Chapter 28

SYSTEMIC HYPOTHERMIA

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INTRODUCTION

Military operations in cold environments have the potential to create vast problems for military medical services. Everything is more difficult to do. Evacuation of casualties is impeded. Infectious diseases are much more common. And cold by itself becomes an agent of injury, as was pointed out in 390 BC by the Greek essayist and general, Xenophon, in his *Anabasis*, a description of a 4th-century BC military expedition through Asia Minor.[1] But perhaps the most spectacular manifestation of cold as a source of attrition in the warfare of the ancient world was that of Hannibal, who may have lost as many as 50% of his men, many from hypothermic injury, while crossing the Alps in 218 BC.[2]

The spectrum of cold injury extends from (*a*) transient injury to the most superficial layers of the skin, through (*b*) full-thickness necrosis of varying amounts of the soft tissue of the hands and feet, to (*c*) fatal systemic hypothermia. By far the most common medical treatment problems caused by military operations in cold environments in past wars have been those associated with damage to superficial tissues in the extremities. Such local cold injury is known by a variety of names: immersion foot, trench foot, and frostbite are the best known. During World War I, cold-induced casualties included 38,000 Italian, 80,000 French, and 115,000 British troops. World War II produced 90,000 cold-induced U.S. casualties and 100,000 German.[3] Similar observations are applicable to more recent wars. During the Korean War, cold weather contributed to at least 10% of U.S. casualties,[3] and during the Falklands War in 1982, hypothermia and local cold injuries were quite common.[3–5]

Systemic hypothermia is less well known but is certain to be a much greater therapeutic challenge for military trauma anesthesiologists than is local cold injury. Strangely enough, not a single official history of the U.S. Army Medical Department in World War II mentions this condition, including *Cold Injury, Ground Type,* the volume that is dedicated to cold injury.[6] It is inconceivable that the condition did not occur. Systemic hypothermia was known to contribute to wartime death by drowning. In fact, the Nazi hypothermia "experiments," the scientific validity of which has recently been debunked, were allegedly prompted in part by the high mortality rate among downed German pilots and U-boat crews.[3,7]

It is reasonable to assume that the contribution of systemic hypothermia to combat mortality was obscured by more-dramatic conditions such as battlefield trauma and disease. Soldiers found frozen to death on the battlefield and whose demise was attributed to "exhaustion" or "exposure" were, no doubt, victims of systemic hypothermia. Certainly, the nearly 10% of George Washington's troops who were reported to perish during the cold winter of 1777 and 1778 died of systemic hypothermia.[3,4] Because such casualties die on the battlefield, the importance of systemic hypothermia would not have been recognized at the hospital level. With more rapid means of evacuation from the battlefield, however, it is likely that cases of systemic hypothermia will be seen more frequently in future wars.

Soldiers fighting on a wintry battlefield are not the only candidates for systemic hypothermia. The battle casualty with substantial blood loss who undergoes a lengthy laparotomy or thoracotomy, and who receives inadequately warmed intravenous fluids in a suboptimally climate-controlled field hospital, is obviously at risk for developing intraoperative hypothermia—with its attendant potential for hemodynamic and coagulation abnormalities. The military anesthesia provider's experience with systemic hypothermia is much more likely to be based on casualties who become hypothermic in the operating room than on casualties whose hypothermia was caused by environmental exposure. Ironically, hypothermia was recognized as a problem for the U.S. Army Medical Department during the Persian Gulf War—a war fought in the desert—but not in World War II or Korea:

> Hypothermia was a frequent and significant clinical problem for combat hospitals. Preoperative and intraoperative hypothermia consumed vast amounts of additional oxygen, personnel, and [intensive care unit] resources. DEPMEDS [the Department of Defense's Deployable Medical Systems] provides for warming blankets for the operating room beds, but lacks other methods for active rewarming.[8(p10-37)]

The infrequent observation of intraoperative hypothermia in past wars may reflect the simple fact that no device for measuring intraoperative core temperature has yet been fielded. PROPAQ monitors (manufactured by Protocol Systems, Beaverton,

Ore.), which are fielded, have a thermistor port, but the thermistor itself is not part of the DEPMEDS equipment. DEPMEDS does, however, field a thermometer that reads as low as 21°C, which will permit diagnoses of hypothermia to be made more commonly in the future.

NORMAL THERMOREGULATION

Man, like other mammals and birds, is a homeothermic animal and thus strives to maintain body temperature within a narrow range despite changes in environmental temperature. There is a normal diurnal variation in temperature in healthy individuals, with the nadir typically occurring between the hours of 0200 and 0400. Oral temperatures of 36.1°C (97°F) are common on morning arising and increase gradually during the day to reach a peak of 37.2°C (99°F) or slightly higher between the hours of 1800 and 2200.[9] This normal pattern is not reversed in individuals who work a night shift.

Temperature Measurement

Most scientific literature uses the term *core temperature* as a basis of reference. The precise definition of core temperature and the most appropriate site or sites for its measurement have, nonetheless, been elusive. Despite the inconsistency of rectal temperature and its lack of intrinsic thermal significance, most clinical knowledge of body temperature depended for years on this site of measurement.[10] Tympanic membrane temperature closely approximates the temperature of the blood supplying the hypothalamus, site of the temperature regulation control center.[11] The tympanic membrane, situated at the base of the middle cerebral fossa and separated from the internal carotid artery by a thin layer of bone, would seem the ideal, easily accessible location for measuring core temperature.[10,12]

With the thermistor tip placed in the mid esophagus, investigators from Yale University determined no significant difference between esophageal and pulmonary artery temperatures during either steady-state conditions or hypothermia and rewarming in patients undergoing cardiac surgery.[13] Positioning the esophageal probe where they heard the loudest heart sounds, anesthesiologists at the Arizona Health Sciences Center showed that esophageal temperature and tympanic membrane temperature have a correlation coefficient greater than 0.75.[14] Furthermore, these investigators demonstrated similar correlation for nasopharyngeal and bladder temperatures. Although conceding that tympanic membrane temperature (measured in this study with a Model 6000 monitor manufactured by Mon-a-therm, Inc., St. Louis, Mo.) best approximates the true value for core temperature, they recommend using esophageal, nasopharyngeal, and bladder temperatures because of possible trauma to the tympanic membrane.[14,15] Whichever site is chosen, a device capable of recording low temperatures must be used so that unsuspected hypothermia will not be missed.[16]

Neural Temperature Control

The hypothalamus contains the negative-feedback integrative centers that initiate adaptive responses to perceived changes in temperature. The central temperature control center responds to a variety of stimuli including endocrine influences (eg, the thermogenic effect of progesterone), exercise, sweating, flushing, and mental stress. Peripheral nerve endings innervating the skin, many areas in the body core, and nervous elements in the spinal cord and brain stem are known to be sensitive to temperature changes.[17,18] Preoptic anterior hypothalamic nuclei monitor core temperature. The hypothalamus responds to the rate of firing of thermosensitive neurons, which is partially dependent on the rate of change of the stimulus. Thus, it is possible to abolish shivering by slightly increasing skin temperature at the onset of shivering, thereby allowing core cooling to continue.[19] For example, when deep divers are warmed with flowing hot water, they may continue to become hypothermic although their perception is that they are warm. Radiant heat from a fireplace may produce the sensation of warmth, prevent vasoconstriction, and thereby hasten the development of hypothermia in an otherwise cold room.[19] This fact may explain the observation of Napoleon's chief surgeon, Baron Dominique Larrey, who, noting the effects of the Russian winter on the troops of the Grande Armèe, found that victims of the cold who were placed closest to the campfire usually died.[3,20,21]

Heat Production

At the basal metabolic rate, heat is produced at 40 to 60 kcal per square meter of body surface area per

hour.[3,22] The ultimate source of heat production is from the action of adenosine triphosphatase (ATPase) on adenosine 5′-triphosphate (ATP) in the sodium pump of all biologically active cell membranes.[9] The core organs (brain, liver, spleen, heart, and kidneys) contribute almost 60% of basal heat production,[19] despite constituting only about 10% of body weight. Nonshivering thermogenesis is enhanced by an increase in catecholamines, thyroxine, and adrenocorticoids. Basal heat production can be markedly increased by muscle activity and shivering.

