MEDICAL ASPECTS OF HARSH ENVIRONMENTS
Volume 1

The Coat of Arms
1818
Medical Department of the Army

A 1976 etching by Vassil Ekimov of an
original color print that appeared in
The Military Surgeon, Vol XLI, No 2, 1917

The first line of medical defense in wartime is the combat medic. Although in ancient times medics carried the caduceus into battle to signify the neutral, humanitarian nature of their tasks, they have never been immune to the perils of war. They have made the highest sacrifices to save the lives of others, and their dedication to the wounded soldier is the foundation of military medical care.

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MEDICAL ASPECTS OF HARSH ENVIRONMENTS VOLUME 1

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Contents

Foreword

Earth's environments have always influenced the planning and conduct of military operations. Past campaigns have been impacted by heat, cold, and altititude, as well as the changes in barometric pressure that divers face in special operations. During the 20th century alone, US armed forces have been involved in terrestrial military operations in hot climates in the North African campaign and Pacific theater operations during World War II, the Vietnam and Persian Gulf wars, and military and humanitarian operations in Panama, Haiti, Grenada, Rwanda, and Somalia. Our major military operations involving cold climates during the past century include World War I and World War II, the Korean War, and most recently in Bosnia and Kosovo. *Medical Aspects of Harsh Environments, Volume 1*, treats the major problems caused by fighting in heat and cold.

The topics of *Medical Aspects of Harsh Environments, Volume 2*, are the effects of altitude, especially as experienced in mountain terrain and by aviators, and the complex interactions between humans and the special environments created by the machines used in warfare. Our warfighters were exposed to mountain terrain during World War II, the Korean War, in military and humanitarian efforts in South America, and most recently in the Balkans. Military action has also occurred in some of the environments considered "special" (eg, on and below the water's surface) in every war that this country has fought, whereas other special environments (eg, air—flights not only within Earth's atmosphere but also beyond it, in space) have become settings for the havoc of war only as a result of 20th-century technology. The second volume also contains a discussion of the personal environment within the protective uniforms worn by service members against the fearsome hazards of chemical and biological warfare. This microenvironment—created by the very encapsulation that protects the wearer—is in some ways different from but in others similar to all closely confined, manmade environments (eg, the stresses that divers face in coping with the changes in barometric pressure). Whatever the environment, this point needs to be kept in mind: indifference to environmental conditions can contribute as much to defeat as the tactics of the enemy.

Medical Aspects of Harsh Environments, Volume 3, emphasizes the need for a preventive approach to decrease attrition due to harsh environments, such as predicting the likelihood of its occurrence and stimulating awareness of how specific factors (eg, gender, nutritional status) are sometimes important determinants of outcome. The third volume concludes with reproductions of two of the classics of environmental medicine: the lectures given by the late Colonel Tom Whayne on heat and cold injury, respectively, at the Army Medical School in 1951; for decades these have been unavailable except as mimeographed handouts to students attending specialized courses.

Military and civilian experts from the United States and other countries have participated as authors of chapters in this three-volume textbook, *Medical Aspects of Harsh Environments.* The textbook provides historical information, proper prevention and clinical treatment of the various environmental illnesses and injuries, and the performance consequences our warfighters face when exposed to environmental extremes of heat, cold, altitude, pressure, and acceleration. The contents are unique in that they present information on the physiology, physical derangements, psychology, and the consequent effects on military operations together in all these harsh environments. This information should be a valuable reference not only for the physicians and other healthcare providers who prepare our warfighters to fight in these environments but also for those who care for the casualties. Military medical personnel must never forget that harsh environments are great, silent, debilitating agents for military operations.

Lieutenant General James B. Peake
The Surgeon General
U.S. Army

Washington, DC
December 2001

Preface

On 1 July 1941, as part of Hitler's attack of the Soviet Union, the XXXVI Corps of the German army crossed the Finnish–Soviet border and began what was planned as a rapid advance some 50 miles to the east, where lay the strategically important railroad that linked the Arctic Ocean port of Murmansk with the Russian hinterland to the south. The German soldiers in their heavy woolen uniforms were greeted not only by determined Soviet resistance, but also by an unexpected enemy: the day was hot, with temperatures in the high 80s (°F), and there were swarms of ferocious mosquitoes. During the next 3 weeks the temperature rose above 85°F on 12 days and twice reached 97°F, and it was soon obvious that military operations were possible only in the relative cool of the "night." By the end of July, after advancing only 13 miles, the attack was called off, with the XXXVI Corps being denounced as "degenerate" by the German high command. Higher commanders obviously never considered that low combat effectiveness might result from the hazardous environmental factors: the heat, insects, and 24 hours of constant light. After all, who would have thought that heat stress might impair combat operations occurring 30 miles north of the Arctic Circle.[1]

The German experience in northern Finland was anything but unique; military history is full of examples where weather conditions influenced the outcome of military campaigns. In fact, the earliest recorded instance of weather's having a direct effect on the outcome of a battle dates back to the Old Testament:

> And it came to pass, as they fled from before Israel, and were in the going down to Beth-horon, that the Lord cast down great stones from heaven upon them unto Azekah, and they died: they were more which died with hailstones than they whom the children of Israel slew with the sword.[2]

The mission of the US Army Research Institute of Environmental Medicine, Natick, Massachusetts, is both to understand how soldiers react to military environmental and occupational stresses and to devise materiel and doctrinal solutions that are protective and therapeutic. The publication of the three volumes of *Medical Aspects of Harsh Environments* will ensure that both healthcare providers and military line commanders do not repeat the mistakes of countless commanders of the past who have underestimated the threats that harsh environments pose to their soldiers. I strongly recommend that all commanders and healthcare personnel become acquainted with the volumes of the *Textbooks of Military Medicine* dealing with harsh environments to better protect and preserve our sons and daughters during their deployments around the world.

The volumes of *Medical Aspects of Harsh Environments* became a reality because of the dedication and hard work of Kent B. Pandolf, PhD, and Robert E. Burr, MD, then a Lieutenant Colonel, Medical Corps, US Army, the specialty editors of this three-volume textbook. Dr. Burr was *primus inter pares* in the group that performed the critically important tasks of deciding on the subject matter and finding appropriate authors; when Dr. Burr left the Army, Dr. Pandolf brought the project to fruition. This first volume, which deals with hot and cold environments, owes its completion to the willingness of its section editors—C. Bruce Wenger, MD, PhD, and Robert S. Pozos, PhD—well-known experts in the fields of heat and cold stress, respectively, to perform the seemingly endless tasks necessary to assure the scientific accuracy of the text. In addition, the specialty and section editors wish to thank Rebecca Pincus for her invaluable help during this book's formation. The forthcoming second and third volumes deal with mountains and special operations environments, and sustaining health and performance during military operations. It is not too much to hope that the labors of the volumes' editors and many authors will lighten the burdens of our military personnel in the years to come.

Brigadier General Russ Zajtchuk
Medical Corps, US Army, Retired
Editor in Chief, *Textbooks of Military Medicine*

December 2001
Washington, DC

1. Ziemke EF. *The German Northern Theater of Operations 1940-1945*. Washington, DC: Headquarters, Department of the Army; 1959: 157–167. DA Pamphlet 20-271.
2. Joshua 10:11, *The Holy Bible,* King James version.

The current medical system to support the U.S. Army at war is a continuum from the forward line of troops through the continental United States; it serves as a primary source of trained replacements during the early stages of a major conflict. The system is designed to optimize the return to duty of the maximum number of trained combat soldiers at the lowest possible level. Far-forward stabilization helps to maintain the physiology of injured soldiers who are unlikely to return to duty and allows for their rapid evacuation from the battlefield without needless sacrifice of life or function.

MEDICAL ASPECTS OF HARSH ENVIRONMENTS
Volume 1
Section I: Hot Environments

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Rudolph Von Ripper *Filling Craters Near Pantellaria* 1943

Pantellaria (also spelled Pantelleria) is a tiny, rocky island in the Mediterranean Sea between Tunisia and Sicily, the occupation of which was considered essential for the planned invasion of Italy in the summer of 1943. Accordingly, the Allied air forces and the Royal Navy were called in to soften up the Italian defenders, a process they performed with great gusto:

> In one of the greatest examples of overkill of the war the air forces in three weeks dropped 6,400 tons of bombs … on Pantelleria.[1(p215)]

After the island had been captured and so that air facilities could be established in time for the invasion of Sicily, the vast multitude of craters left on Pantellaria by the bombs and shells had to be quickly filled by hard, manual labor in Sahara-like heat. Surviving records, however, do not indicate whether heat casualties were a significant problem.

Quotation: (1) D'Este C. *Bitter Victory: The Battle for Sicily, 1943.* New York, NY: Harper Perennial; 1991: 215. Painting: gouache on paper. Reproduced courtesy of Army Art Collection, US Army Center of Military History, Washington, DC.

Chapter 1

INTRODUCTION TO HEAT-RELATED PROBLEMS IN MILITARY OPERATIONS

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INTRODUCTION

As an introduction to the heat illness section of *Medical Aspects of Harsh Environments*, this chapter considers the effects of heat in the format of the classic epidemiological triad: the agents, the disease, and the host factors. First, the physical and physiological factors that are responsible for heat illness, especially in a military environment, are delineated. Second, insofar as possible, the physical and physiological factors that have affected military operations in the past are described, often by those who were directly involved.

Recognition that people can be killed by exposure to heat is documented in the earliest writings of man. The Bible reports the death of the young son of a farmer from exposure to the midday heat during the harvest in his father's fields (in about 1000 BC): he "went out to ... the reapers, said unto his father, 'my head, my head,'" and died in his mother's lap.[1] Sunstroke is specifically mentioned later in the Bible. Judith's husband, Manasseh, was out in the fields, supervising the binding of the barley sheaves: "he got sunstroke, and took to his bed and died."[2] The effects of heat on fighting men are also noted in the Bible: when the "sun stands still in the heavens," it helps the Hebrews fighting the more heavily armored Canaanites.[3]

The most critical time of year for heat stress was clearly identified millennia ago. By 3000 BC the appearance of the dog star, Sirius, representing the nose of the constellation Canis Major, was recognized as ushering in the "dog days" of summer. Sirius, described by ancient Egyptians, Greeks, and Romans as bringing on fever in men and madness in dogs, was introduced into medical literature as siriasis, the medical term for all types of heat illness well into the 20th century. In Homer's *Iliad* (ca 1100 BC), King Priam muses, while watching red-haired Achilles advance,

> blazing as the star which comes forth at harvest time, shining amid the host of stars in the darkness of the night, the star men call Orion's Dog. Brightest of all, but an evil sign, bringing much fever on hapless men.[4(pp401–402)]

Reports on the effects of heat on military operations have recurred repeatedly since then. King Sennacherib of Assyria had problems with heat while attacking Lashish, about 720 BC. In about 400 BC, Herodotus[5] provided one of the first reliable reports on the effects of heat on military operations. He describes the effects of the interaction among the load carried, protective clothing worn, and heat stress, when he states that both the Athenian attackers and Spartan defenders were worn out by "thirst and the sun," and when he reports on the discomfort of fighting in full armor under the summer sun, citing Dienekes the Spartan, at Thermopylai, in 480 BC, who, when told that the multitude of incoming Persian arrows would blot out the sun, calmly replied, "Then we might have our battle with them in the shade."[5(p551)]

In 332 BC, Alexander the Great's military advisers insisted that a march across 180 miles of the wind-blown Libyan Desert was too risky; if the army should use up its water supplies, they would experience great thirst for many days. At that time the camel, which could travel for 3 to 4 days without drinking while carrying a significant load of water for the troops, was already recognized as superior to horses, donkeys, or oxen, which had to drink several times a day. In any event, as Plutarch[6] suggested, the gods were extremely kind, providing plentiful rains, which relieved the fear of thirst and made the desert moist and firm to walk on. (According to studies[7] carried out at the US Army Research Institute of Environmental Medicine [USARIEM], Natick, Massachusetts, the latter characteristic may have cut the heat production of Alexander's soldiers by as much as 50%.) In the summer of 330 BC, while pursuing Persian King Darius after his crushing defeat at the Battle of Arbela, water supply became a problem as Alexander approached what is now the Turkmenistan border. When a foraging party finally returned to camp with water, Alexander, who was almost choked with thirst, again won the hearts of his troops when, offered a helmet of water (according to Plutarch), he

> took the helmet in his hands, and looking about, when he saw all who were near him looking earnestly after the drink, he returned it without tasting a drop of it. "For," said he, "if I alone drink, the rest will be out of heart."[8(p113)]

In midsummer 327 BC, Alexander split his 40,000 foot soldiers, sending one group through the Khyber Pass while leading the rest on a more difficult route into India. Troop movement was slow, and Alexander realized that the problem was not only the environment and terrain but also the heavy weight

of booty accompanying them. One dawn, after

> all the freight was loaded, Alexander set fire to his personal baggage wagon and then commanded that his soldiers' wagons be burned too.[8(p129)]

Thus unencumbered, the troops continued the march much more rapidly. But by 325 BC, his troops, demoralized by years of fighting away from home, were unwilling to continue.[9] Unable to inspire his troops to continue across the Hyphasis River, and after receiving a face-saving report from his soothsayers that the omens suggested that the gods did not want him to cross the river, Alexander started the march back. He started in September with about 20,000 in his entourage, many of whom were family members and camp followers. According to Plutarch, after 2 months of extreme heat, lack of water, and the trackless desert, only about 5,000 survived to reach the Persian palace at Pura, 200 miles from the southern border of what is now Iran. As has often been observed subsequently, the problem was not an effect of heat but the lack of water (and, in this case, of food also).

