200] This class III agent has both antiarrhythmic and antifibrillatory activity. It increases the VF threshold, the action potential duration, and the effective refractory period. Because the optimal dosage and ideal infusion rates are unknown,[201,202] standard normothermic doses should be administered.

The other pharmacological options are very limited. Lidocaine does not seem effective in facilitating hypothermic ventricular defibrillation. Procainamide is reported to increase the incidence of VF, whereas quinidine and magnesium sulfate may be beneficial.

Transvenous intracardiac pacing is used in the management of bradydysrhythmias; the technique is extremely hazardous in patients with systemic hypothermia, however, because of its propensity to cause potentially fatal dysrythmias such as VF. External noninvasive pacing via large, low-resistance electrodes has been a successful alternative to emergency transvenous pacing and is preferable in hypothermia.[203]

Endocrine System

Acute cold stress initially stimulates cortisol secretion. The patient may already have a very high cortisol level secondary to the underlying stress. In clinical series, serum cortisol levels are commonly elevated.[4,176] The percentage of cortisol bound to protein is increased with hypothermia, and therefore the active free fraction is decreased. Cortisol

utilization is also decreased during hypothermia.[204]

Considering these factors, the routine use of steroids in accidental hypothermia cannot be recommended. Steroids should be withheld unless a definite suspicion of hypoadrenocorticism exists. If the patient fails to rewarm, medical officers should recheck the history for evidence of adrenocortical insufficiency or steroid dependence. At that juncture, the intravenous administration of 30 mg/kg methylprednisolone sodium succinate or 250 mg hydrocortisone should be considered. In addition, some argue that steroids are worth considering in extremis, in view of reports from the field that large intravenous doses, given as a last resort, have had unexpectedly beneficial effects.[47]

Empirical treatment with thyroxine (T_4) should also be reserved for patients believed to be myxedematous. Myxedema coma can be precipitated by trauma, stress, or infection. Thyroid hormone replacement is advisable if there is a history of hypothyroidism, a neck scar from a subtotal thyroidectomy is apparent, or a failure to rewarm with appropriate therapy.[205] After thyroid function studies are obtained, 250 to 500 μg of levothyroxine should be administered intravenously over several minutes. Daily injections of 100 μg will be necessary for 5 to 7 days. One hundred to 200 mg of hydrocortisone should be added to the first several liters of crystalloid. There is no role for 3,5,3'-triiodothyronine (T_3) in acute replacement therapy because its rapid onset of action induces cardiovascular instability.[206]

Laboratory Evaluations

Hypothermic patients being managed in a hospital will benefit from many commonly available laboratory tests such as those that determine blood pH and coagulation status.

Acid-Base Balance

It is not possible to reliably predict the acid–base status in accidental hypothermia.[4] There is less room for error while attempting to maintain ideal acid–base balance in hypothermia than at a normal temperature. Cold blood buffers poorly. In normothermia, when the partial pressure of carbon dioxide (PCO_2) increases 10 mm Hg, pH decreases 0.08 units. At 28°C, the decrease in pH will double. Overzealous ventilation will cause dramatic swings in the pH and can increase ventricular irritability. The medical officer must be aware that the correction of arterial blood gas parameters is pointless.[207,208] Arterial blood samples are always warmed to 37°C in the pH meter before electrode measurements are obtained.

The best intracellular pH reference is electrochemical neutrality, when pH = pOH, or $[H^+] = [OH^-]$. Because the neutral point of water at 37°C is pH 6.8, this normal 0.6-unit pH offset in body fluids should be maintained at all temperatures.[209] Just as the neutral pH rises with cooling, so should actual blood pH. Intracellular electrochemical neutrality ensures optimal enzymatic function at all temperatures.[210,211] As a result, relative alkalinity of tissues makes physiological sense.[212–214]

To accurately interpret the uncorrected arterial blood gases, simply compare values with the normal values at 37°C. If the uncorrected pH is 7.4 and the PCO_2 is 40 mm Hg, then alveolar ventilation and acid–base balance are normal at any temperature during rewarming.