Preshivering enhancement of muscular tone increases heat production by up to 100%.[22] Visible shivering can increase heat production (and oxygen consumption) by 500%.[23,24] Because of fatigue and glycogen depletion, this degree of heat production from shivering is limited to a few hours.[22] Thermoregulatory shivering demonstrates a waxing and waning pattern (4–8 Hz) and differs distinctly from generalized postanesthetic tremor.[25] Unfortunately, activity and shivering are uneconomical ways to increase heat production because they are often accompanied by increased skin and muscular blood flow, which further increases heat loss (Figure 28-1).[19,23]

Heat Loss

Humans are essentially warm-weather, subtropical animals that do not physiologically tolerate or adapt well to cold environments.[4,26,27] Unclothed, we probably could not survive at temperatures much below 21°C (71°F).[22] Human adaptation is primarily behavioral and intellectual: constructing shelters and acquiring clothes and artificial heat. Thermally, the body can be envisioned as a core, consisting of the vital organs and a shell, the inner layer of which comprises the skeletal muscle mass, and the superficial layer of which includes superficial muscle, subcutaneous fat, and skin.[19,21,27,28] Ninety-five percent or more of daily heat production must be lost to the environment.[22] This loss occurs through the shell and the respiratory tract by radiation, conduction, convection, and evaporation.

Radiation

Radiation (the transfer of heat between objects by electromagnetic waves) normally accounts for 55% to 70% of heat loss. The amount of heat loss depends on the temperature gradient between the body surface and the environment, and the amount of exposed body surface area. The uncovered head can be an important source of radiant heat loss. Due to poor vasoconstriction of the face and scalp, at 4°C (39°F), one half the total basal metabolic heat production can be lost from an exposed head.[19,29]

Conduction and Convection

Conduction refers to the transfer of thermal energy through direct contact. Normally, only 2% to 3% of heat loss occurs via conduction to other objects; however, the loss dramatically increases (up to 5-fold) during contact with cold ground or snow. Water's thermal conductivity is some 30-fold greater than that of air, and immersion causes extremely rapid heat loss. Approximately 1,500 passengers on the *Titanic* died in 0°C water in less than 2 hours.[30] Insulation (both clothing and subcutaneous fat) can prolong survival times.[30–32]

After heat is moved from the body's core to the shell by conduction, it can be dissipated by air movement over the skin—a process called *convection*. Heat loss by convection is quite variable and depends on wind velocity (with the cooling effect varying approximately as the square root of the wind velocity)[29] and the degree of insulation provided by clothing. Wet garments, rather than trapping air next to the skin and retarding convective heat loss, become effective conductive heat transmitters.

Evaporation

Evaporation is the conversion of a liquid to its gaseous phase. The conversion of 1 g of water from a liquid to a gas requires 0.58 kcal.[22,28,29] In humans, heat loss by evaporation from the skin and respiratory tract generally accounts for 20% of total heat loss, but this can be increased remarkably by sweating.[22,29] Relative humidity and ambient temperature markedly affect the degree of heat loss by evaporation, with the greatest loss occurring in dry, cold environments.[3]

Heat Conservation

By means of a remarkable ability to decrease or increase blood flow, primarily through cutaneous arteriovenous shunts[33] in the extremities, the human body regulates delivery of heat to its surface. Cutaneous blood flow can decrease from its normal 300 to 500 mL/min to 30 mL/min and can increase to more than 3 L/min with maximum vasodilation.[22,27,34] Capillary flow, as well as arteriovenous shunt flow, decreases with higher degrees of vasoconstriction.[35]

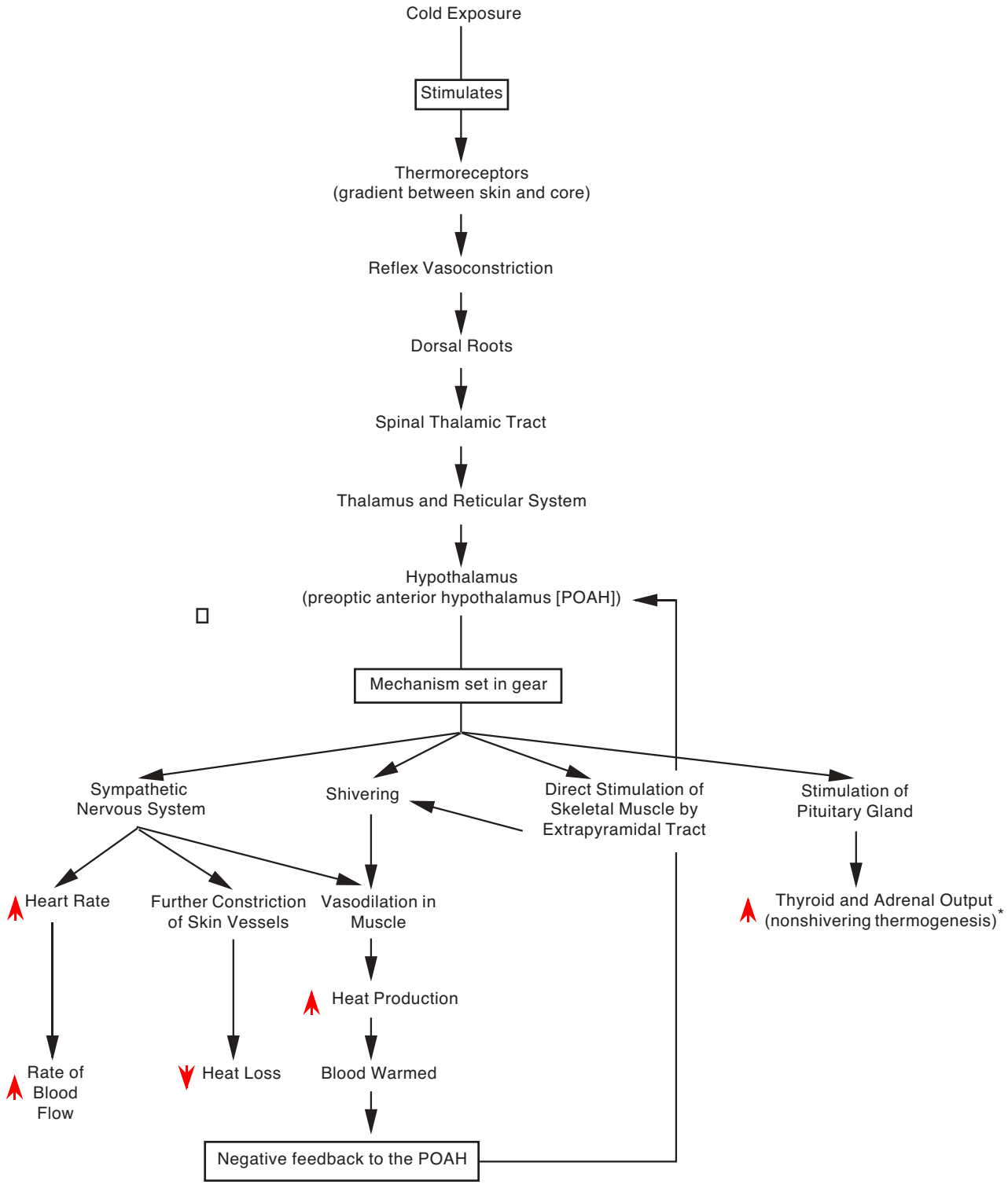

Fig. 28-1. The stepwise and integrated physiological response to cold in the normal human.
Adapted with permission from Reuler JB. Hypothermia: Pathophysiology, clinical settings, and management. *Ann Intern Med*. 1978;89:520.