Three hundred years later, in 24 BC, a Roman legion led into Arabia by Aelius Gallus suffered a malady that

> proved to be unlike any of the common complaints, but attacked the head and caused it to become parched, killing forthwith most of those who were attacked.[10]

The Roman legionary used an early form of auxiliary cooling, inserting rushes into his headgear and keeping them wet with water. The Roman legions made extensive use of auxiliaries to carry as much of the legionnaires' load, and do as much of the engineering digging as possible, thus sparing the fighting edge of the legion. In the 1930s, during Italy's invasion of Eritrea, the descendants of the legionnaires (who were advised by Aldo Castellani, a leader in 20th-century military medicine) made similar use of auxiliaries.[11]

In the Middle Ages, the crusaders to the Holy Land had severe heat problems compared with the Saracens; the final battle of the Crusades was lost by heavily armored crusaders fighting under King Edward. The loss is usually attributed to an advantage of the native Arabs who, as a result of living in the heat, were, in theory, better acclimatized to heat than were the European crusaders. However, the 20th-century experience of oil companies hiring native workers in Bahrain, who suffered more heat casualties by far than their nonnative workers, appears to support the sentiment in Noël Coward's song that "mad dogs and Englishmen go out in the mid-day sun"[12(p163)]; that is, the Arabs benefited from having learned to avoid working in the heat rather than better heat acclimatization, which would have been acquired by gradually increased levels or durations of work, or both, in the heat. Modern understanding of the problems of military operations in the heat would suggest that the weight and impermeability of the protective armor worn by the crusaders were the primary problems. Active fighting, while wearing crusaders' armor, would have been stifling. Indeed, many combatants were reported to have been "suffocated."[13(p30)] When unhorsed, such knights had to be "cracked open and broken like lobsters"[13(p30)] to be dispatched. (The reference to lobsters perhaps applied equally to the skin color of the knights as to their shell-like casing.)

The potentially epidemic nature of heat illness was documented in Rome in 1694 by Baglivi.[14] In July 1743, 11,000 people died in Peking, China, during a 10-day heat wave.[15] In the 1800s, heat affected Spanish military operations in the New World; the Dutch suffered while taking the East Indies and the British while taking India; nevertheless, all these campaigns were successful. And during the 20th century, many heat deaths occurred among pilgrims making the Hajj to Mecca in years when it coincided with high temperatures.[16] However, although severe heat exposures can and have produced many casualties, reported losses in military effectiveness and lives are difficult to clearly separate between heat, per se, and other causes in reports made before the 20th century.

PROBLEMS OF DEFINITION AND COMPREHENSION

Terminology of Heat Effects

One of the problems with delineating the effects of heat on military operations from the literature before the 1950s, and even with some more recent reports, is that of terminology. The implied differential diagnosis of siriasis (ie, heatstroke) from sunstroke confounds many of the reports. In the 19th and early 20th centuries, siriasis was believed to be caused by the "actinic rays" of the sun and mandated wearing actinic orange underwear, a spine pad, and solar topee (a cork or pith helmet) to pre-

vent these rays from penetrating to the brain and spinal cord. Even today, heatstroke may not be properly differentiated from "physical heat exhaustion," "heat exhaustion collapse," or "hyperventilatory blackout" unless a patient dies—in which case it is almost always considered a heatstroke death, albeit it was not necessarily so. US military medical reports lumped all heat ailments under the term "sunstroke" during the American Civil War, as "heatstroke" during the Spanish–American War, as "effects of excessive heat" during World War I, and defined as three categories of heat illness during World War II: "ill-defined effects of heat," "heat exhaustion," and "heatstroke." Because deaths occurred in all three categories, it seems clear that a problem of definition existed.

Unfortunately, such problems of definition still exist. For example, during one of the many field studies that I helped design and conduct,[17] a 2:00 AM trip to a forward aid station was required to prevent a young medical officer from classifying militarily ineffective soldiers as "heat casualties" (a term used then in place of Leithead and Lind's "transient heat fatigue"[18(Table 30-6)]—despite ambient temperatures in the 10°C range. Tired, cold, dehydrated, and demoralized after many hours in chemical–biological protective clothing, yes; but not the usual "heat" casualties.

Even more difficult to assess than terminology is determining the consequences of long-term exposure in the tropics or, as Huxley is said to have defined heat acclimatization, "getting used to not getting used to the heat." In the 1920s, the Italian, Aldo Castellani, wrote on the importance of rotating white men from the tropics back to temperate climates to avoid an ill-defined syndrome, termed "heat fatigue." In the 1930s, Castellani was appointed the Chief Health Consultant in East Africa for the Italian Campaign in Ethiopia, where he introduced one of the first cohesive programs for avoidance and treatment of heat casualties.[11] In 1944, Douglas H. K. Lee evaluated troops of the Australian Army and Air Force for "tropical fatigue" and its detrimental effects on the health and performance of troops.[19] Hans Selye's formulation in 1949 of the concept of "stress" and the "generalized adaptation syndrome"[20(p837)] may be the best explanation for such vague, but real, malaise.

Epidemiology of Heat Illness

A second problem is the lack of understanding of the epidemiology; that is, the role played by various host and agent (environmental and operational) factors, and the nature of the diseases broadly termed "heat illnesses." Military operations are particularly likely to produce large numbers of heat problems, as discussed below. However, because of (*a*) the select nature of most military forces (ie, troops are usually young, fit, well conditioned, and at least partially heat acclimatized as a result of their physical conditioning); and (*b*) generally good, informed command and control by the military leadership, death due to heatstroke tends to occur as an isolated case. In the Israeli Armed Forces, for example, a heatstroke death is considered a failure of command control. In my own experience with heat death in civilian workers, when heatstroke is not a direct result of supervisory failure, it may be associated with increased susceptibility of the individual as a result of

- dehydration, often as a sequela of high alcohol consumption;
- febrile onset as a result of infection or recent immunizations; or
- loss of physical condition or acclimatization, or both, as a result of extended absence from the job, whether from illness or vacation.

It has been suggested (but not in the open literature) that individuals with low innate fitness (eg, those with < 2 L/min of maximum oxygen uptake [as a result of small cardiac stroke volume due to either small heart size or low maximum heart rate, usually a concomitant to aging]), have unique susceptibility to all forms of heat illness.

THE SIX "AGENTS" OF HEAT EFFECTS

Air temperature per se is seldom the cause of heat problems; it is only one, and rarely the most important, of the six factors—or, in terms of the epidemiological triad, the "agents"—that result in heat stress as it affects military operations. Four of these six are environmental factors:

1. ambient air temperature (T_a);
2. air motion, or wind velocity (WV);
3. air relative humidity (rh), expressed more relevantly as the vapor pressure of the moisture in air (P_a); and
4. mean radiant temperature (MRT).

The details of their measurement and calculation are outside the purview of this chapter, but interested readers can consult the chapter by Santee and Matthew, Evaluation of Environmental Factors, in the third volume of *Medical Aspects of Harsh Environments,* and other textbooks[21] that discuss the subject.

In considering the effects of heat on military operations, rarely are any of these four environmental factors as important as two behavioral factors:

5. the amount of metabolic heat (M) produced by the body; and
6. the clothing worn, and its insulation (clo)[22] and moisture permeability (I_m),[23] and how these change with wind or body motion or both (as characterized by a "pumping" coefficient).

These behavioral factors can be considered *agent* rather than *host* factors because they tend to be established by the operation rather than by the individual, particularly in a military setting. Thus, any consideration of thermal stress should explore these six key factors.

Environmental and Behavioral Tradeoffs

Tradeoffs have been established between these six factors with respect to their effects on human comfort. It is useful to examine the tradeoffs among these for comfort, and then infer from them the effects of five (in relation to the sixth, air temperature), as epidemiological "agents" for heat illness:

1. rh: a 10% change can be offset by a 0.5°F change in T_a;
2. WV: a change in wind speed of 20 feet per minute (fpm) is equivalent to a change of 1°F (up to a maximum of 5°F) in T_a;
3. MRT: a change of 1°F can be offset by a 1°F change in T_a;
4. clo: a change of 0.1 clo has the effect of a 1°F change in T_a at up to 2.5 met (the unit of measure for metabolic rate) of activity, and 2°F at higher levels; and
5. M: an increase of 25 kcal/h is equivalent to a 3°F increase in T_a.

The normal comfort range[24] is a 6°F-wide band of air temperature between 72°F and 78°F when the following conditions are met for the other five agents:

- rh is 40%;
- WV is 44 fpm (ie, 0.5 mph);
- MRT = T_a;
- clo = 0.6; and
- M = 1 met.

The clo unit of clothing insulation was defined so that an average man with 1.8 m^2 of body surface area must transfer 10 kcal of heat per hour (by radiation and convection) per Centigrade degree difference between the air temperature (T_a) and skin temperature (T_{sk}), typically 35°C when warm. A long-sleeved shirt and trousers provides 0.6 clo; the surface air layer next to the body adds another 0.8 clo, to bring the total insulation to 1.4 clo units. Thus, the total insulation that limits heat loss, without sweat evaporation, is about 1.4 clo for a soldier who is wearing a fatigue ensemble; this increases to about 2.5 clo when a chemical protective ensemble (which also usually offers increased resistance to sweat evaporative cooling) is worn. As a result, maximum nonevaporative heat loss is about 72 kcal/h at 25°C with 1.4 clo [ie, (35°C – 25°C) • 10/1.4], but only 40 kcal/h with 2.5 clo. Note that at rest, producing 90 kcal/h, about 25% of M (~ 22 kcal/h) is lost by respiration and evaporation of the normal, nonsweating, moisture diffusing from the skin, so that heat balance (ie, heat production [90 kcal/h] $\simeq$ heat loss [72 + 22 kcal/h]) is achieved without sweating, and an individual is comfortable at 25°C (77°F) with 1.4 clo of total clothing and still air insulation. This helps explain why the comfort range for office workers is 72°F to 78°F, while soldiers can get heat illness at these same temperatures.

Metabolic rate (M) is expressed in mets. One met, the resting heat production, is defined as 50 kcal/h per square meter of body surface area; or, for an average 1.8 m^2 man, 90 kcal/h, which equals 105 watts (W). M increases with the pace of military operations, and load carried, to levels of as much as 10 met, which can only be sustained for a short time (~ 15 min). The metabolic demand of marching at 3.25 mph on a blacktop road can be estimated as 2 kcal/h per pound of body weight plus load weight (eg, a 165-lb infantryman carrying a 60-lb load [clothing, weapon, pack, etc] will produce ~ 450 kcal/h, or 5 met).[25] Marching over sand will more than double this heat production,[7] to the 10-met level, which will rapidly result in physical exhaustion.

To better understand why military operations are so susceptible to being affected by heat, we can estimate the comfort temperature range for combat

EXHIBIT 1-1

TRADEOFF ANALYSIS

Soldiers performing military operations are vulnerable to the effects of heat. The tradeoff analysis—a stepwise arithmetical process—demonstrates how the cumulative effects of working in the sun in uniform can change an exposure from benign to unbearably hot:

1.		20.0°C (68°F) T_a
2.	+	0.5°C (1°F) for the extra 20% rh
3.	+	7.0°C (13°F) for the MRT effect
4.	–	2.8°C (5°F) the maximum "wind" benefit
5.	+	6.7°C (12°F) for the extra 0.6 clo (5 met)
6.	+	24.0°C (43°F) for the extra 360 kcal/h
	=	55.4°C (132°F) equivalent air temperature

clo: unit of clothing insulation; MRT: mean radiant temperature; rh: relative humidity; T_a: ambient temperature; met: unit of measure for resting metabolic rate

infantrymen on an approach march (ie, 5 met, or 450 kcal/h) with a total of 2.0 clo of insulation (helmet, body armor, battle dress uniform, pack, etc). In an otherwise comfortable environment, with T_a of 20°C (68°F) and 60% rh, with high wind and an increase in MRT of 7°C (13°F, which is typical for full sun exposure), the tradeoff analysis gives an equivalent air temperature of 55.4°C (132°F) (Exhibit 1-1).

Exacerbating Aspects of Military Operations

During military operations these six "agents" (air temperature, wind velocity, relative humidity, mean radiant temperature, metabolic heat production, and clothing insulation) that contribute to heat illness can, in turn, be exacerbated by the occurrence of unusually extreme environmental exposures, and by extremes of heat production and clothing; the former can seldom be avoided, and the latter are an inescapable component of battle.

Environmental Extremes

The civilian population is seldom exposed to extremes of any of the four environmental factors introduced above. Avoidance of extremes of heat is ingrained in semitropical and tropical cultures, and embedded in such traditions as the afternoon *siesta.* However, worldwide weather extremes are much less severe than those that can occur inside crew compartments in military armored fighting vehicles (Table 1-1), where the average increase in interior temperature above the ambient outside is 13 Fahrenheit degrees, with peak increases exceeding 26 Fahrenheit degrees. In addition, whereas the highest ambient air temperatures (eg, > 100°F) are never accompanied by high humidity, the even higher air temperatures in crew compartments can be accompanied by quite high humidity as occupants' evaporated sweat accumulates. Finally, the fully saturated (100% rh), trapped air next to the skin of soldiers encapsulated in heavy clothing with reduced moisture permeability, or in light but impermeable protective clothing, routinely produces heat casualties.[26] (The ratio of the index of permeability to moisture to the insulation provided by clothing [I_m/clo] is discussed later in this chapter.) Heat casualties should be anticipated in less than 1 hour at ambient air temperatures above 30°C (86°F) even at low activity levels and, given the introduction of reduced permeability membranes in combination with the high insulation of the latest Extended Cold Weather Clothing System (ECWCS), possibly with only a few hours' heavy work even at –30°C (–22°F).