Hematological Evaluation

The severity of any blood loss is easy to underestimate. The medical officer should anticipate that prior to rehydration there will be a deceptively high hematocrit secondary to the decreased plasma volume. The hematocrit directly increases 2% for every temperature decrease of one Centigrade degree. Recurrent evaluation of serum electrolytes during rewarming is essential. There are no safe predictors of their values or trends.[1,215] The severity and chronicity of hypothermia and the method of rewarming will alter the serum electrolyte values.[216]

The plasma potassium level is independent of the primary hypothermic process. An important caveat is that hypothermia enhances cardiac toxicity and obscures premonitory electrocardiographic changes. Although hyperkalemia is often associated with several associated conditions, including metabolic acidosis, rhabdomyolysis, and renal failure, hypokalemia is most common with chronically induced hypothermia.[1,217] Hypokalemia results from potassium's entering muscle and not from a kaliuresis.[218,219] If the potassium level is lower than 3 mEq/L, then addition of 20 to 30 milliequivalents of potassium per liter of crystalloid may be necessary for treatment of gastrointestinal ileus or congestive heart failure during rewarming.

The BUN and creatinine levels will be elevated with any preexisting renal disease or decreased clearance. Because of hypothermic fluid shifts, the hematocrit and the BUN levels are poor indicators of the actual fluid status.

The blood sugar level also provides a subtle clue to the duration of hypothermia. Acute hypothermia

initially elevates the blood sugar via catecholamine-induced glycogenolysis. In contrast, chronic hypothermia depletes glycogen. Many of the symptoms of hypoglycemia may be masked by hypothermia. A cold-induced renal glycosuria does not imply hyperglycemia. Correction of hypoglycemia and the resultant central neuroglycopenia will only correct the level of consciousness to that of the corresponding level of hypothermia.

Severe hypothermia predictably causes serum enzyme elevation because of the ultrastructural cellular damage.[220] Rhabdomyolysis is commonly associated with cold exposure.

The extent of hyperamylasemia seen with accidental hypothermia can correlate with mortality. Ischemic pancreatitis may result from the microcirculatory shock of hypothermia that activates proteolytic enzymes.[221]

Hypothermic Coagulation

Hypothermia produces coagulopathies via three major mechanisms[222,223]: (1) the coagulation cascade of enzymatic reactions is impaired, (2) platelets are sequestered and function poorly, and (3) plasma fibrinolytic activity is enhanced.

The medical officer will frequently observe a major disparity between the in vivo clinically evident coagulopathy and the deceptively "normal" PT or PTT reported by the laboratory[224]. Remember that the kinetic tests of coagulation are performed in the laboratory at 37°C. When the enzymatic coagulation factors are rewarmed in the machine, they are activated, and the reported in vitro PT and PTT will be "normal." The only effective treatment is rewarming, not the administration of clotting factors.[225]

A physiological increase in coagulation occurs with hypothermia, and a disseminated intravascular coagulation type of syndrome is reported.[226] Hypothermic patients also develop coagulopathies because the enzymatic nature of the activated clotting factors are depressed by the cold.[223,227] The clotting prolongation is proportional to the number of steps in the coagulation cascade. Clinically significant coagulopathies occur and are commonly associated with trauma.[228,229]

Septicemia

The classic signs of infection are invalid during accidental hypothermia.[230] Fever will be absent and rigors will resemble shivering. The history, physical, and initial laboratory data are often unreliable.[15,157,231] Septicemia complicates recovery from accidental hypothermia; broad culturing is generally indicated.