DEFINITION AND CLASSIFICATION OF HYPOTHERMIA

In humans, hypothermia is defined as a core temperature less than 35°C (95°F).[3,19,22,28,29] In addition to the hypothermia that is deliberately induced in certain surgical patients, there are two broad categories of pathological hypothermia:

1. *primary* (accidental), in which an otherwise healthy person is exposed to a cold environment and the thermoregulatory system's compensation is inadequate[36]; and
2. *secondary*, in which another illness or condition predisposes the individual to accidental hypothermia.[3,22,29,36]

Further subdivisions into *acute* and *chronic* (which afflicts particularly the elderly who live in insufficiently heated winter houses) and *immersion* and *nonimmersion* have been made.[3,36] Another supplemental subclassification distinguishes *very rapid* (usually immersion) hypothermia, in which the cold stress quickly exceeds the body's ability to produce heat and maintain core temperature, and *intermediate* (exhaustion) hypothermia, in which the individual's heat production is able to maintain core temperature against a cold stress only as long as sufficient energy sources are available.[19] In very rapid hypothermia, heat production is unimpaired and the person will have little difficulty rewarming on removal from cold stress. In intermediate hypothermia, heat production is markedly impaired when exhaustion ensues, and "even a relatively mild degree of cold exposure may be sufficient to cause continued cooling."[19(p102)] (This subject is also discussed in Chapter 23, Metabolic Derangements and Nutritional Support.) Primary hypothermia is the concern of this chapter, although military anesthesia providers should bear in mind that they are most likely to encounter casualties with secondary hypothermia: injured soldiers in a cold operating room tent.

A number of diverse clinical conditions lead to impaired thermoregulation, and can generally be classified as central, peripheral, metabolic, pharmacological or toxicological, and dermatological (Exhibit 28-1). Of these, the most significant are probably

- spinal cord transection, which, by interrupting the spinothalamic tract, breaks the afferent thermoregulatory loop;
- trauma associated with hypotension and hypovolemia, which results in generalized vasoconstriction and decreased metabolic heat production;
- general anesthetics, which shift the threshold for vasoconstriction and shivering to a lower temperature range; and
- acute ethanol ingestion, which causes generalized vasodilation and thereby increases heat loss.

CLINICAL AND LABORATORY MANIFESTATIONS

Hypothermia per se is not lethal to tissue: cellular death occurs only with denaturation when ice crystals are formed. "What does occur as cells are gradually cooled is a progressive decrease in functional performance that ultimately kills the organism."[37(p620)] Hypothermia is known to affect most organ systems, the metabolism, and the blood and its chemistry (Table 28-1), but the effects of hypothermia depend on the degree of cooling. Three stages of hypothermia are commonly recognized: mild (> 32°C), moderate (32°C–26°C), and severe (< 26°C). Much of the material that follows is summarized in Table 28-2.

Organ-System Effects

Central Nervous System Effects

An initial deterioration in mental status may progress to dysarthria, incoordination, withdrawal, irritability, apathy, disorientation, confusion, and finally to lethargy, intermittent stupor, and frank coma. Visual evoked potentials decrease[38] and the latency period of somatosensory evoked potentials is prolonged[39] by hypothermia. Although cerebrovascular autoregulation is reportedly preserved to 25°C,[3] hypothermia produces a predominance of slower-frequency waves on the electroencephalogram, which eventually becomes electrical silence at 19°C to 20°C.[3,19] Hyporeflexia progresses to areflexia and is accompanied by an increase in rigidity.[19]

Cardiac Effects

In hypothermia, the usual cause of death is progressive deterioration in cardiopulmonary function. Following an initial increase in heart rate and blood pressure, hypothermia causes a progressive bradycardia and myocardial depression, eventu-

EXHIBIT 28-1

CLINICAL CONDITIONS POSSIBLY RESPONSIBLE FOR IMPAIRED THERMOREGULATION

Central Nervous System Conditions (Affecting Hypothalamic Function)
- Cranial trauma (especially basilar fractures)
- Chronic subdural hematomas, and intracerebral hemorrhages
- Central nervous system infection
- Cerebrovascular accidents
- CNS infiltrative lesions including neoplasms, systemic lupus erythematosus, and sarcoidosis
- Spontaneous periodic hypothermia
- Shapiro's syndrome (dysgenesis of the corpus callosum)
- Wernicke's encephalopathy

Peripheral Conditions
- Neoplasms, especially Hodgkin's disease and other lymphomas[1,2]
- Peripheral neuropathies
- Spinal cord transection*
- Trauma associated with hypotension and hypovolemia*
- Carcinomatosis
- Pancreatitis
- Miliary tuberculosis
- Myocardial dysfunction with decreased cardiac output

Metabolic Conditions
- Hypothyroidism (myxedema)
- Hypoglycemia
- Hypopituitarism
- Hypoadrenalism
- Ketoacidosis
- Anorexia nervosa
- Marasmus and kwashiorkor

Pharmacological/Toxicological Conditions
- General anesthetics*
- Major conduction anesthetics,(ie, subarachnoid block and epidural)
- Ethanol ingestion, acute and chronic*
- Barbiturates
- Phenothiazines
- Benzodiazepines
- Cyclic antidepressants
- Lithium
- Carbon monoxide
- Clonidine
- Narcotics

Dermatological Conditions
- Burns
- Epidermolysis bullosum
- Exfoliative dermatitis

*Most important

(1) Buccini RV. Hypothermia in Hodgkin's disease. *N Engl J Med*. 1985;312:244. (2) Chang M, Gill T. Hypothermia, neurologic dysfunction, and sudden death in a man with carcinoma. *South Med J*. 1981;74:1509.

TABLE 28-1

PHYSIOLOGICAL DERANGEMENTS ASSOCIATED WITH PROLONGED HYPOTHERMIA

System	Effects
Central Nervous	Incoordination
	Disorientation
	Confusion
Cardiac	Decreased heart rate, mean arterial pressure, cardiac output
	Conduction slows, T waves are inverted, QT interval increases
	ST elevation occurs between 32°C–33°C (J wave)
	Atrial and ventricular fibrillation occur < 30°C
Respiratory	Respiratory rate increases early on
	Anatomical and physiological dead spaces increase (at 25°C)
Renal	Cold diuresis occurs late in the course of the injury, leading to volume depletion, oliguria, and azotemia
Hematological	Hemoconcentration occurs, with increased hematocrit and hemoglobin levels
	Disseminated intravascular coagulation can occur from peripheral microvasculature failure
Metabolic	Hyperglycemia and mild ketosis
	Lactic acidosis
Gastrointestinal	Decreased intestinal motility (at < 34°C)
	Hemorrhagic pancreatitis is seen occasionally in fatal cases

Notes:

- The metabolism of all drugs is decreased in hypothermia, as is the liver's ability to detoxify drugs. This should be considered when administering medications to hypothermic patients.
- In the absence of other overriding medical considerations (eg, severe trauma), no one is dead until *warm* and dead.

Source: Bellamy RF, ed. *Combat Casualty Care Guidelines: Operation Desert Storm*. Washington, DC: Office of The Surgeon General, Walter Reed Army Medical Center, and Borden Institute; 1991: 154.

ally terminating in asystole or ventricular fibrillation. Some controversy exists over whether asystole or ventricular fibrillation is the more common terminal rhythm.[3,21,40] Rather clearly, the conduction system is sensitive to cold, and both PR and QT intervals are characteristically prolonged.[3] Ventricular irritability develops near 29°C, and there are reports of ventricular fibrillation in hypothermic patients induced by mechanical stimulation secondary to placement of pulmonary artery catheters, esophageal probes, and endotracheal tubes.[19,37,41]

The J wave (also known as the Osborne wave) is said to be present in up to 90% of hypothermic patients (Figure 28-2).[37] This positive–negative deflection immediately follows the QRS complex—probably caused by delayed depolarization of the left ventricle—and is occasionally observed in patients with subarachnoid hemorrhage, left ventricular ischemia, and in some healthy young adults.