Heat Production Extremes

Many aspects of the military setting result in sustained periods of extremely high heat production. First, the pace of work is seldom set by the individual, and often not even by his immediate unit leaders, but by a remote commander or by the enemy. The well-known military tradition of "hurry up and wait" is a natural consequence. The problem was stated by John Pringle, Surgeon General to the English army, in 1752:

> The life of a foot soldier is divided between two extremes of labour and inactivity. Sometimes he is ready to sink beneath fatigue, when, having his arms, accoutrements and knapsack to carry, he is obliged to make long marches, especially in hot or rainy weather.[27]

Second, almost all individuals taken into the military are given extensive training to bring them to peak ability to perform heavy work, despite some evidence that most troops are seldom exposed to anything like the sustained high work levels experienced during their training. This very demanding training, and an initial lack of acclimation or acclimatization to work in the heat, accounts in part for the high incidence of heat casualties suffered by

TABLE 1-1

INTERIOR TEMPERATURES* IN ARMORED FIGHTING VEHICLES IN HOT–DRY ENVIRONMENTS

Vehicle	Location	Ambient Temp (°F)	Interior Temperature, Degrees Above Ambient	
			Average (°F)	Maximum (°F)
M114 ARV	Driver compartment	91–114	13	29
	Crew compartment		14	30
M50 APC	Crew compartment	100	—	40
M113 APC	Crew compartment	105–108	10	14
M106AI SPM	Crew compartment	90–102	21	25
M557 CPC with CPV	Crew compartment	102	—	37
	Crew compartment	90–100	20	34
	Driver compartment		21	35
M701 MVCV	Turret	100	17	19
	Squad compartment		17	21
	Driver compartment		17	19
LVTPX 12	Driver compartment	95–109	6	14
	Commander's compartment		10	20
	Cargo compartment		12	25
T98EI SPH	Turret	94–107	18	53
T19EI SPH	Turret	97–101	23	30
M109 SPH	Crew compartment	99–111	11	16
	Driver compartment		14	20
MSSI AR/AAV	Turret	104–110	13	25
	Driver compartment		7	12
M41M Tank	Turret	94–111	5	11
M48 Tank	Turret	95–107	7	13
T45 Tank	Turret	96–107	6	9
M 60 Al Tank	Turret	90–113	9	20
M43EI Tank	Turret	93–107	10	18
	Turret bustle	99–102	55	57
Ambient Temperature Range:		90–114		
Approximate Average Temperature Above Ambient:			13.1	23.7

*Add + 10°F for positive pressure (few of these vehicles could be overpressurized but the limited data on overpressurized vehicles [the USSR approach] suggests adding 10°F to interior temperatures)

recruits during military training. (The term "acclimatization" pertains to the physiological adjustment to an environment in nature, whereas "acclimation" pertains to physiological adjustment to environmental conditions in a controlled setting.) Of greater importance in the overall issue of heat stress during military operations is the unreasonably heavy loads carried by combat troops. Mules may have developed their reputation for stubbornness as a result of their adamant refusal to move when overloaded. Troops are less resistant to excessive loads and, despite peacetime loads that

approximate the 45 pounds recommended by a British Royal Commission in 1847, often carry almost twice that weight into combat. Each pound (whether of body or load) moved when walking at a normal pace (about 3.25 mph) requires a heat production of 2 kcal/h, and even more as load weights exceed about one third of body weight, or as speed increases. The effects of such heavy loads in exhausting the soldier and inducing casualties have been a focus of attention since Roman times. Lothian[12] reviewed the soldier's load from the classical Greek hoplite era through 1918; I have expanded Lothian's chart up to the Vietnam era (Figure 1-1). S. L. A. Marshall, in his essay "The Soldier's Load and the Mobility of the Nation,"[28] provides an extensive critique of the effects of the excessive loads imposed on a soldier. Indeed, tables describing the energy costs of various tasks characterize identical caloric costs for a given task differently for the military population than for a civilian work force (eg, a task rated at the same number of calories will be characterized as "light" for a military population, but "moderately hard" for civilian workers who usually are older, generally less fit, and often include workers who would have been screened out by any military selection process). Finally, Kennedy and Goldman[29] in a report on the design of load carriage equipment, suggest that load carriage capacity, and even uniform pockets, should be minimized to reduce the traditional accumulation of trophies (or loot) common to the combat soldier since earliest history.

Clothing Extremes

As the effectiveness of weapons increased there was a natural desire to provide increasing protection against them, insofar as possible. An example of this is the blue paint daubed on Zulu warriors by their witch doctors to repel the British bullets. At least this psychological prop did not degrade the performance capabilities of the wearer, unlike most items of modern protective clothing and equipment. Concerns for heat stress in four groups of fighters have formed a major portion of this writer's career: the military, firefighters, hazardous-waste site workers, and football players. Football players are at increased risk of heat illness because of the impermeable plastic protective shoulder padding and helmet worn, in combination with their high, albeit intermittent, levels of heat production. Hazardous-waste site workers are at risk because of the impermeability ($I_m < 0.12$) of their thin (clo ~ 1.0), encapsulating, protective ensembles. Firemen typically wear about 3 clo of insulation, with varying degrees of reduced permeability from water-repellent treatments or "breathable" waterproof membranes, or both, which have an I_m of less than 0.3. The resulting approximately 0.1 I_m/clo, coupled with high radiant or ambient temperatures, or both, and short periods of very heavy exertion, result in significant heat stress problems.

Military personnel body armor is essentially impermeable as well as highly insulating. It adds enough weight and impedance of movement that, it has been argued,[30] it slows the wearer down and makes him an easier target. It has also been argued[30] that the increase in protection afforded military personnel and firefighters leads both groups to take greater risks than they otherwise would. Finally, nuclear, biological, and chemical (NBC) protective clothing, which was developed initially during World War II, was resurrected after reports during the late 1950s that facilities were being constructed at Russian airfields for the storage of chemical munitions. This led to 25 years (1959–1984) of intense research and development focus on the effects of wearing chemical protective clothing (Table 1-2), on development of more permeable chemical protective clothing, on heat stress and tolerance time limitations for military performance, and on models to predict them.[31]

Recognition that troops on today's battlefields wearing body armor and chemical protective clothing have limits on load carriage and sweat evaporative cooling similar to those of the armored crusaders of the Middle Ages has led to development programs for advanced combat infantry clothing (Generation II and 21st Century Land Warrior [21CLAW]). However, further development of the auxiliary cooling systems included in earlier programs (eg, the Soldier Integrated Protective Ensemble [SIPE]) has been rejected as impractical. At the same time, the most recent versions of the battle dress overgarment (BDO) provide about 20% less potential for sweat evaporative cooling than earlier versions (eg, I_m/clo now = 0.12, vs 0.15). Thus we can expect questions to continue on the effects of heat on military operations.

The sources of heat that most seriously affect military operations are (1) a sustained, high metabolic heat production; and (2) high temperature and humidity, particularly in crew compartments of armored fighting vehicles, where the interior temperatures average 7 Centigrade degrees above ambient and can be as much as 17 Centigrade degrees higher. These are complicated by (3) difficulty in losing this heat through the heavier insulation of protective clothing. Of these, the third, that of pro-

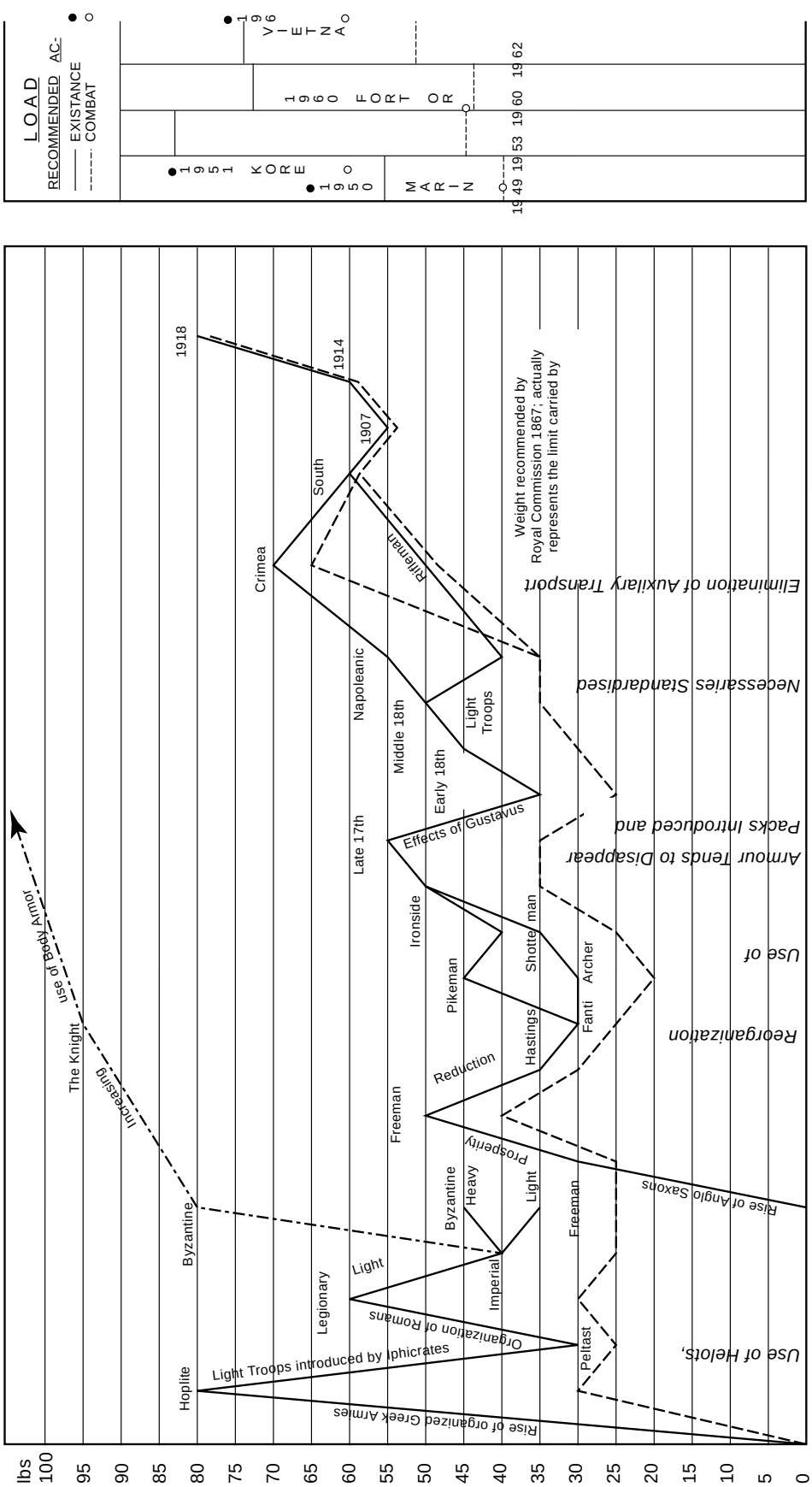

Fig. 1-1. The soldier's load: estimated, recommended, and actual. This chart, presented by N. W. Lothian in 1922, was updated by R. F. Goldman in 1969 to include data for the late 1940s through the Korean War in 1951; the subsequent, noncombat Cold War; and the Vietnam War in 1967. The chart shows that despite recognition of the adverse effects of heavy load carriage on military operations as early as the introduction of the armor of the Greek hoplite,[1] and the recommendation of a British Royal Commission in 1867[2] that 45 lb represented the limit carried by average troops without distress, the cycle of increasing the soldier's load in wartime—until it becomes clear that he is at a severe disadvantage against more lightly loaded opponents—has continued over the last two millennia and seems likely to continue into the third. The *combat* load, close to the 45-lb recommendation in peacetime, and the *existence* load (ie, tentage, sleeping bag, poncho, etc, carried on a march), which hovers around 65 lb in peacetime, both increase dramatically during wartime, to the soldier's detriment.[3] According to the US Army Research Institute of Environmental Medicine, the current nomenclature and recommended weights for soldiers are as follows: fighting load, 41.5 lb; approach load, 67.5 lb; and sustainment load, 97.5 lb.[4] Sources: (1) Xenophon. *Anabasis*. 3.4.48. (2) British Royal Commission. *The Influence of Accoutrements on Health*. Cited by: Lothian NW. The load carried by the soldier. *J Roy Army Med Corps*. 1921;37:241–263, 324–351, 448–458, and 1922;38:9–24. Distributed by: Washington, DC: Office of The Quartermaster General, Research and Development Branch, Textile, Clothing and Footwear Division. Tentage and Equipage Series Report 11. Released for public information by The Office of Technical Services, US Department of Commerce; 1954: 7. (3) Marshall SLA. *The Soldier's Load and the Mobility of a Nation*. Washington, DC: Combat Forces Press; 1950. (4) Pandolf KB. Senior Research Scientist, US Army Research Institute of Environmental Medicine, Natick, Mass. Personal communication, 11 May 2000. Chart: Adapted with permission from Goldman RF. Physiological costs of body armor. *Mil Med*. 1969;134(3):209.