Host defenses are compromised during hypothermia, and serious bacterial infections develop easily. Gram-negative septicemia may cause secondary hypothermia, which can be mistaken for primary accidental hypothermia; and coexistent infections from Gram-positive cocci, Enterobacteriacae, and oral anerobes are common in accidental hypothermia.[173,232]

Routine antibiotic prophylaxis in hypothermic adults, unlike that in the elderly and in children, does not appear warranted. Medical officers should use antibiotics if the clinical picture is consistent with septic shock, if there is failure to rewarm, or if the patient has aspirated. Cellulitis, myositis, bacteriuria, or infiltrates present on chest roentgenograms all warrant immediate antimicrobial therapy. Broad-spectrum coverage is ideal, and in serious infections, the combination of an aminoglycoside with a β-lactam antibiotic is indicated.

MEDICAL OUTCOME AND DISPOSITION OF CASUALTIES

Because of the variability of human physiological responses in general, and to hypothermia in particular, outcome is difficult to predict. In the past, the treatment dictum was that "no one is dead until they are warm and dead," and this dictum remains important in the management of hypothermia. Some casualties are, however, cold and dead, and it would be particularly useful if they could be safely identified in the field.[233,234]

Trauma affects survival unpredictably. Outcome prediction based on the Glasgow Coma Scale is unreliable.[52] Although there are no validated prognostic neurological scales for use during hypothermia, the Glasgow Coma Scale score should still be recorded, because the trend may be useful. A hypothermia outcome score developed from a large database might enable multiple observers at differing sites to assess treatment modalities and out-come predictors.[235] Some of the significant predictors of outcome include prehospital cardiac arrest, a low or absent presenting blood pressure, elevated BUN, and the need for either endotracheal or nasogastric intubation in the medical treatment facility. Negative survival factors in some studies include asphyxia, asystole, a slow rate of cooling, and the development of pulmonary edema or adult respiratory distress syndrome.[236] The search continues for a valid triage marker of death.[237–241] Grave prognostic indica-

tors include evidence of intravascular thrombosis (fibrinogen < 50 mg/dL), cell lysis (hyperkalemia > 10 mEq/L), and ammonia levels higher than 250 μmol/L.[225]

Previously healthy patients who have mild primary accidental hypothermia (core temperatures of 35°C–32.2°C) will usually rewarm easily. They can be medically released provided a suitably warm environment is available. Most patients with more-severe hypothermia (core temperatures < 32.2°C) and those with secondary hypothermia require prolonged medical attention. Medical officers should consider cardiac monitoring in patients with persistent metabolic abnormalities. This is essential for those patients displaying cardiovascular instability or an inadequate rate of rewarming.[240]

SUMMARY

Hypothermia may masquerade as a variety of conditions, including death, in a variety of situations and seasons. The initial findings are often quite subtle, and may simply consist of personality changes or impaired judgment.

Military service members with mild hypothermia, in the absence of underlying disease, do well with any of the treatment alternatives. Field treatment of moderate and severe hypothermia should consist of gentle handling, spontaneous rewarming, and if available, active core rewarming with heated, humidified oxygen. Active external rewarming in the field should generally be limited to the trunk. Spontaneous rewarming is indicated in mild hypothermia and in stable, thermogenically capable patients. Casualties with moderate and severe hypothermia should be transported to a medical treatment facility and monitored. Some severely hypothermic patients are best managed in facilities with cardiopulmonary bypass capabilities.

Many hypothermic patients will have or will develop factors necessitating active core rewarming. Airway warming with heated, humidified oxygen is a safe, practical option in all healthcare facilities. Truncal active external rewarming is safest with healthy, preferably young, acutely hypothermic patients. Medical officers should consider the choice of the method of active core rewarming after evaluation of the patient's current pathophysiological condition.

Short of freezing, there is probably no absolute temperature from which humans will not rewarm. The temperature from which any individual will not rewarm spontaneously, however, depends on the metabolic rate and the effectiveness of the total body insulation. A versatile approach to therapy may require the simultaneous, sequential, or combined use of various rewarming techniques. Acid–base imbalance, coagulopathies, and septicemia can all be life-threatening complications in hypothermic patients.

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