Respiratory Effects

After an initial increase in respiratory rate following sudden exposure to cold, respiratory rate, tidal volume, minute ventilation, and the respiratory response to carbon dioxide all progressively decrease.[22,42,43] Despite a decrease in carbon dioxide production, minute ventilation becomes inadequate to remove carbon dioxide below approximately 32°C.[3,22] The cough reflex and ciliary motility are depressed, resulting in an impaired ability to deal with frequently encountered bronchorrhea and pulmonary edema.[3,19,22] Autopsy findings often include not only pulmonary edema but also parenchymal hemorrhages.[22,44]

TABLE 28-2

CHARACTERISTICS OF THE THREE STAGES OF HYPOTHERMIA

Stage	Core Body Temperature (°C)	Core Body Temperature (°F)	Characteristics
Mild	37.6	99.6 ± 1	Normal rectal temperature
(> 32°C)	37	98.6 ± 1	Normal oral temperature
	36	96.8	Increase in metabolic rate
	35	95.0	Urinary temperature 34.8°C; maximum shivering thermogenesis
	34	93.2	Amnesia; dysarthria, and poor judgment develop; normal blood pressure; maximum respiratory stimulation
	33	91.4	Ataxia and apathy develop
Moderate	32	89.6	Stupor; 25% decrease in oxygen consumption
(26°C–32°C)	31	87.8	Extinquished shivering thermogenesis
	30	86	Atrial fibrillation and other arrhythmias develop; poikilothermia; pulse and cardiac output 67% of normal; insulin ineffective
	29	85.2	Progressive decrease in level of consciousness, pulse, and respiration; pupils dilated
	28	82.4	Decreased ventricular fibrillation threshold; 50% decrease in oxygen consumption and pulse
	27	80.6	Loss of reflexes and voluntary motion
	26	78.8	Major acid–base disturbances; no reflexes or response to pain
Severe			
(<26°C)	25	77	Cerebral blood flow 33% of normal; cardiac output 45% of normal; pulmonary edema may develop
	24	75.2	Significant hypotension
	23	73.4	No corneal or oculocephalic reflexes
	22	71.6	Maximum risk of ventricular fibrillation; 75% decrease in oxygen consumption
	20	68	Lowest resumption of cardiac electromechanical activity; pulse 20% of normal
	19	66.2	Flat EEG
	18	64.4	Asystole
	16	60.8	Lowest adult accidental hypothermia survival
	15.2	59.2	Lowest infant accidental hypothermia survival
	10	50	92% decrease in oxygen consumption
	9	48.2	Lowest therapeutic hypothermia survival

Reprinted with permission from Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PS, Gheer EC, eds. *Management of Wilderness Environmental Emergencies*. 2nd ed. St. Louis, Mo: Mosby–Year Book; 1989: 39. In: Danzl DF. Accidential hypothermia. In: Rosen et al, eds. *Emergency Medicine: Concepts and Clinical Practice*. 2nd ed. St. Louis, Mo: CV Mosby; 1988.

Vascular, Renal, and Gastrointestinal Effects

Vascular and Renal Effects. The remarkable ability of the body to constrict blood vessels in the extremities produces an initial, relative, central hypervolemia, which is interpreted as a volume overload leading to suppression of the antidiuretic hormone and the well-known cold diuresis. As

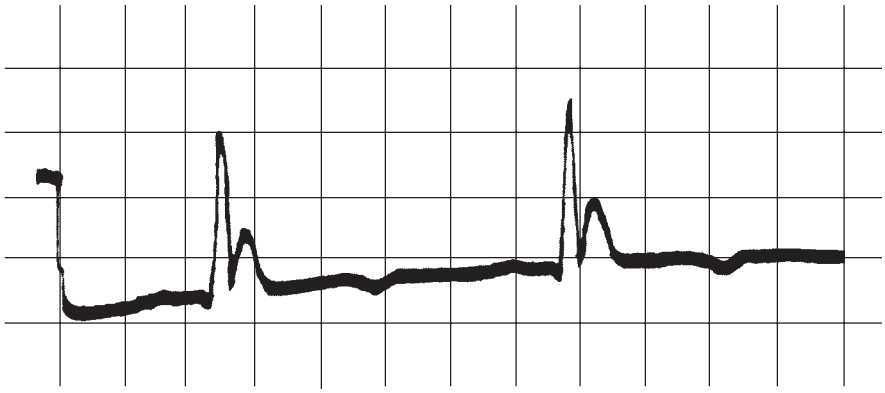

Fig. 28-2. This electrocardiograph shows typical J waves (also called Osborne waves), which follow the QRS complex that develops at temperatures below 32°C.

renal tubular dysfunction progresses, little of the glomerular filtrate is reabsorbed, and this, combined with shifts of fluid from the intravascular space, produces hypovolemia.

Gastrointestinal Effects. Gastrointestinal hemorrhage and pancreatitis are common autopsy findings.[21,45] Gastrointestinal motility decreases concomitant with the decline in metabolic rate. Functional hepatic impairment occurs with little if any structural damage,[37] altering the rate of clearance and metabolism of many drugs.

Metabolic and Hematological Effects

Metabolic Effects. Following an initial increase in metabolic rate up to 6-fold greater than the basal rate[3,22–24,46] at 35°C, hypothermia slows all metabolic processes, with the metabolic rate decreasing to 50% of normal at core temperatures of 28°C to 30°C.[28,42] A reduction in metabolism and oxygen consumption of 7% per degree Celsius decrease in temperature is widely reported.[16,42]

Hematological Effects. As a consequence of hemoconcentration and a reported[40,47] 2% increase in viscosity per degree Celsius decrease in temperature, hematocrit usually increases along with significant "sludging" and interference with tissue perfusion.[3,21,36] Platelet and leukocyte counts are frequently depressed because of splenic and hepatic sequestration.[28,42] Platelet sequestration occurring in the portal bed is largely reversible on rewarming.[48,49]

Blood Chemistry Abnormalities

Electrolytes and Serum Glucose

Preexisting clinical conditions can result in transcellular membrane shifts of electrolytes and produce significant alterations. Unfortunately, there are no predictable changes in serum sodium, chloride, potassium, magnesium, calcium, or phosphate[3,22]; therefore, frequent laboratory determinations are necessary during resuscitation from hypothermia. Cold- and catecholamine-induced glycogenolysis and decreased insulin secretion[37] cause an initial hyperglycemia that proceeds to hypoglycemia with glycogen depletion.[3] Note that insulin is ineffective below 30°C.[3] Therefore, persistent hyperglycemia during rewarming can indicate diabetic ketoacidosis, pancreatitis, or both.

Dissolved Gases and the Acid–Base Balance

The solubility of gases in liquids increases at colder temperatures; therefore, the partial pressures of both oxygen and carbon dioxide (P_{O_2} and P_{CO_2}) decrease in the hypothermic patient. The partial pressure of arterial oxygen (Pa_{O_2}) declines 7.2% per degree Celsius decrease, and the partial pressure of arterial carbon dioxide (Pa_{CO_2}) declines 4.4% per degree Celsius decrease.[50] Because blood samples are warmed to 37°C before determination of P_{O_2} and P_{CO_2}, temperature correction of both of these laboratory values was formerly advocated. Such temperature correction of P_{O_2} is useful clinically. A Pa_{O_2} of 60 mm Hg at 27°C corresponds to a Pa_{O_2} of 130 at 37°C.[3] The lower Pa_{O_2} at 27°C represents not oxyhemoglobin desaturation but simply a lower partial pressure of oxygen due to its increased liquid solubility at the lower temperature.