TABLE 1-2

SELECTED CHRONOLOGY OF TESTING NUCLEAR, BIOLOGICAL, AND CHEMICAL PROTECTIVE CLOTHING

Year	Study or Event	Site or Testing Organization
1915	First use of gas, Germans	
1918	AEF #1433: Defense Against Gas. *Troops Need Practice Wearing Respirator for Longer Periods*	
1959	Evaluation of CB protection during the Cold War	Camp Pickett, Va; Fort Knox, Ky; EPRD*
1959	Protect I	Fort McClellan, Ala; Dugway Proving Ground, Ut
1960	Copper Man Studies of NBC Clothing	USARIEM†
1961	Climatic Chamber Studies	USARIEM†
1961	Effects of Hycar Underwear on Heat Stress	Edgewood Arsenal, Md
1961	Responses Wearing Protective Clothing in Hot-Dry	Dugway Proving Ground, Utah
1962	Fort Lee Field Studies	USARIEM†
1962	Project Samples-Mask Studies	Fort McClellan, Ala
1962	Jackpot	Fort McClellan, Ala
1963	Road Operations in a Toxic Environment (Panama)	USARIEM†
1963	Samples	Fort McClellan, Ala
1964	Road Operations in a Toxic Environment	Fort Ord, Calif; CDEC*
1965	IPR CB Protective Overgarment	NARADCOM*
1966	Mandrake Root (Computer Study)	MUCOM; OPRESG*
1967	Mandrake Root Addendum Study	United States, USSR*
1967	Mission Degradation	MUCOM*
1966–1968	Effectiveness in a Toxic Environment–METOXE	CDC; CAG (many sites)*
1969	US Amphibious Assault 69-10	NMFRL*
1969	Doctrinal Guidance for NBC Wear	USARIEM†
1970	Copper Man Evaluations	USARIEM
1971	Gum Tree	United Kingdom, Malaysia
1971	Chillitog	United Kingdom, NW Europe
1971	Reducing heat stress in NBC ensembles	TTCP/Edgewood*
1971	DCGE/DREO 2/71	Canada
1972	Jeremiah	United Kingdom (done in Suffield, Canada)
1975	Grand Plot III	CDCEC; IDF*
1975	US/CDA/UK Companion Study	Dugway Proving Ground, Ut*
1976	Unit Chem. Defense (SCORES-MIDEAST)	TRADOC, Fort Monroe, Va*
1977	USAF Chem. Defense	TAC OPS Eglin Air Force Base, Fla*
1977	Ill Wind (CPX)	Fort Benning, Ga
1978	Wetted cover to reduce heat stress in NBC	USARIEM†
1978	XM-29AH and the AH-IS Sight	Fort A. P. Hill, Va*
1979–1980	Reducing Heat Stress in a CB Environment	TRADOC, Fort Monroe, Va; Field Manual 21-40
1979	Performance Degradation Modeling (PDGRAM)	AMAF Industries*
1980	Heat Stress for XM-I CVC in CW Protection.	USARIEM†
1980	Heat Stress in USN Carrier Flight Operations	USARIEM†
1980	Early Call III	APRE, Aldershot, United Kingdom
1980	Australian Infantry Performance in NBC	Australia*
1980	Thermal Stress in M-I Tank With NBC	APG, USARIEM†
1980–1982	Rapid Runway Repair (R3) in NBC	US Air Force*
1981	Mobility Through Contaminated Areas (MOCAT)	CDCEC, Fort Ord, Calif*
1981	CW Protective Posture Performance (CWP3)	CDCEC, Fort Ord, Calif*
1981	Auxiliary Cooling and Tank Crew Performance	USARIEM†
1981–1982	Forward Area Refuel/Rearm Performance (FARP)	AAMRL, Brooks Air Force Base, Tex*
1982	Thermal Stress and Flight Performance	US Air Force; AMRL*
1983	Tank Crew Performance with Auxiliary Cooling	USARIEM; APG†
1983	Heat Stress and Performance in Nuclear Reactor Repair	GPUN/TMI-2*†
1983–1984	Cane I	CDCEC*

*Author involved as consultant or collaborator
†Author ran the study
AAMRL: Army Aviation Medical Research Laboratory, Dothan, Ala
AEF: Army Expeditionary Force
AMAF: American Machine and Foundry (contractor)
AMRL: Armored Medical Research Laboratory, Fort Knox, Ky
APG: Aberdeen Proving Ground, Aberdeen, Md
APRE: (Royal) Army Personnel Research Establishment
CDC/CAG: Combat Development Command, Combined Arms Group
CDCEC: Combat Development Command Experimentation Center, Fort Ord, Calif
CDCEC.IDF: Combat Development Command Experimentation Center and Israeli Defence Force (at Fort Ord)
EPRD: Environmental Protection Research Division, Natick, Mass
GPUN/TMI-2: General Public Utilities Nuclear, Three Mile Island Facility
MUCOM/OPRESG: Munitions Command, Operations Research Group
NARADCOM: US Army Natick Research and Development Command
NBC: Nuclear, Biological, and Chemical
NMFRL: US Navy Medical Field Research Laboratory, Camp Le Jeune, NC
TAC OPS: tactical operations
TRADOC: Training and Doctrine command, Fort Monroe, La
TTCP: Tripartite Technical Cooperation Program (US, UK, Canada)
USARIEM: US Army Research Institute of Environmental Medicine, Natick, Mass

tective clothing insulation as a limit to cooling, is the most insidious. The potential for soldiers wear protective clothing that is heavily insulated or relatively impermeable, or both, to become overheated is further compounded by such clothing's limitations on sweat evaporative cooling. This limitation results not only from the reduced permeability to moisture (I_m) of any clothing (even for ordinary civilian clothing, I_m values are ~ 0.45) but also from the need for the evaporating sweat to pass through the insulation. The actual fraction of maximum evaporative cooling a wearer might be able to obtain in any given environment is determined by the ratio, I_m/clo. For example, with multilayered protective ensembles, such as cold weather clothing, which range up to 3 clo, even if moisture permeability is not reduced by waterproof materials or water-repellent treatments, the I_m/clo ratio of 0.45/3 means that the wearer can get only 15% of the maximum evaporative cooling power of the environment. And the insulation of the US Army's arctic clothing can reach 4 clo.

HEAT ILLNESS: THE "DISEASE" SPECTRUM

During World War II, considerable effort was spent in trying to categorize various forms of heat illness. A more modern approach is to consider heat illness not as a single disease but as a continuum of accumulating effects of heat, with specific disease entities defined as specific organs or systems are affected. In particular, I have found it possible to differentiate "heat exhaustion collapse" (ie, a soldier, temporarily unconscious, falls to the ground until the blood, not having to fight gravity, can again flow to the brain) from "physical heat exhaustion," in which a soldier remains standing but is "obtunded" (ie, unable to respond to a direct order, unaware of what is going on around him or where he is, albeit still trying to keep moving). In the latter condition, troops have been known to walk into vehicles, off ledges, and the like.

Both heat exhaustion collapse and physical heat exhaustion (previously termed "transient heat fatigue"[18]) (Table 1-3) can be viewed, simplistically, as an inability of the body to

- maintain sufficient cardiac output (blood flow) to deliver oxygen to the brain, muscles, and other body tissues;
- remove heat and products of metabolism from the tissues; and
- transfer heat to the skin so that it can be lost to the environment, if allowed by clothing, ambient temperature and vapor pressure, and air motion.

A useful basis for understanding why heat accumulates in the body, producing this continuum of heat illness, is suggested by three simple equations, which show that cardiac output is the most important determinant, with removal of tissue heat and metabolic by-products and heat transfer from skin to environment both secondary. These avoid any medical differential diagnosis but characterize, simplistically, the physiological basis for the categories of heat illness as a disease. The first equation states that cardiac output of blood (ie, the volume of blood [L/min] pumped by the heart), at rest or work in any environment, is simply a function of heart rate and stroke volume:

(1) Cardiac output (C.O.) = heart rate • stroke volume

The maximum heart rate (ie, the maximum number of beats per minute) is primarily a function of age (220 beats per minute minus the subject's age in years). The stroke volume (ie, the amount of blood pumped per beat) is a function of the size of the heart (primarily set by genetic inheritance, but heart size can be somewhat increased by physical conditioning), assuming that the volume of blood returned from the body to the heart is sufficient to fill it in the time between beats.

The second equation states that the volume of oxygen ($\dot{V}O_2$, in L/min) supplied to the various body organs (brain, muscles, heart, etc) is simply the product of cardiac output multiplied by the difference between the oxygen concentration in the arterial blood (Cao_2), as it is circulated to these organs, and the oxygen in the venous blood (Cvo_2) as it leaves them:

(2) $\dot{V}O_2$ delivery to organs = C.O. • ($Cao_2 - Cvo_2$)

The third equation states that the maximum amount of heat (in kcal/min) that can be transferred from the body's core (ie, muscles and other organs) to the skin is a function of the cardiac output multiplied by the difference between the temperature of the body's core (T_c), which is the primary site of heat production during work, and the temperature of the skin (T_{sk}):

(3) Maximum heat delivery to skin = skin blood flow • ($T_c - T_{sk}$)

Almost all the heat produced by the body must be

TABLE 1-3

HEAT STRESS

Heat Stress Disorder	Cause and Problem	Signs and Symptoms	Treatment
Heat Cramps	Failure to replace salt lost through sweating Electrolyte and muscle problems	Painful muscle cramps	Drink lightly salted water, lemonade, tomato juice, or "athletic" drinks
Hyperventilation	Overbreathing Low blood CO_2 level problem	Dizziness; tingling around lips; carpopedal spasm; blackout	Slow, deep rebreathing from paper bag
Heat Exhaustion	Excessive heat strain with inadequate water intake Cardiovascular problem (inadequate venous return, filling time) Orthostatic hypotension problem	Weakness, unstable gait, extreme fatigue; wet, clammy skin; headache, nausea, collapse	Rest in shade and drink lightly salted fluids
Dehydration/Physical Exhaustion	Failure to replace water loss Excessive work in heat problem	Excessive fatigue; weight loss	Replace fluids; rest until weight and water losses are restored
Heatstroke	High T_c, typically > 105°F Damage to or dysfunction of multiple organ systems is frequent	Mental status changes, including irrational behavior or delirium; loss of consciousness, convulsions, and/or shivering may occur	Rapid, immediate cooling by cold-water immersion, or wrap in wet sheets and fan vigorously. Continue until T_c is < 102°F. Treat for shock if necessary once temperature is lowered. *Heat stroke is a medical emergency. Brain damage and death can result even if treatment is timely.*

T_c: core body temperature

delivered to the skin before it can be lost, through any clothing, to the environment by convective, radiant, or evaporative heat transfer, separately or in combination.

These three equations suggest that there is competition for cardiac output between (*a*) the need to transport oxygen to the muscles (and other organs in the body's core) and the brain and (*b*) the need to transport heat from the body's core to the skin. (This competition is discussed in greater detail in Chapter 2, Human Adaptation to Hot Environments.) The first line of defense for the body against heat stress is to increase blood flow to the skin, thus raising skin temperature. The second line of defense is to increase the "wetness" of the skin by initiating sweating as skin temperature reaches 35°C, and increasing the amount of sweat produced as the body's requirement for evaporative cooling increases. Bear in mind, however, that the interpretation of these equations needs to be tempered by a consideration of the following factors: (*a*) although the sustainable sweat rate is only about 1 L/h, under severe heat stress up to 3 L/h can be produced; (*b*) at least initially, almost all the sweat produced represents fluid drawn from the circulating blood volume; (*c*) the average adult has only about 5 liters of blood; and (*d*) when we are inactive and hot, blood tends to pool in the skin. Obviously, the com-

EXHIBIT 1-2

OVERHEATING IN HUMANS AND AUTOMOBILES

Humans, like automobiles, overheat faster when

- the air temperature is close to the radiator (skin) temperature
- fluid, trapped in the radiator (skin), is not circulating
- the radiator (skin) is covered with dirt (clothing)
- radiator fluid (blood volume) is low
- too little fluid returns to the pump (heart), or the pump is too small, too weak, or leaks
- the radiator (skin) is partially blocked (skin eruptions)
- they start with warmer radiator (body) temperature

Humans, unlike automobiles, also heat faster when

- clothing is impermeable, heavy, or both (ie, low I_m/clo)
- environmental vapor pressure is near 44 mm Hg
- the body's control of blood flow or sweating is impaired by alcohol, drugs, disease, or inoculations to prevent it
- sweating is impaired by skin eruptions

petition for cardiac output (implied by the three equations above) is increased, particularly if water intake is less than sweat loss.

A fourth equation,

(4) Maximum heat loss from the skin
$= [10/clo \bullet (36 - T_a)] + [22 \bullet I_m/clo \bullet (44 - P_a)]$

establishes the maximum heat transfer (kcal/h) at any environmental or ambient air temperature (T_a) and vapor pressure (VP) of the moisture in air (P_a) from a warm (36°C), fully sweating (VP = 44 mm Hg) skin to the environment through (*a*) clothing insulation (in clo units) as radiative and convective heat loss, and (*b*) the clothing's resistance to evaporative heat loss. (Clothing resistance to evaporative heat loss is expressed by the relation clo/I_m, the inverse of the evaporative potential, I_m/clo.)

Overheating in humans can be compared to overheating in automobiles, although in humans the process is more complex (Exhibit 1-2). These simple analogies should help the reader understand the causes of the "diseases of heat" that affect military operations, and the interactions that make a continuum of these disorders. Figure 1-2 summarizes the heat illnesses and their etiology[32] and adds less common heat ailments, such as tetany associated with hyperventilation (panting).

"HOST" FACTORS IN HEAT ILLNESS

Unlike studies in laboratory animals in which there is little inbred variability, studies of the human responses to heat show large differences between individuals. As discussed above, host factors of concern include heart size, physical fitness, skin eruptions, initial body temperature elevation from anxiety, fever (or prefebrile state) associated with many diseases (or inoculations against them), and dehydration.

Heart Size

As indicated above, small heart size implies lower cardiac output; this results in lower work capacity (ie, maximal oxygen uptake, $\dot{V}O_2max$) and greater problems under heat stress. Indeed, it has been suggested[33] that individuals with a $\dot{V}O_2max < 2.5$ L/min cannot perform hard work in the heat.