The situation with appropriate evaluation of pH and Pa_{CO_2} is somewhat more complicated but has now essentially been resolved in favor of *not* correcting for temperature when measuring pH and P_{CO_2}.[3,51,52] The word *correcting* is to be understood in

the following sense: commercially available pH meters automatically warm a blood sample to 37°C, the temperature at which the pH is actually measured. There are two options for reporting the results of this measurement: (1) to report the results at 37°C or (2) to "correct" them to the original temperature of the sample. Because pH normally increases as temperature falls, the uncorrected pH measured at 37°C will be lower than the pH corrected to the hypothermic casualty's actual body temperature.

Studies of cold-blooded (ie, ectothermic) vertebrates show that their blood pH increases and their P_{CO_2} decreases as their body temperatures cool.[51–53] Some of the decrease in P_{CO_2} results from the increased solubility of carbon dioxide at the lower blood temperature, but there is also decreased carbon dioxide production incident to the decreased metabolic rate. In these animals, total body carbon dioxide content[3,51] does not change with decreasing temperature because ventilation decreases less than does metabolism. As body temperature falls, the pH of the blood of these animals increases in such as way as to remain slightly alkalotic. By way of contrast, in some small hibernating mammals, pH does not change as temperature falls. These animals hypoventilate and their total body carbon dioxide increases. Thus, their blood becomes progressively acidotic.

There are two basic approaches to managing pH alterations during hypothermia, both of which resemble the natural physiological responses described above. In the technique called *alpha-stat pH management*, chemical neutrality is maintained by allowing pH to increase as temperature falls. Conversely, in the approach known as *pH-stat management*, pH is maintained constant at 7.4 regardless of the temperature.[51,52]

Alpha-stat pH management employs uncorrected blood gases, whereas pH-stat management utilizes corrected gases and strives to maintain a constant pH of 7.4 regardless of the temperature. The former term derives from the alpha imidazole ring on the histidine moiety of various body proteins.[51,52] The neutral pH of water (ie, that pH at which $[H^+] = [OH^-]$ is 6.8 at 37°C),[53] and the normal 0.6 pH units of alkalinity of human blood may well be important for enzymatic function.[3,51–5] In addition,

> [m]any essential enzymes (such as lactic dehydrogenase, sodium-potassium-adenosine triphosphatase, and cytochrome C reductase) have been found to be temperature sensitive and to exhibit optimum activity when pH is increased during hypothermia.[51(p1643)]

The key factor is probably chemical neutrality rather than a specific hydrogen ion concentration.

Several studies done in endotherms, including humans, have demonstrated improved cardiac performance and electrical stability when the pH under hypothermic cardiopulmonary bypass was deliberately determined by noncorrected laboratory values.[55,56] For example, the pH determined at 37°C might be found to be 7.4, which would correspond to a pH of 7.7 when measured at 28°C. Similarly, increased cerebral blood flow was also seen when the *noncorrected* values for P_{CO_2} and pH were used and the animals were allowed to remain hypocarbic and alkalotic.[57] In a 1986 study[58] of 181 cardiopulmonary bypass patients, 40% of those managed with the pH-corrected technique developed spontaneous ventricular fibrillation, whereas only 20% of those managed with the noncorrected technique did so.

Other authorities have also concluded that pH correction can be deleterious in the patient with hypothermia:

> Potentially deleterious effects of this alkalosis on other organ systems have yet to be identified. However, it is clear that maintaining the corrected pH at 7.4 and P_{CO_2} at 40 mm Hg during hypothermia depresses cerebral and coronary blood flow and cardiac output, and increases the incidence of lactic acidosis and ventricular fibrillation. A correction of pH and P_{CO_2} in hypothermia is unnecessary and potentially deleterious.[3(p46)]

In other words, attempting to maintain an actual pH of 7.4 in a hypothermic casualty will have a deleterious effect because the corresponding uncorrected pH is profoundly acidotic.

CLINICAL PREVENTION AND TREATMENT

In cold environments, body temperature can be maintained by increasing body heat production, decreasing body heat loss, and supplying external heat sources. However, hospital operating rooms are kept cool and patients undergoing surgery are not warmly clothed, so the most practical and efficient methods to decrease heat loss (ie, clothing and shelter—our primary adaptations to cold) are contravened. This is especially true in field hospitals that are deployed in tents. The fact that casualties

are not kept warm during surgery led directly to the development of the medical unit, self contained, transportable (MUST) and DEPMEDS hospitals, which allowed temperature-controlled interiors.

All anesthetized patients become hypothermic in rooms cooler than 20°C unless preventive measures are used.[59,60] Ambient temperatures must be 24°C to 26°C to prevent a decrease in esophageal temperature.[60] Radiation and convection account for approximately 80% of heat loss, and most of this occurs during the first hour of anesthesia.[42,60] Methods of heat conservation *must* be a priority of the operating room staff and *must* begin before the casualty is brought into the operating room. In addition to increasing the ambient temperature, the operating room staff must also ensure that the casualty—particularly his head—is covered as much as possible,[19] and must minimize evaporative heat loss from open surgical wounds, especially those of the trunk.

All the methods described below, except cardiopulmonary bypass, increase temperature rather slowly, in the range of 0.5°C to 1.5°C per hour.[3,61] There is no proof that rapid rewarming improves survival rates.[3]

Supplemental Warming Devices

Warm blankets provide some conductive heat transfer as well as decrease radiant and convective heat loss.[42] Although not available in DEPMEDS-equipped hospitals, the "space blanket," a reflective blanket using aluminized Tyvek (manufactured by King-Seeley Thermos Co., Winchester, Mass.) is particularly useful when 60% or more of the body surface area can be covered.[62] In 1980, researchers conducted a study of patients undergoing major vascular procedures with an average operating room time of 6 hours. Before they were draped for surgery, the patients were placed in operating rooms that were cold (14°C–18°C) or warm (23°C–26°C). The latter group also received intravenous fluids and blood that were warmed to 37.5°C and were placed on a heating blanket at 37.8°C. The researchers were able to confirm greater heat loss prior to draping in the cold-room group, but were unable to demonstrate a difference in outcome. The key factor seemed to be the ability of the patients to compensate for internal heat loss that had occurred over an extended period of time. Because many procedures do not last 6 hours, and because the patients in this (and other) studies initially lose more heat in cold operating rooms,[60] operating rooms need to be warmed.[42]

Placing blankets that contain warm, circulating fluid between the operating-table mattress and the patient does *not* prevent intraoperative hypothermia when used alone.[63,64] This finding is not surprising because (*a*) there is little well-perfused tissue in contact with the blanket and (*b*) the thermal conductivity of the usual operating-table mattress is rather low. (However, these blankets have caused burns.[42]) Warmed, humidified, inspired gases used in conjunction with a warming blanket, however, produce a synergistic effect and better temperature preservation than when either is used alone.[64]

The efficacy of warmed, humidified gases as a method of conserving heat is easily understood when it is appreciated that a patient breathing dry gases at *room* temperature can consume 25 kcal/h (about one third of the basal heat production) in warming that air to *body* temperature.[42,65] Several studies have demonstrated the efficacy of passive heat and moisture exchangers in significantly reducing the rate of fall of core temperature,[65–67] especially when lower fresh-gas flows are used. In addition to preventing the expenditure of approximately 25 kcal/h to warm and humidify dry room-temperature gases,[3,42,65] heated (45°C), humidified gases delivered at 20 L/min provide about 30 kcal/h heat exchange to a patient with a core temperature of 28°C (Table 28-3).[61]

In awake hypothermic volunteers, one group of researchers[68] demonstrated an increase in the rate of core rewarming of 0.3°C/h for each 10 L/min increase in ventilation of moist air with an inspiratory temperature of 44°C. Clearly, lesser rates of heat transfer occur if the gases are cooler and if the patient's core temperature is higher.[61] Other groups of researchers[61,69] performed their calculations using 45°C as an accepted maximum airway temperature, and still others[70] used inspired gases at 42°C to 47°C at the Y connector of the anesthesia tubing.

In one notable study,[71] however, researchers studied dogs that had been mechanically ventilated for 6 hours. The group monitored temperatures at the internal end of the endotracheal tube and demonstrated an acute airway injury consisting of

> diffuse mucosal degeneration with focal necrosis, shedding and sloughing of the pseudostratified columnar epithelium, and an acute inflammatory response in the submucosal tissue [when] average tracheal temperature exceeded 40°C.[71(pA490)]

These investigators did not control the peak temperature, however, and report peaks of 46°C "when the average tracheal temperature was

TABLE 28-3

ESTIMATED HEAT GAIN FROM ENDOGENOUS AND EXOGENOUS SOURCES

Heat Source	Calories Provided at Core Temperature 28°C*
Normal metabolic rate	70 kcal/h
Maximum shivering	350 kcal/h
Heated (45°C) humidified O_2 at 20 L/min	30 kcal/h
Heated (45°C) intravenous fluid (1 L)	17 kcal
Heated (45°C) peritoneal dialysis fluid (1 L);	17 kcal
flow rate 5 L/h over 1h	85 kcal/h
Heated (42°C) cardiopulmonary bypass perfusate (1 L);	17 kcal
flow rate 28 L/h	476 kcal/h
Trunk immersion in 45°C water:	
Vasoconstriction	600 kcal/h
Vasodilation	2,400 kcal/h

*A 70-kg human requires a gain of 60 kcal of heat to increase body temperature 1°C

Reprinted with permission from Bangs C, Hamlet MD. Hypothermia and cold injuries. In: Auerbach BS, Geehr EC, eds. *Management of Wilderness and Environmental Emergencies*. New York: Macmillan; 1983: 38. Original data source: Myers RAM, Britten JS, Cowley RA. Hypothermia: Quantitative aspects of therapy. *Journal of the American College of Emergency Physicians*. 1979:8(12):523–527.

42°C."[71(pA490)] In addition to retarding heat loss and providing heat gain, heated and humidified gases stimulate pulmonary cilia and retard cold-induced bronchorrhea.[3,72]

Blood and Fluid Warmers

Approximately 15 kcal (20% of basal heat production) are required to warm 1 L of 20°C intravenous fluid to 30°C.[42,61] Because general anesthesia ablates shivering and produces increased blood flow to the skin, rendering the normally homeothermic human a poikilotherm (ie, an ectotherm)[17,36,73] and therefore unable to increase caloric production, a patient's temperature will decrease more rapidly when challenged with cold intravenous fluids. The rapid administration of cold (4°C) blood products represents a considerable thermal challenge, one that is capable of reducing temperature approximately 0.5°C for every liter transfused over a 15-minute interval.[42] In addition to long-available heating systems (eg, the Dupaco Hemokinititherm Fluid Warmer, manufactured by Dupaco, Oceanside, Calif., which uses a countercurrent multiplier system of tubing immersed in a container of warmed water), the Level 1 Fluid Warmer (manufactured by Life Systems, Inc., Southfield, Mich.), is now available and can warm fluid and cold blood to 35°C at flows up to 500 mL/min.[74] Additionally, glucose-free intravenous fluids in flexible plastic containers can be preheated in a microwave oven.[3,75,76] (Preliminary testing of the warming characteristics of each oven should be done, and the fluid should be thoroughly mixed before it is administered.[3]) Packed red blood cells can be mixed with warm, calcium-free, isotonic crystalloid,[77] or can be prewarmed in a standard operating room fluid-warming cabinet,[78] provided moderate temperature ranges and proper temperature control are maintained.

Treatment in the Postanesthesia Care Unit

The methods of treatment previously discussed should be continued for casualties in the postanesthesia care unit who have subnormal temperatures. To prevent or diminish postanesthesia shivering and its consequent increase in oxygen consumption, high-risk casualties with temperatures lower than 35°C should be kept intubated, sedated, and paralyzed until they are rewarmed. The rewarming rate is the same when using either radiant heat lamps or heated blankets changed every 30 minutes,[79] and both methods are more effective than

only one application of a warmed blanket.[80] Radiant heat and other methods of skin-surface warming also prevent or inhibit shivering.[19,37,81] A new device, the Bair Hugger (manufactured by Augustine Medical, Inc., Eden Prairie, Minn.), provides a microenvironment of warmed air through slits in a disposable blanket made of paper and plastic. In a postoperative patient lacking thermoregulatory responses, the Bair Hugger effectively transferred sufficient heat to increase the mean body temperature approximately 1.5°C/h,[82] and should be similarly effective during anesthesia. Spinal and epidural anesthetics may extend the risk of hypothermia longer into the recovery period than does general anesthesia, by virtue of the prolonged sympathectomy and associated vasodilation[83] combined with their interruption of afferent fiber activity from peripheral thermoreceptors.[36] But military trauma anesthesiologists must remember that an awake, nonshivering, postoperative patient is not necessarily a normothermic one, and rewarming may well still be required.

Nonhypothermic Shivering

Although the precise mechanism of nonhypothermic shivering remains unclear, even nonhypothermic postanesthetic tremor exacts a high metabolic cost[25] and must be curtailed in patients who are less able to tolerate the increased oxygen consumption. Meperidine, but not morphine or fentanyl, often stops postanesthetic shivering.[84–86] This may be because residual anesthetic inhibits descending cortical control, which, in turn, results in reflex hyperactivity.[25] A dose of 12.5 mg to 25 mg, administered intravenously, is effective in 67% to 73% of patients, and 50 mg is successful in 89%.[84]

Core Rewarming Techniques

The first report of core rewarming appeared in 1957 in *Time* magazine: 78 L of warm saline was used as a mediastinal irrigation via thoracotomy.[87] Intragastric and intracolonic warm irrigation have also been used. Cardiopulmonary bypass is the most rapidly effective means of active body core rewarming[3,21,61] and can, moreover, sustain viability until adequate myocardial function resumes. Although less effective, hemodialysis is also an effective modality. Heated peritoneal dialysis is the most efficacious technique of core rewarming, second only to cardiopulmonary bypass and hemodialysis.[3,61,72,88] This is a relatively simple procedure to perform by either a "mini-laparotomy" or the percutaneous approach, using isotonic dialysate at 40°C to 50°C.[3,88–90] Peritoneal dialysis with 45°C dialysate at 5 L/h will provide 85 kcal/h, which should increase core temperature about 1°C/h (see Table 28-3).[61]

Surface Rewarming Techniques

Although it has been calculated to yield up to 2,400 kcal of heat transfer per hour,[61] active external rewarming by immersing the casualty's trunk in 45°C water may be dangerous, especially if active core-rewarming methods are not simultaneously used.[3,30,37,40,91] Reflex vasodilation from external heat application can cause *rewarming shock* as circulating vascular volume in an already hypovolemic core is diverted to the peripheral circulation.[30,37,40,92] This is a much greater problem in patients with chronic exposure hypothermia[30,61] than in patients with sudden immersion hypothermia, who have not yet developed hypovolemia. In addition, cardiopulmonary resuscitation is impossible with a patient immersed in a water bath.