Physical Fitness

Lack of physical fitness, whether a result of genetics (inadequate selection pool or criterion for military recruits) or lack of adequate training (intensity, duration, and frequency of exercise), cardiac or respiratory disease, and so forth, will reduce heat tol-

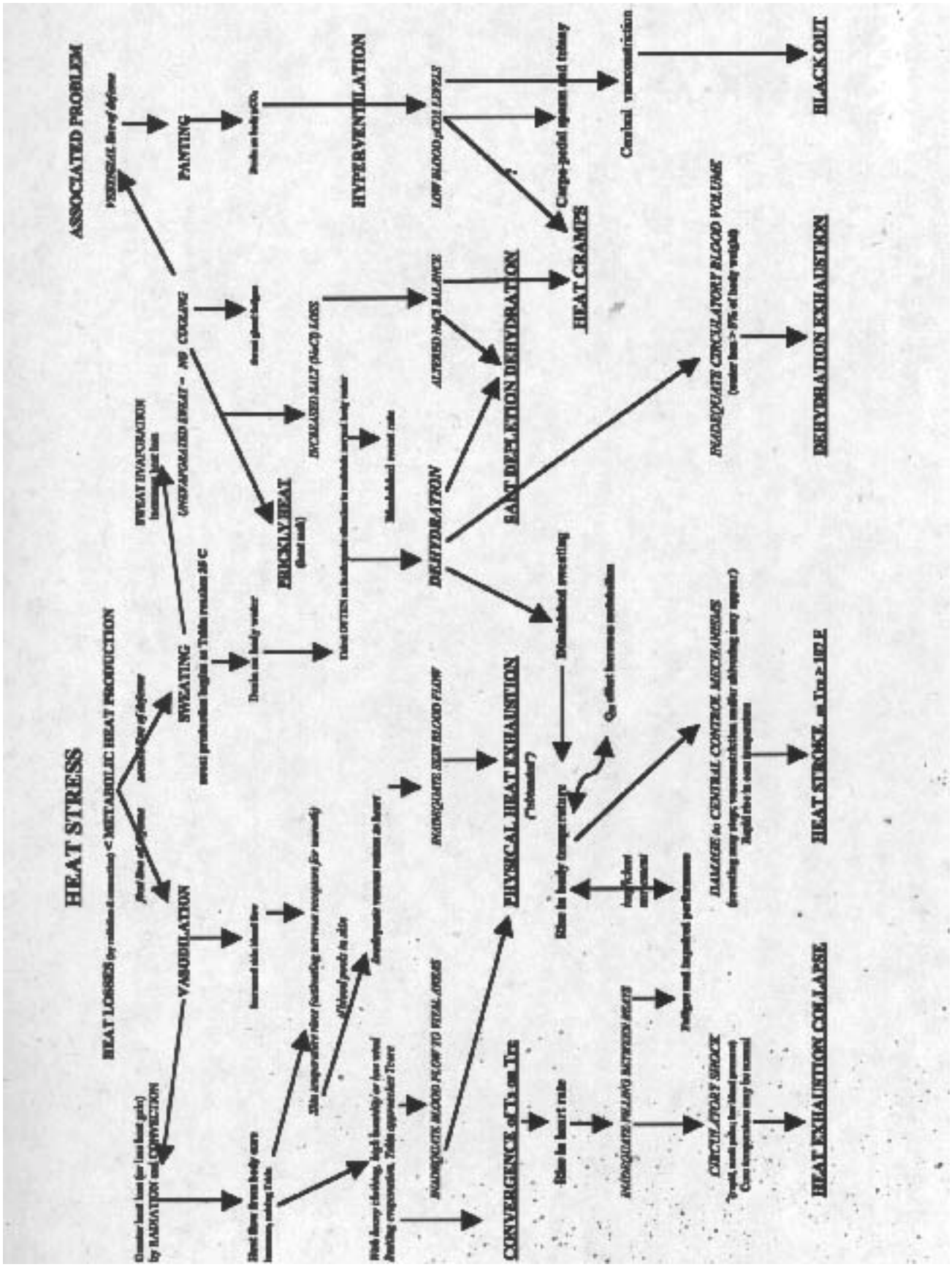
HEAT STRESS
HEAT LOSSES
METABOLIC HEAT PRODUCTION
SWEATING
VASODILATION
PANTING
HYPERVENTILATION
ASSOCIATED PROBLEM
HEAT CRAMPS
DEHYDRATION
SALT DEPLETION DEHYDRATION
PHYSICAL HEAT EXHAUSTION
HEAT EXHAUSTION COLLAPSE
HEAT STROKE
DEHYDRATION EXHAUSTION
BLACK OUT

Fig. 1-2. Heat stress and its associated physiological responses and pathologies. If troops cannot get rid of the heat that they produce, then their body temperature must rise. As the individual becomes hotter, the body has only two mechanisms—vasodilation and sweating—by which to rid itself of the extra heat. The heart beats faster, pumping more blood from the muscles to the skin, to help get rid of the heat; that will not work if the skin cannot lose the heat through the clothing to the environment. The skin can also increase the amount of sweat it releases, putting out as much as 2 to 3 L of sweat per hour, at least for the hour or so before a trooper suffers heat exhaustion or dehydration exhaustion (ie, the loss of more water from the body than is replaced by drinking). Sweat also contains salts, and if too much salt is lost from the body, a variety of heat illnesses result.[1] Although these heat illnesses can be differentially diagnosed and categorized, they tend to represent a continuum of effects rather than separate entities.

Heat Exhaustion. As the heart rate increases to greater than 160 to 180 beats per minute, there is no longer enough time between beats to completely refill the heart; with each beat it pumps a little less blood to the brain, and the worker begins to feel dizzy, nauseous, and weak. If one sits down to take a break, or slows down enough that too much blood is out in the skin, not being pumped back into the central circulation by body activity, then not enough blood returns to the heart to fill it. As a result, there is not enough blood to be pumped to the brain. The individual is then said to have *heat exhaustion collapse,* and faints or blacks out. Blood flow to the brain is restored when one is lying on the ground, and splashing cold water on the body and massaging the legs and arms may help return blood to the central circulation. The troopers might be able to resume their mission but the heat stored in the body has not yet been removed; if they are allowed to return to work, there is an increased risk of a second collapse from heat exhaustion, or of heatstroke, which could be fatal.

Dehydration Exhaustion. Individuals who lose much more water than they drink will be among the first to be distressed. An adult man has only about 5 liters of circulating blood; most of it is water and most of the sweat produced comes, initially, from this circulating blood reservoir. If the soldier does not replace this lost water by drinking enough water, then *dehydration exhaustion* and collapse are more likely.

Although the tremendously tired feeling that accompanies heat exhaustion, and the collapse that can occur, is frightening, it is not in itself terribly dangerous. Some men have suffered heat exhaustion collapse during exercises in the field for 5 or 6 consecutive days with no residual damage.[2] However, they were allowed to recover completely (cold shower, full rehydration, and overnight rest) before being reexposed to work in the heat the next day.

Heatstroke. The most serious risk of the body's inability to rid itself of heat is not heat exhaustion collapse but that individuals—from those in the best to those in the worst physical condition—may reach core temperatures (T_c) above 41°C. This represents a serious risk because the body is unable to cope with such high temperatures and may suffer permanent damage to its ability to sweat, increase blood flow to the skin, and thermoregulate. If T_c is high enough, and stays high long enough, *heatstroke* can be fatal, from the brain damage that temperatures above 41°C can produce.[3]

Heat Cramps. Individuals whose food intake is inadequate, or who do not add extra salt to their food during the first few days of hard work in the heat, may lose enough body salt that their muscles become sore and *heat cramps* ensue. These muscle cramps can occur simultaneously in a number of sites and can be completely disabling. Salt pills are not the answer. Drinking more, lightly salted, water will prevent heat cramps and, in most instances, cure them if they occur.

Salt Depletion Dehydration. Failure to replace the body's salt losses, and prolonged, daily exposure to heat stress can cause a serious but relatively infrequent condition. The body may reach a new, abnormal balance of salt and water, *salt depletion dehydration,* a condition that is difficult to treat. The body may not recognize that it is short of either salt or water and simply "dump" the extra provided. Ensuring that extra salt is ingested by heavy use of a salt shaker with meals during the first few days' work in the heat, will maintain body salt levels.

Hyperventilation. Some individuals exposed to heat stress (particularly in hot–humid environments) will attempt to pant (like a dog) to rid themselves of extra body heat. However, humans do not lose heat by panting; instead, they may exhale so much of the carbon dioxide normally in the blood that they feel dizzy, tingling of the lips, and faint. The small muscles of the hand may cramp and render the hand useless. Slow, deep rebreathing from a paper bag may be necessary to recover from *overbreathing* (hyperventilation).

(1) Minard D. Physiology of heat stress. In: *The Industrial Environment: Its Evaluation and Control.* Cincinnati, Ohio: National Institute of Safety and Health; 1973. (2) Joy RJT, Goldman RF. A method of relating physiology and military performance: A study of some effects of vapor barrier clothing in a hot climate. *Mil Med.* 1968;133:458–470. (3) Bynum GD, Pandolf KB, Schwette WH, et al. Induced hyperthermia in sedated humans and the concept of critical thermal maximum. *Am J Physiol.* 1978;4:R228.

Figure legend adapted with permission from Goldman RF. Standards for human exposure to heat. In: Mejkavik IB, et al, eds. *Environmental Ergonomics.* New York, NY: Taylor and Francis; 1988: 106, 108–109.

erance. Hard physical conditioning (eg, running for 2 h/d to near exhaustion, for several months) will maximize an individual's cardiac output, while also weeding out less fit individuals who may have met the requirements for military induction but still lack the genetic potential to be fully conditioned to work in the heat. Generally, the most that can be expected from even the best conditioning programs is an increase of about 15% in maximum work capacity, or, in the case of extremely sedentary individuals, who are well below their genetic normal level of fitness owing to their sedentary life-style, perhaps 25% to 30%.

Weight

It is relatively easy to predict the energy expenditure (and hence the heat production) of an individual who is carrying a load while either standing or walking. Energy expenditure for standing or walking on a firm, level surface is shown by the following equation[34]:

$$(5) \quad M = 1.5\ B + 2.0\ (B + L)\ (L \div B)^2 + 1.5\ (B + L)\ V^2$$

where *M* represents metabolic rate, in watts; *B* represents body weight, in kilograms; *L* represents load carried, in kilograms; and *V* represents walking speed, in meters per second.

Equation 5 is the sum of three components. The first is a linear function of body weight, and is the energy expenditure associated with standing without a load. The second is the additional energy expenditure associated with supporting a load while standing. Although the second component is negligible for small loads, it increases with the square of the load. The third component is the additional energy expenditure associated with walking rather than standing still, and increases with the square of the speed of walking.

Obviously, heavier individuals will have a higher heat production than lighter ones, but this is balanced by the heavier individual's greater surface area for heat loss. However, lighter individuals carrying the same weight loads have a higher heat production and, as loads exceed one third of body weight, the risk of physical exhaustion (from accumulating lactate buildup) or heat exhaustion (as skin temperature rises toward the core temperature), or both, increases (see Table 1-3). Obesity, although not generally a problem in the military, is a major risk factor for heat illness in the civilian workforce. If a march is not on a level, paved road the effect of more-difficult terrain becomes another multiplier for the third component above; marching in light brush increases metabolic rate by 30%, in heavy brush by 60% to 80%, and on soft sand by more than 200%. The effects of terrain and increased march rate when carrying loads are also easily seen and, using the simplistic ratio discussed at the start of this chapter—that each increase of 25 kcal/h in heat production is roughly equivalent to 1.67 Centigrade degrees (3 Fahrenheit degrees) in the perceived temperature—it is easy to see the need to reduce the soldier's load, march rate, and the interval between rest breaks to lower the hourly average heat production. Table 1-4 presents the metabolic heat production demands for a variety of typical military activities.

Gender

The classic belief has been that women are less able than men to tolerate work in the heat. Indeed, after attempting to regulate work in heat for the American workforce, the National Institute for Occupational Safety and Health (NIOSH) recommended[35] a considerable differential between male and female workers. It now appears that the perceived difference was primarily one of social and cultural differences; as a group, women had tended to be less physically conditioned and had less exposure than men to conditions that would induce full heat acclimatization.

Subsequent studies[36] suggest that if women are physically conditioned and fully acclimatized to heat, they may have a slight advantage over men under hot–humid conditions, and be at a slight disadvantage under hot–dry conditions, as an effect of their generally smaller body size and ratio of surface area to mass. Any gender differences are a matter of variation within an otherwise comparable group, with some men being less able to work in heat than most women.

Age

Because of the concomitant reduction in maximum heart rate with age, heat tolerance will also be reduced with increasing age, but there are large interindividual differences in physiological, as opposed to chronological, age. The capacity to meet the demands for military tasks is presented in Table 1-5, which shows the decreasing work capacity for troops (both men and women) as a function of age and level of fitness. In this regard, it is important to note that the level of voluntary hard work sustainable by an individual for 3 to 4 hours is 45% of maximum capacity.[37] Work demands that represent 60% of the work capacity can be sustained for only

TABLE 1-4

ESTIMATION OF HEAT PRODUCTION BY ACTIVITY LEVEL*

Work Rate	Activity	Watts (W)	kcal/h
Very light (105–175 W)	At rest, lying down	105	90
	Standing in a foxhole/riding in a truck	116	100
	Guard duty	137	118
	Flying a plane	145	125
	Driving a truck	163	140
Light (175–325 W)	Cleaning a rifle	198	170
	Walking on hard surface at 1 m/s (2.25 mph):		
	no load	210	180
	with 20-kg load	255	219
	with 30-kg load	292	251
Moderate (325–500 W)	Walking on soft sand at 1 m/s (2.25 mph):		
	no load	326	280
	Walking on hard surface at 1.56 m/s (3.5 mph):		
	no load	361	310
	with 20-kg load	448	385
	Scouting patrol	454	390
	Working with pick and shovel/crawling with pack	465	400
	Field assaults	477	410
Heavy (> 500 W)	Walking on hard surface		
	at 1.56 m/s (3.5 mph): with 30-kg load	507	436
	at 2.0 m/s (4.5 mph): no load	525	451
	Digging emplacements	540	465
	Bayonet drill	616	530
	Walking on soft sand at 1 m/s (2.25 mph):		
	no load	642	552

*for a 70-kg, young, physically fit soldier
Source: Adapted from US Army Research Institute of Environmental Medicine. *Heat Illness: A Handbook for Medical Officers.* Natick, Mass: USARIEM; June 1991. Technical Note 91-3 (AD A238974).

about 1 hour before physical exhaustion occurs without any complications from heat per se.

In the military, a more serious effect of age is the perceived need for younger, and also for older, troops to prove that they can cope with heat and, in general, perform as well as the rest of the unit. This factor may be more responsible than physiological differences for the greater incidence of heat casualties observed in both younger and older individuals. This may be particularly relevant for younger and older unit leaders; in one field study on the effects of chemical protective uniforms on a platoon's ability to set up hasty defensive positions, all the unit leaders suffered heat exhaustion collapse during the first few hours. Of course, the extra physical work of leaders in trying to ensure that the entire area was prepared as rapidly and as well as possible may have been a contributing factor.[38]

TABLE 1-5

MAXIMUM WORK CAPACITY (IN WATTS*) BY AGE AND FITNESS LEVEL

	Fitness Level				
Age	**Poor**	**Fair**	**Average**	**Good**	**Excellent**
Men					
17–19	< 924	948–1,042	1,066–1,161	1,185–1,256	> 1,279
20–24	< 829	853–924	948–1,066	1,090–1,232	> 1,256
25–29	< 805	829–900	924–1,042	1,066–1,184	> 1,208
30–34	< 782	805–877	900–1,019	1,042–1,137	> 1,161
35–39	< 758	782–829	853–971	995–1,090	> 1,113
40–44	< 711	734–805	829–900	924–1,042	> 1,066
45–49	< 663	687–758	782–829	853–1,019	> 1,042
50–54	< 616	640–711	734–782	805–995	> 1,019
Women					
17–19	< 782	805–900	924–995	1,019–1,113	> 1,137
20–24	< 758	782–853	877–948	971–1,066	> 1,090
25–29	< 711	734–805	829–924	924–1,019	> 1,042
30–34	< 663	687–758	782–877	900–971	> 995
35–39	< 616	640–711	734–829	853–924	> 948
40–44	< 577	592–663	687–782	805–877	> 900
45–49	< 521	545–616	640–734	758–829	> 853
50–54	< 474	497–569	592–687	711–782	> 805

*To convert these values from watts to kcal/h, multiply by 0.86.
Adapted by Goldman RF, from a table that was developed at US Army Research Institute of Environmental Medicine, Natick, Mass; circa 1975.

Type of Task

The effects of heat on physical task performance can be inferred from those seen previously in Table 1-4. Decrements in psychological task performance, on the other hand, are more difficult to assess but they can be offset by training, experience, leadership, and motivation to a much greater degree than the decrements in physical work tasks. However, failures in such cognitive tasks as perception, judgment, and decision making can be far more deadly, particularly if troop leaders are affected. Exhibit 1-3 suggests levels of rectal temperatures at which a variety of physical and psychological task capacities may be adversely affected.

Skin Diseases

Only one common skin disease of military relevance, prickly heat (miliaria rubra), has been shown to reduce heat tolerance.[39] Any impairment of sweat gland function (eg, delayed onset of sweating, congenitally fewer sweat glands), by exposure to chemical warfare agents or antidotes, or total absence of sweat glands, will reduce heat tolerance.

Race

Although the concept of "race" was first articulated by Kant in the 1800s, and race is difficult to define as a distinct grouping of characteristics, it has been suggested that, as a group, "blacks" are less heat-tolerant than "whites." This is certainly supported by US Army medical reports, as analyzed by Colonel Tom F. Whayne, later in this chapter. Although the incidence of heat illness among black troops was less than it was for white troops during the American Civil War, Spanish–American War, World War I, and World War II, the severity of heat illness and the death rates during those same wars

EXHIBIT 1-3

PHYSIOLOGICAL LEVELS OF CONCERN

1. Rectal Temperature (with elevated skin temperature)

Physical Tasks

38.2°C	NIOSH limit (discomfort)*
39.2°C	25% risk of heat casualties†
39.5°C	50% risk of heat casualties
40.0°C	100% casualties ("heat ineffectives")

Cognitive Tasks

37.7°C	Threshold of decrement
38.2°C	Slowed cognitive function
38.5°C	Increased errors in judgment
39.6°C	Suggested functional limit

Motor Tasks

37.9°C	Decreasing manual dexterity
38.8°C	Loss of tracking skills

2. Heat Storage

Level	Effect
80 kcal	Discomfort
120 kcal	Performance degrades (T_{re} ~ 39°C)
160 kcal	Tolerance time limit (50% risk)
> 200 kcal	Potential damage

3. Heart Rate‡

Level	Effect
≥ 120 bpm	Discomfort (8-h tolerance)
≥ 140 bpm	4-h limit
≥ 160 bpm	2-h limit
> 170 bpm	Potential damage (*f* age)

*Recommended (but not formally approved) upper limit proposed for civilian workforce.
†Actual heat exhaustion and heat collapse casualties will be about half the number at risk, if the operational scenario allows the level of activity (M) to be reduced as troops approach heat exhaustion.
‡For a civilian workforce of uncertain fitness and age, an increase in heart rate > 30 bpm above the individual's resting level is to be avoided.[1]
bpm: beats per minute; *f:* function of; NIOSH: National Institute for Occupational Safety and Health; T_{re}: rectal temperature
(1) Brouha L. *Physiology in Industry.* New York, NY: Pergamon Press; 1960.

were significantly higher among black troops. A similar situation appears in British army medical reports in reference to Indian troops. However, analyses with prediction models for heart rate and rectal temperature responses to rest, work, and recovery in the heat, developed at USARIEM by Goldman and associates,[40,41] show that prediction models developed primarily on data from white soldiers provide an equally good fit to the responses of black mine workers in South Africa.[42] This suggests that social, rather than physiological, differences may be responsible for this variability in heat injury.

Acclimatization and Acclimation

Seven to 10 days of work in the heat, for 2 h/d, is generally considered sufficient to produce "full" acclimatization to heat exposure that is no more severe than the conditions of heat and exercise that produced the acclimatization. A large part of the beneficial effect of acclimatization is a result of increased sweating (due both to earlier onset of sweating and to increased secretory capacity of the sweat glands). Under conditions in which sweat can freely evaporate, acclimatization can result in a dramatic improvement in heat tolerance. However, if sweat evaporation is limited, either by lower-permeable or heavier protective clothing (lower I_m/clo) or elevated ambient vapor pressure, the benefits of heat acclimatization may be sharply decreased. Wearing body armor has been equated[43] to an increase of about four Centigrade degrees (seven Fahrenheit degrees) in the wet bulb globe temperature (WBGT) index, while complete encapsulation in chemical–biological protective ensembles (mission-oriented protective posture [MOPP 4] gear) has been equated to an increase of about 5.5 Centigrade degrees (10 Fahrenheit degrees).

With such heavy protective clothing it is conceivable that heat acclimatization could turn into a liability; the more rapid fluid loss will result in greater dehydration, without any real gain in sweat evaporative cooling. A well-conditioned soldier (ie, one in the best physical condition allowed by his genetic potential for $\dot{V}O_2max$) will have roughly the

equivalent of an induced acclimatization after 3 days of work in the heat, just from his or her superior cardiovascular conditioning. However, the full benefit of 7 days of work in the heat cannot be obtained from work, no matter how hard, without concomitant heat exposure.

For the military, the psychological and behavioral adaptations developed by exposure to work in the heat may be more important than the physiological changes, in a manner somewhat analogous to what the British used to term "blooding the regiment," particularly if we consider the limitations imposed by protective clothing. Troops, and especially leaders, who are experienced in the problems of heavy work in the heat can take a variety of steps to reduce the potential heat stress: reducing the heat production (reduced load, rotation of heavy tasks, more frequent rests—in shade rather than in sun) and increasing water intake by command control (predrinking, provision of extra canteens and, most effective, requiring water intake at every rest break) are more beneficial than acclimatization when wearing protective clothing. In one field exercise, troops who were briefed on the potentials for heat stress and given a simple field instrument with which to identify high-risk conditions were able to operate with minimal heat casualties, compared with troops lacking such briefing or instruments.[44]

Hydration

Maintaining normal levels of body water, and even prehydration by ingesting 1 or 2 pints of water, is an important factor in resistance to heat illness. The effects of dehydration are explicated later in this chapter.

Electrolyte (Sodium Chloride) Intake

For this discussion, sodium is the essential electrolyte. The daily intake of salt pills during World War II was perhaps the worst possible doctrine to accompany the doctrine of water discipline. Supplementary salt *may* be useful for unacclimatized troops during the first few days of work in heat, before they become fully acclimatized, if they are not eating normally. However, any supplementary salt intake can interfere with one aspect of the heat acclimatization process: a decreasing content of sodium in the sweat.

The normal US military diet usually contains ample—even excessive—salt; supplements are usually unnecessary. Dasler and associates,[45] who conducted research studies on salt intake and heat acclimatization for the US Navy in the early 1970s, suggested that supplementary salt intake—beyond the already high salt content of a normal diet—retarded development of heat acclimatization. Unfortunately, the late-20th-century marketing success of "sports drinks," which contain large amounts of electrolytes and glucose, seems certain to continue to provide funds to (1) support research proving the benefits of such drinks and (2) attempt to convince the military that purchase of such drinks would be beneficial to troops. These commercial marketing attempts may well prevail despite ample evidence that such drinks represent unnecessary—and possibly detrimental—supplements to a normal military diet, which typically contains large amounts of salt.

Initially Elevated Body Temperature

Any heat produced (M) or received (H_R [radiant] or H_C [convective]) by the body that cannot be eliminated from the skin through the protective clothing to the ambient environment, must be stored in the body. The temperature of an average 70-kg body increases by one Centigrade degree for every 60 kcal of heat that it must store. Resting heart rate also increases with heat storage. Thus, any initial increase in body temperature means that there is simply that much less capacity to store additional heat before suffering heat exhaustion collapse (see Table 1-3 and Exhibit 1-3).

Body temperature elevations produced by any of the following will cause problems with heat to occur sooner and to be more severe:

- bacterial, viral, or parasitic diseases, or inoculations against them;
- previous activity with inadequate recovery, identified by a greater rate of increase in heart rate on reexposure to activity than the initial rate of rise[46]; or
- dehydration, a frequent sequela of even moderate alcoholic beverage ingestion during the preceding 24 hours.

In addition, a destabilizing effect of alcohol intake on the body's vasomotor control of blood pressure has also been observed[47,48] as a result of the accompanying dehydration (personal observation). Indeed, although there are no data on heat casualties during the US Revolutionary War, the comments of Dr. Benjamin Rush, a signer of the Declaration of Independence and physician who served during the American Revolutionary War, seem remarkably prescient:

> What should I say to the custom of drinking spiritous liquors which prevails so generally in our Army? I am aware of the prejudices in favor of it. It requires an arm more powerful than mine; the Arm of a Hercules to encounter it. The common apology for the use of rum in our Army is that it is necessary to guard against the effects of heat and cold. But I maintain that in no case whatever does rum abate the effects of either of them upon the constitution. On the contrary, I believe it always increases them. The temporary elevation of the spirits in the summer, and the temporary generation of warmth in the winter, produced by rum, always leave the body languid, and more likely to be affected with heat and cold afterward.[47(p6)]

EFFECTS OF HEAT ON MILITARY OPERATIONS

Having explored the usual epidemiological triad of agent, host, and disease, we now turn to the observed effects of heat on military operations in the heat. The primary sources of information are experimental studies of the effects of heat on humans and analyses of heat illness in the civilian population, medical analyses of wartime heat casualties, and afteraction reports of military operations in the heat.

Experimental and Analytical Studies

Most experiments and analytical studies were carried out to answer questions that were generated by wars. World War II generated questions on heat, cold, and altitude; the Korean War turned the focus to cold; and the Vietnam War and the later involvements in Iraq redirected the focus to heat. Most of the relevant studies were conducted in military laboratories or under contracts funded by the military. As might be expected, the US Army Research Institute of Environmental Medicine (USARIEM) and its predecessor organizations, the Environmental Protection Research Division (EPRD) of the Office of the Quartermaster General, at Natick, Massachusetts, and the Office of The Surgeon General's Armored Medical Research Laboratory (AMRL), at Fort Knox, Kentucky, were primary centers for these studies. (For interested readers, a short description of the origins and kinds of heat-related research done at USARIEM is attached to the end of this chapter.) Two themes dominated the heat studies: the effects of work under hot conditions and the role of dehydration in military operations.

Physiological Effects of Dehydration

In the 1950s, the US Army Quartermaster and Engineers, charged with delivering potable water to the troops, developed a chart entitled "Daily Water Requirements for Three Levels of Activity" (Figure 1-3). Although more extensive studies have been conducted subsequently,[49] these still seem reasonable guidelines. Doctrine promulgated in 1981, entitled "Water Intake, Work/Rest Cycles During Field Operations for Heat Acclimated Units," also supported these guidelines (Exhibit 1-4), and the Quartermaster group also developed a set of graphs entitled "Insufficient Water Intake and Impairment of Operational Effectiveness" (Figure 1-4). The guidance contained

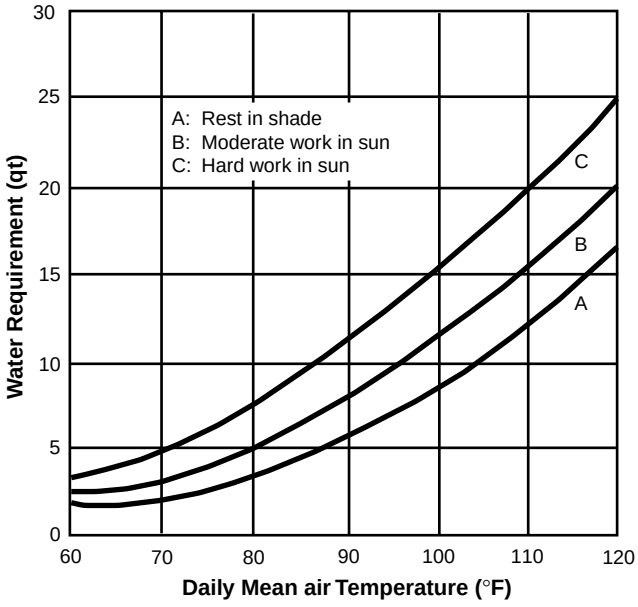

Fig. 1-3. Daily water requirements for three levels of activity. This graph shows theoretical water needs, in quarts per day, for men at rest and at work in sun and shade, in relation to the daily mean air temperature measured in degrees Fahrenheit. Curve A: rest in shade; Curve B: moderate work in sun; Curve C: hard work in sun. For example, if a man does 8 hours of hard work in the sun (Curve C) when the average temperature of the day is 100°F, then his water requirements for that day will be approximately 15 qt. Adapted with permission from Adolph EF, Brown AH. Economy of drinking water in the desert. In: Wulsin FR. *Response of Man to a Hot Environment.* Washington, DC: Climatic Research Unit, Research and Development Branch, Military Planning Division, Office of The Quartermaster General; 1 Aug 1943: Figure 10. First published in the present format in Environmental Protection Research Division. *Environment of South East Asia*. Natick, Mass: US Army Natick Laboratories; Aug 1953. EPRD Report 219.