An effective field device for surface rewarming that relies on a charcoal-fueled heating element to provide energy was developed by the Norwegian military and is available for use in the U.S. Army: the Heatpac Personal Heater (manufactured by Alcatel Innova A/S, P.O. Box 60, Økern, 0508 Oslo 5, Norway; and distributed by Norsk Enterprises Inc., Alexandria, Va.). The Heatpac is a 23-cm x 12-cm x 6-cm package weighing 500 g with a combustion time of 6 to 20 hours and heat output of 40 to 250 W. The proprietary charcoal fuel pack weighs 120 to 160 g. The heat energy from charcoal combustion is distributed via a 1.5-V, D-cell–driven electric fan and a series of gas ducts. Carbon monoxide pollutants are scavenged by a catalyst. The unit has been field-tested by the U.S. armed forces and found to be a practical adjunct in the treatment of hypothermic casualties. The Heatpac has been adapted successfully to the U.S. casualty evacuation bag (National Stock Number [NSN] 6530-01-109-9030). Test documentation may be requested from Norsk Enterprises Inc. or the U.S. Army Medical Bioengineering Research and Development, Fort Detrick, Frederick, Maryland 21701-5010. Heatpac is available in the inventories of the North Atlantic Treaty Organization countries and the U.S. Army:

- the charcoal heater bag is NSN 6530-01-255-0835,
- the heating unit alone is NSN 6530-01-254-6492, and
- the charcoal element is NSN 6530-01-254-4130.

CARDIOPULMONARY RESUSCITATION

The presence of such severe prognostic signs as rigor mortis; livor mortis; and fixed, dilated pupils are not reliable criteria for withholding cardiopulmonary resuscitation (CPR) in casualties with hypothermia.[3,93] Considerable controversy still exists, however, regarding the initiation, maintenance, and techniques of CPR in the pulseless, apneic, severely hypothermic (< 30°C) person.[3,37,94,95] The recommendation to search for a pulse for 1 full minute has been made.[94] The clinical aphorism that a patient is not dead until he is *warm* and dead continues to apply.

CPR requires protocol modification with progressive degrees of hypothermia. This fact is explicitly recognized in the most recent recommendations for the treatment of systemic hypothermia made by the American Heart Association (Figure 28-3)[96]; (note that the American Heart Association's temperature criteria for the severity of hypothermia are different from those given in Table 28-2). Patients are divided into two categories by this protocol: those with both pulse and breathing present and those with pulse or breathing or both absent. Those with spontaneous cardiac and respiratory activity are further stratified by core temperature. Above 34°C, passive rewarming (which in the field means placing the casualty in a warm environment, or covering with blankets or a warm sleeping bag) is all that should be necessary. Patients with core temperatures between 30°C and 34°C may benefit, in addition to passive rewarming, from active external rewarming using the Heatpac Personal Heater. Those whose core temperature is below 30°C and who have a supraventricular rhythm and stable vital signs should undergo active internal rewarming. In the field, this means intravenous infusion of warm (43°C) fluids; breathing warm (42°C–46°C), humidified oxygen; and, possibly, peritoneal lavage with warm (43°C), potassium-free fluid.

Active internal rewarming using extracorporeal bypass incorporating a heat exchanger is an extremely effective modality but is not available in DEPMEDS-equipped hospitals. A procedure similar to hemodialysis was originally proposed, with blood being taken from the femoral artery, passed through a heat exchanger, and then transfused into the femoral vein. Nowadays, however, femoral vein–to–femoral artery bypass with a pump is used because the amount of blood that can be warmed is greatly increased. If the casualty is being ventilated and has effective cardiac action, an oxygenator is not absolutely necessary but is included in the circuit because of the propensity of such patients to develop ventricular fibrillation—even when treated prophylactically with antifibrillatory bretylium tosylate and while avoiding fibrillary stimuli (eg, chest compression and placement of intracardiac catheters).

The vulnerability of hypothermic patients to ventricular fibrillation is an important issue. In fact, some authorities[97,98] recommend that CPR be avoided in the severely hypothermic patient until asystole or ventricular fibrillation can be electrocardiographically demonstrated. As myocardial tissue cools, the function of the conduction system degrades and dispersion of relative refractory periods occurs. These phenomena may contribute to the propensity for malignant dysrhythmias that is seen with severe hypothermia. A continuum of effects is seen:

- The higher pacing centers are progressively inactivated, starting with the sinus node.
- Prolongation of intervals and changes in T-wave morphology is noted.
- The terminal deflection of the QRS, the J-wave, is classically seen. (Although Osborne, their discoverer, asserted that J-waves were the result of injury, this is now believed to be unlikely.[98])
- At 32°C, atrial dysrhythmias are expected.
- As the core temperature approaches 28°C, ventricular fibrillation may occur with minimal stimulation.

Unfortunately, these dysrhythmias are refractory to the standard dosages of antidysrhythmics, as well as to electrical defibrillation, below 28°C to 30°C.[99] This phenomenon included ventricular fibrillation that is unresponsive to lidocaine and procainamide. Bretylium tosylate has been credited with effective chemical defibrillation,[100] following earlier reports wherein bretylium's prophylactic role, as opposed to its therapeutic one, was experimentally appreciated.[101]

Airway security is an important goal in the severely hypothermic patient who is at increased risk for aspiration. However, endotracheal intubation itself can cause iatrogenic dysrythmogenic problems. The key is gentle manipulation of the airway at the time of intubation.[102] The goal of minimizing stimulation during intubation is emphasized.[103] Nasotracheal intubation is advocated by some.[104,105]