EXHIBIT 1-4

WATER INTAKE, WORK/REST CYCLES DURING FIELD OPERATIONS FOR HEAT-ACCLIMATIZED UNITS*

Heat Condition	Botsball WGT (°F)†	Water Intake (qt/h)	Work/Rest Cycle (min)
Green	80°F–83°F	0.5–1.0	50/10
Yellow	83°F–86°F	1.0–1.5	45/15
Red	86°F–88°F	1.5–2.0	30/30
Black	≥ 88°F	2.0	20/40‡

*Provisional doctrine, April 1981; guidance for unacclimatized troops was given in Headquarters, Department of the US Army. *Prevention, Treatment and Control of Heat Injury.* Washington, DC: HQ, DA; July 1986. Technical Bulletin Medicine 507.

†WGT is a successor to WBGT (see below); to convert WGT to WBGT, add two Fahrenheit degrees. Below 80°F, drink up to 0.5 qt/h; 50/10 work/rest cycles.

‡Depending on the condition of the troops.

WBGT: wet bulb globe temperature; WGT: wet globe temperature

To maintain physical performance:

1. Drink 1 quart of water in the morning, at each meal, and before any hard work.
2. Take frequent drinks because they are more effective than drinking all at once. Larger men need more water.
3. Replace salt loss by eating three rations per day.
4. As WGT increases, rest periods must be more frequent, work rate lowered, and loads reduced.
5. Use water as a tactical weapon and maintain top efficiency by drinking each hour.

NOTE: The Botsball was developed in 1971 by James Botsford, an industrial hygienist working for ALCOA, to measure the heat stress in the area around metal-smelting furnaces. The Botsball is a small (3″), hollow cylinder covered with a thin, black, cotton fabric, which is kept wet by the water in a cylinder (6″ x 2″) stem at the top of the 3″ cylinder. A standard metallic stem dial thermometer is inserted into the 6″ stem so that its sensing tip is sited at the center of the now-wetted black globe. Thus, this wet globe thermometer (WGT) provides a single numerical reading for the combined effects of thermal radiation, ambient temperature and humidity, and air motion. Goldman adapted Botsford's device to incorporate the green, yellow, red, and black sections for the numerical readings on the dial, and was able to get triservice agreement to adopt this device as a useful measure of heat stress in 1978. The WGT is obviously much easier to obtain, read, and interpret than the conventional wet bulb globe temperature (WBGT). Although vastly simpler than the conventional WBGT, and so portable that it can be carried and used on a march, its use has been questioned as a less-accurate index by some laboratory scientists. How a simple color indication of the actual heat stress conditions at the site where troops are working can be deemed "less accurate" than an index calculated by a weatherman reading three separate instruments and providing a numerical value for conditions at a weather station some distance away (usually at an airfield) is an interesting scientific sophistry. For further information, interested readers can see Onkaram B, Stroschein LA, Goldman RF. Three instruments for assessment of WBGT and a comparison with the WGT (Botsball). *Am Ind Hyg Assoc J.* 1980;41:634–641.

Adapted with permission from Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, eds. *Protecting Personnel in Hazardous Waste Sites.* Stoneham, Mass: Butterworth Publishers; 1985: 246.

in Exhibit 1-4 was reevaluated in the late 1990s because of an increasing incidence of water intoxication in military basic trainees. Although that guidance recommended water consumption of more than 1.5 qt/h in heat categories 4 and 5 (red and black flag conditions), it was found that requirements for fluid replacement never exceeded 1.5 qt/h, at least when the prescribed work/rest cycles were followed.[50] Based on this work, the army's policy on water replacement requirements during training have been revised to incorporate the recommendations in the paper by Montain, Latzka, and Sawka.[50] The new water replacement guidelines are also discussed in Chapter 7, Clinical Diagnosis, Management, and Surveillance of Exertional Heat Illness; see Exhibit 7-7 in that chapter.

Data from physiological studies on human volunteers (1) reaffirm that ad libitum water intake is inadequate to maintain full hydration and (2) sug-

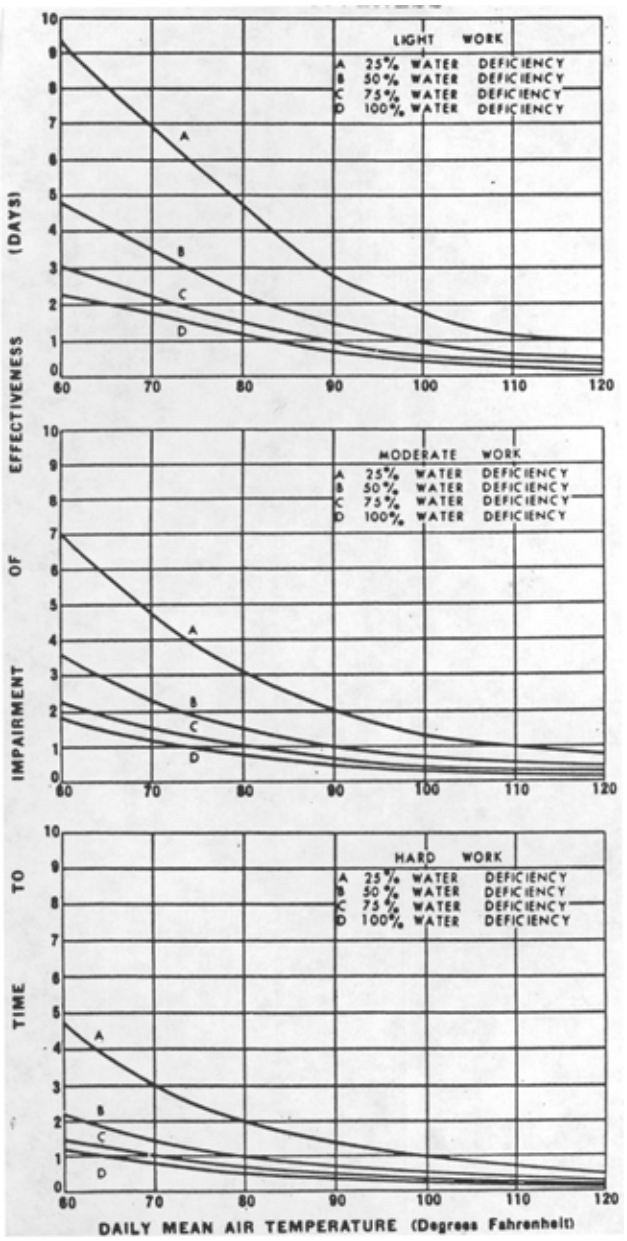

Fig. 1-4. Insufficient water intake and impaired operational effectiveness. Dehydration occurs when water intake is not adequate to replace the water lost from the body. When the amount of body water lost exceeds 5% of the body weight (about 3.5 qt), operational effectiveness is impaired. The three graphs, which represent, from top to bottom, light, moderate, and hard work, show the time required for impairment of effectiveness at various average daily temperatures. Reprinted from Environmental Protection Research Division. *Environment of South East Asia*. Natick, Mass: US Army Natick Laboratories; Aug 1953. EPRD Report 219.

gest that rectal temperature (T_{re}) is elevated by about 0.15 Centigrade degree for each 1% of dehydration above the threshold 2% (little effect is seen at the threshold level). The corresponding rise in heart rate is about 3.5 beats per minute per percentage of dehydration, whereas sweat rate falls by about 100 mL/h per percentage of dehydration.

Effects of Work and Temperature

A number of studies have been conducted throughout the world on conditions that produce significant discomfort at different work rates (ranging from the metabolic heat production of 90 kcal/h at rest up to 425 kcal/h), as well as on the inability of highly motivated, physically fit, young men to tolerate at least 4 hours of work without collapse. The results, plotted in Figure 1-5 as a function of the effective temperature index (ET, the air temperature at 100% humidity with minimal air motion, at which different combinations of T_a, rh, and air motion would *feel* the same as the ET), clearly show the linear relationship between heat production and ambient heat. The effect of "significant discomfort" can be seen to begin at an ET of about 78°F (25.5°C) while working at about 450 kcal/h; this level of work represents a "voluntary, self-paced, hard work" level sustainable for 3 to 4 hours.[37] An ET of 78°F is somewhat above the upper boundary of thermal comfort for sedentary workers (M ≈ 125 kcal/h) reported in the American Society of Heating Refrigerating and Air-conditioning Engineers Comfort Standard 55; at this level of heat production, "significant discomfort" is shown to be about 92°F (33°C). At rest (M = 100 kcal/h), "4-hour tolerance" is shown at 97°F (36°C), whereas 88°F (31°C) appears as the 4-hour limit for work at 425 kcal/h.[51] These severe discomfort and 4-hour tolerance limits for fit young men (frequently soldiers) can be compared with "Permissible Heat Exposure Threshold Limit Values" that were proposed in 1970 for an average American worker by the American Conference of Government Industrial Hygienists (ACGIH)[52] (Table 1-6).

Laboratory research studies on more severe heat conditions were conducted between 1923 and 1967 in a number of countries on "heat tolerance" (ie, the time, during exposures of up to 3 h of heat, to reach T_{re} 39.2°C at rest, or 39.5°C during work, or a heart rate of 170 to 180 beats per minute). The data show remarkable agreement when plotted as a function of the Oxford Index, WD (or wet–dry), calculated as

$$0.85\ (T_{wb}) + 0.15\ T_{db}$$

where T_{wb} represents the wet bulb thermometer

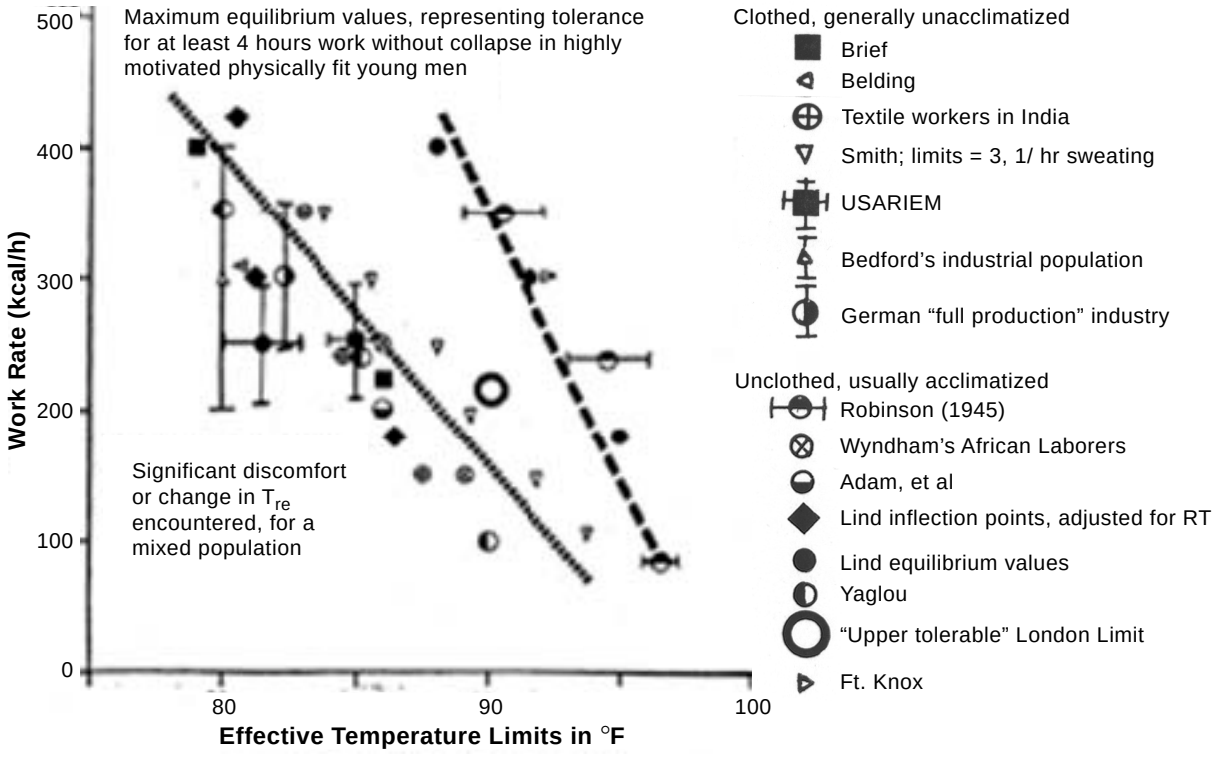

Fig. 1-5. The effective temperature limit at a given work rate, resulting in "significant discomfort" (dotted line) or "tolerance for at least 4 hours" (dashed line). "Mixed" refers to mixed military and civilian, or very fit and less fit, populations. Adapted with permission from Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, eds. *Protecting Personnel in Hazardous Waste Sites.* Stoneham, Mass: Butterworth Publishers; 1985: 249.

Sources for data points:

Clothed, generally unacclimatized: (1) Brief RS, Confer RG. Comparison of heat stress indices. *Am Ind Hyg Assoc J.* 1971;32:11–16. (2) Belding HS, Hentig BA, Riedesel ML. Laboratory simulation of a hot industrial lab to find effective heat stress and resulting physiologic strain. *Am Ind Hyg Assoc J.* 1960;21:25. (3) Mankiber NS, ed. *Thermal Stress in the Textile Industry.* New Delhi, India: Government of India; 20 July 1957. Ministry of Labor and Employment Report 17. (4) Smith FE. *Indices of Heat Stress*. London, England: Her Majesty's Printing Office; 1955. Medical Research Council Report 29. (5) Iampietro PF, Goldman RF. Tolerance of men working in hot humid environments. *J Appl Physiol.* 1995;20:73–76. (6) Goldman RF, Iampietro PF, Green EB. Tolerance of hot, wet environments by resting men. *J Appl Physiol.* 1965;20:271–277. (7) Bedford T, Warner GC. Observations on the working capacity of coal miners in relation to atmospheric conditions. *J Ind Hyg*. 1931;13:252–260. (8) Ehrismann O, Hasse A. Uber die zulassige Arbeitszeit bei hoher Temperatur und Luftfeuchtigkeit [On the allowable work time at elevated temperature and humidity. In German.]. *Archives Gewerbepath & Gewerbehyg*. 1938;8:611–638. (9) Hasse A. Leistung und klimatische Bedingungen in Berban [Performance and climatic conditions in mining. In German.]. *Arbeitsphysiologie*. 1935;8:459–475.