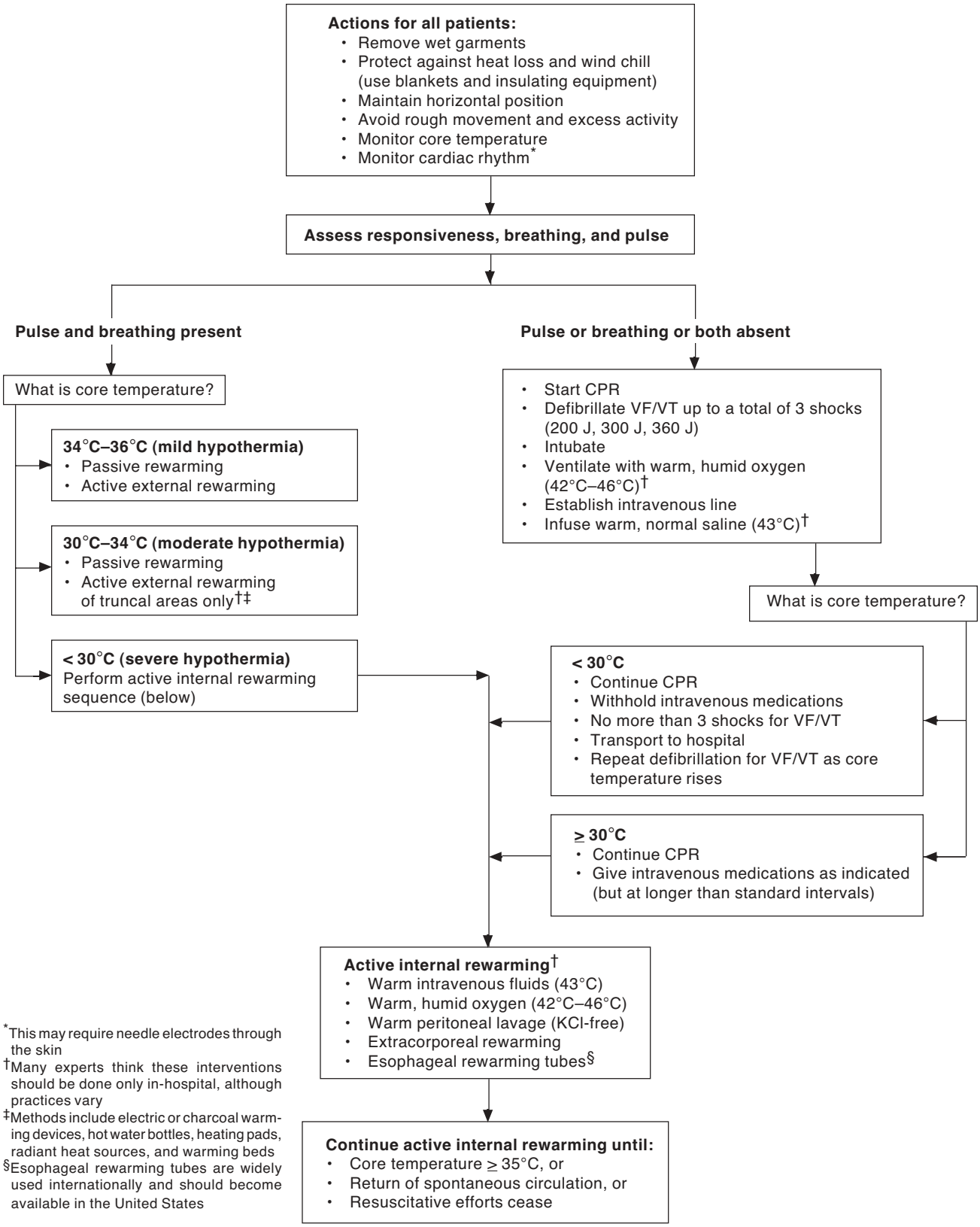

Fig. 28-3. Algorithm for the treatment of hypothermia. CPR: cardiopulmonary resuscitation; VF: ventricular fibrillation; VT: ventricular tachycardia. Reprinted with permission from Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA*. 1992;268:2245.

It should be borne in mind, as previously mentioned, that avoiding respiratory alkalosis is important. Accordingly, low ventilatory rates (eg, 5 breaths per min) are indicated in the severely hypothermic patient.

The American Heart Association recommendations for managing the pulseless, apneic, hypothermic patient are to begin CPR and to attempt defibrillation if ventricular fibrillation is diagnosed. The latter intervention is unlikely to be successful if the core temperature is below 30°C. Rapid rewarming using a femoral-to-femoral bypass should begin as soon as possible. An oxygenator is required when the heart is arrested. Blood is taken from the femoral vein, passed through an oxygenator and heat exchanger, and then pumped back into the femoral artery. Although in the past, access to the femoral vessels required their surgical exposure, there has been increasing success using percutaneous techniques, which greatly simplifies this intervention.[106] Bypass rewarming is continued until the patient is hemodynamically stable or cardioversion at 28°C to 30°C is successful. Given the lack of cardiopulmonary bypass capability in the combat zone, the only therapeutic option for hypothermic casualties who are both apneic and in cardiac arrest is to perform a thoracotomy, through which open-chest cardiac massage and warmed saline pericardial irrigation can be carried out.

Closed-chest cardiac massage in severely hypothermic patients has not been exhaustively studied, but it has proven to be effective. Recommendations to alter the absolute or relative rates of ventilation and closed chest massage are based on clinical experience and animal experimentation. CPR rates of massage and ventilation that are one-half normal are indicated in severe hypothermia. Both the reduced metabolic demand of hypothermic patients and the desirability of avoiding potentially deleterious alkalosis underlie the rationale. One group of researchers investigated changes in regional blood flow that were the result of hypothermia-induced cardiac arrest in pigs. Initially, the hypothermic pigs had lower perfusion indices than their normothermic counterparts, probably due to decreases in chest-wall compliance. Hypothermia reduced the cardiac output 50%; cerebral blood flow, 55%; and myocardial blood flow, 31% vis à vis arrested normothermic pigs. Interestingly, within 20 minutes of cardiac arrest and initiation of CPR, the normothermic group suffered continued decline in these parameters so that there was no significant difference between the two groups.[107]

The controversial issue of blood-gas interpretation remains unresolved. Nevertheless, treatment of profound acidosis using the alpha-stat method has been demonstrated clinically[108] in a severely hypothermic patient. This approach challenges the recommendation of some authorities[22] to defer bicarbonate treatment until the patient's temperature has reached 32°C to 35°C.

The determination of death is complicated by not only the unreliability of pupillary signs but also the invalidity of electrocardiography and electroencephalography, the standard assessment tools.[109] A definitive end point of resuscitation is irreversibility of arrest in a patient whose core temperature is 35°C or higher.

OTHER CONCERNS OF SPECIAL INTEREST

In addition to the matters previously discussed and their application to anesthesiologists both inside and outside the operating room, hypothermia has some other effects of particular concern to military trauma anesthesiologists and critical care specialists:

- Minimal alveolar concentrations of halothane and isoflurane decrease with temperature in a manner similar to the decrease in oxygen consumption (ie, a 5% decrease compared to a 7% decrease in oxygen consumption per degree Celsius decrease in temperature).[16,42,110]
- Although data do not exist for all drugs used by anesthesiologists, the functional hepatic impairment caused by hypothermia increases the half-life of free morphine from 3.7 minutes at 37°C to 98 minutes at 25°C.[37] We can logically deduce that other narcotics, barbiturates, and benzodiazepines would be similarly affected by hypothermia.
- The effect of neuromuscular blocking drugs is enhanced and generally prolonged by hypothermia.[111–114]
- In addition to its other effects on pulmonary function, hypothermia both significantly increases pulmonary vascular resistance and inhibits hypoxic pulmonary vasoconstriction.[115]

SUMMARY

Environmental cold has the potential to cause two significant problems for the military anesthesia provider practicing combat casualty care: local cold injury, of which frostbite is the best known, and systemic hypothermia. The morbidity resulting from the former may cause significant attrition, but the condition is unlikely to be fatal. The importance of the latter as a source of combat mortality seems to be underrated, possibly because death in such casualties is usually ascribed to more obvious physical trauma or disease. It may also be that the lack of a means to accurately measure core temperature in the field means that there is no way to diagnose systemic hypothermia.

Normal thermoregulation depends on a sequence of events that starts with the activation of peripheral thermoreceptors, the integration of the afferent signals in the preoptic nucleus of the hypothalamus, and the activation of efferent pathways that cause vasoconstriction in the skin and the fibrillary contraction of skeletal muscles known as shivering. The latter mechanism increases the production of heat, while the former decreases heat loss. The mechanisms responsible for thermoregulation in the otherwise healthy combat casualty are likely to be impaired by the casualty's trauma—the decreased heat production associated with shock and the increased heat loss from large, open wounds or surgical incisions—and by the general anesthesia, which, although it makes possible the needed resuscitative surgery, may also prevent peripheral vasoconstriction and shivering. To make matters worse, the casualty may arrive from the field already hypothermic, and the ambient temperature within the typical combat zone operating room, especially if the facility has deployed in tents, is likely to be either too cold or too hot.

It is essential that military anesthesia providers assure that there is no further cooling of the hypothermic casualty in the operating room. Although the mildly hypothermic combat casualty (core temperature > 34°C) may initially require only passive external rewarming, conditions may develop during a long operation that will magnify the heat loss. External active rewarming, and such active internal rewarming modalities as intravenous infusion of fluids heated to 43°C and even peritoneal lavage with warm saline, should be undertaken prophylactically. Casualties whose core temperatures are below 30°C will certainly need active internal rewarming, as they are at grave risk of developing ventricular fibrillation. If available, active internal rewarming using a pump oxygenator and femoral-to-femoral bypass should be instituted. Hypothermic combat casualties who have sustained cardiac arrest are unlikely to be salvageable. Closed-chest massage should be started while active rewarming proceeds. Defibrillation may be possible but is unlikely to be successful until core temperature reaches 30°C to 32°C. In lieu of cardiopulmonary bypass, open-chest cardiac massage with lavage of the heart and mediastinum with warm saline may be all that can be done.

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