Unclothed, usually acclimatized: (1) Robinson S, Turrell ES, Gerking SD. Physiologically equivalent conditions of air temperature and humidity. *Am J Physiol.* 1945;143:21. (2) Wyndham CH, Strydom NB, Morrison JF, et al. Criteria for physiological limits for work in heat. *J Appl Physiol.* 1965;20:37. (3) Adam JM, Jack JW, John RT, MacPherson RK, Newling PSB, You PS. *Physiological Responses to Hot Environments of Young European Men in the Tropics, IV: The Response to Hot Environments of Young Men Naturally Acclimatized to Tropical Conditions.* London, England: Medical Research Council; 1953. Royal Naval Personnel Report 53/767. (4) Adam JM, Jack JW, John RT, MacPherson RK, Newling PSB, You PS. *Physiological Responses to Hot Environments of Young European Men in the Tropics, II and III: Further Studies on the Effects of Exposure to Varying Levels of Environmental Stress.* London, England: Medical Research Council; 1955. Royal Naval Personnel Report 55/831. (5) Lind AR. Determination of environmental limits for everyday industrial work. *Ind Med Surg*. 1960;29:515. (6) Lind AR, Hellon RF. Assessment of physiologic severity of hot climates. *J Appl Physiol*. 1957;11:35. (7) Yaglou CP. Indices of comfort. In: Newburgh LH, ed. *Physiology of Heat Regulation and the Science of Clothing.* Philadelphia, Pa: Saunders; 1949.

Upper tolerable London limit: (1) McArdle B, Dunham W, Holling HE, et al. The prediction of the physiologic effects of warm and hot environments: The P4SR Index. *Medical Research Council (London), Royal Navy Personnel Report.* 1947;47:391.

Fort Knox: (1) Nelson N, Eichna LW, Horvath SM, Shelley WB, Hatch TF. Thermal exchanges of man at high temperatures. *Am J Physiol.* 1947;151:626. (2) Hatch TF. Assessment of heat stress. In: Hardy JD, ed. *Temperature, Its Measurement and Control in Science and Industry.* Vol 3. New York, NY: Reinhold. 1963: 307.

TABLE 1-6

HEAT EXPOSURE THRESHOLD LIMIT VALUES FOR THE AVERAGE AMERICAN WORKER

Work/Rest Regimen	Work Load (at WBGT [°C])		
	Light	Moderate	Heavy
Continuous work	30.0	26.7	25.0
75% work/25% rest*	30.6	28.0	25.9
50% work/50% rest	31.4	29.4	27.9
25% work/75% rest	32.2	31.1	30.0

*percentage of each hour (ie, 75% work = 45 min; 25% rest = 15 min)
WBGT: wet bulb globe temperature
Adapted with permission from Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, eds. *Protecting Personnel in Hazardous Waste Sites.* Stoneham, Mass: Butterworth Publishers; 1985: 243.

temperature and, as before, T_{db} represents the ambient temperature. As shown in Figure 1-6, tolerance times at rest exceed 3 hours for WD < 98°F (~ 37°C), and fall steeply to < 40 minutes as WD increases to 108°F (~ 42°C). Although these WD levels are extreme for outside ambient conditions, they are not uncommon inside buttoned-up (ie, fully closed) armored fighting vehicles, where interior temperatures average 13 Fahrenheit degrees (7 Centigrade degrees) above outside air temperatures, and can reach as high as 30 Fahrenheit degrees (17 Centigrade degrees) above, while the tankers' sweat also elevates interior humidity. A WD of 37°C may also occur as the microclimate within heavy, less-permeable protective clothing ensembles during hard work under warm dry bulb temperature conditions.

At moderately hard work levels (280–350 kcal/h, for a civilian work force), tolerance time is limited to about 3 hours at a WD of 91°F (33°C), a condition regularly found in clothing microclimates, crew compartments, and shelters without air conditioning. Physically trained, young, military troops may not have as much problem at this WD and work level, but tolerance times will drop to 40 minutes at a WD of 98°F (36.7°C) and thereafter approach 10 minutes as WD approaches 120°F (49°C), even in the fittest individuals. The human body appears to tolerate up to 20 minutes of exposure, at rest or at work; the lag in rectal temperature response associated with the mass of an adult body[40] accounts for this 20-minute minimum tolerance time to the most severe heat conditions, as long as skin temperature remains below the pain threshold for skin (at T_{sk} of ~ 45°C). Note the very high correlation coefficient (r = 0.96) shown for the combined rest and work data points when plotted as a log-log function of tolerance time (scaled differently for work and rest) and WD. This implies that > 90% of the tolerance time response is a direct effect of WD, which is consistent with the observation that interindividual and intraindividual human variability dramatically diminishes with increasing heat stress. An important meaning of this finding is that when a military unit suffers more than one or two heat casualties during an operation, the remaining troops have a high probability of becoming heat casualties *if* they are doing comparable tasks, at comparable rates, and the first few casualties were not unusually impaired by preexisting problems (eg, dehydration, infection, lack of acclimatization, etc).

Finally, Figure 1-7 shows the predicted times, at elevated WBGT levels, at which a 50% risk of heat casualties will occur[38] under the most severe heat exposures apt to be experienced by military personnel while wearing chemical protective ensembles, either in

- closed suit (MOPP 4): wearing gas masks with protective hoods, gloves, and overboots, with all openings sealed to provide as complete encapsulation as possible against chemical–biological agents, or
- open suit (MOPP 1): without the hood, gloves, and overboots, and with neck and wrists as open as possible.

At the sustainable level of voluntary hard work for 3 to 4 hours in fit, young soldiers (M = 450 kcal/h), any difference in a 60-minute time to 50% risk of heat casualties for both open and closed suits is negligible at WBGT levels above about 86°F (30°C). At lower WBGT levels, the predicted differences between ensemble configurations during light work become more meaningful, with 60 minutes at a WBGT of 70°F (26°C) in a closed suit versus about 90 minutes in an open suit. In the studies on which these predictions were based,[38] regardless of open or closed suit configuration, 25% of the troops were stopped for safety reasons when their rectal temperatures reached 39.5°C, before they suffered heat exhaustion collapse (ie, loss of consciousness), and 25% of the troops had already collapsed (hence, the specification of 50% risk). For moderate work (M = 325 kcal/h), the predicted time to incur 25% frank heat casualties, with another 25% having been stopped for safety reasons, at a WBGT of 80°F (26.5°C), is about 2 hours in the closed suit but about 4 hours in the

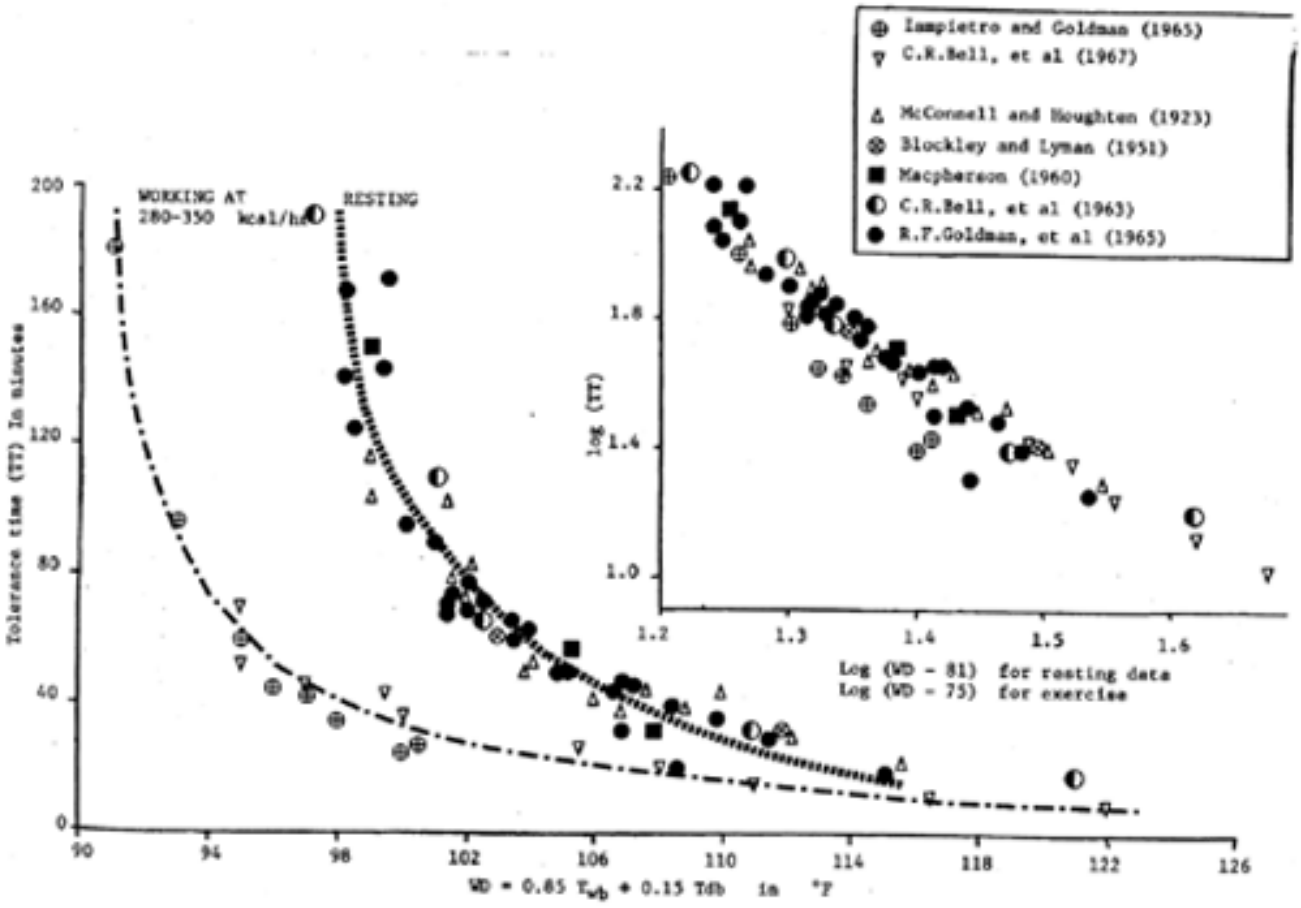

Fig. 1-6. Tolerance time (defined as minutes to reach a deep body temperature [T_{re}] of 39.5°C [103°F] or a heart rate of 180 beats per minute, or both) at rest (| | | | | line) or at work (metabolic rate = 280–350 kcal/h, — • — • — • line), expressed as a function of the wet–dry (WD, or Oxford index = 0.85 T_{wb} + 0.15 T_{db}) Index. The outstanding agreement between the data from these seven studies, conducted between 1923 and 1965 in several countries, suggests the uniformity of response under conditions of high heat stress (WD > 90°F) at rest, and at moderate work (280–350 kcal/h) levels. This suggestion is supported on examination of the inserted log-log plot, which shows that work data can be combined into a single line by simple adjustment of the log of the WD index (ie, WD –81 at rest, and WD –75 at moderate work). The correlation coefficient (r) for these combined data points to the log-log line is 0.96, which indicates that 92% (r^2) of the tolerance time is accounted for by the WD index, with only 8% associated with other factors such as individual variability. Adapted with permission from Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, eds. *Protecting Personnel in Hazardous Waste Sites.* Stoneham, Mass: Butterworth Publishers; 1985: 250.

Sources for data points: (1) Iampietro PF, Goldman RF. Tolerance of men working in hot humid environments. *J Appl Physiol.* 1965;20:73–76. (2) Bell CR, Walters JD, Watts AN. *Safe Exposure Times for Men Working in Hot and Humid Conditions.* London, England: Royal Navy Personnel Research Committee, Medical Research Council; 1967. RNPRC Report RNP 67/1092. (3) McConnell WJ, Houghton FC. Some physiological reactions to high temperatures and humidities. *American Society of Heating and Ventilating Engineers Transactions.* 1923;29:125–129. (4) Blockley WV, Lyman J. *Studies of Human Tolerance for Extreme Heat.* Dayton, Ohio: Wright Patterson Air Force Base; 1950. USAF Materiel Command Technical Report 5831. Blockley WV, Lyman J. *Studies of Human Tolerance for Extreme Heat, IV: Psychomotor Performance of Pilots as Indicated by a Task Simulating Aircraft Instrument Flight.* Dayton, Ohio: Wright Field Air Development Command, Aeromedical Laboratory; 1951. (5) MacPherson RK, Ellis FP. *Physiological Responses to Hot Environment.* London, England: Medical Research Council, Her Majesty's Stationery Office; 1960. (6) Bell CR, Hellon RF, Hiorns RW, Nicols PB, Provins KA. *Exposure to Very Hot Conditions.* London, England: Royal Navy Personnel Research Committee, Medical Research Council; 1963. RNPRC Report RNP 63/1035. (7) Goldman RF, Green EB, Iampietro PF. Tolerance of hot, wet environments by resting men. *J Appl Physiol.* 1965;20:271–277.

Closed suit (MOPP 4): CBR protective ensemble worn with gas mask with protective hood, gloves, and overboots, with all openings sealed to provide as complete encapsulation as possible against chemical–biological agents and nuclear radiation.

Open suit (MOPP 1): CBR protective ensemble worn without the hood, gloves, and overboots, and with neck and wrists as open as possible.

MOPP: mission-oriented protective posture gear

WBGT: wet bulb globe temperature (= 0.7 T_{wb} + 2 T_{globe} + 0.1 T_{db})

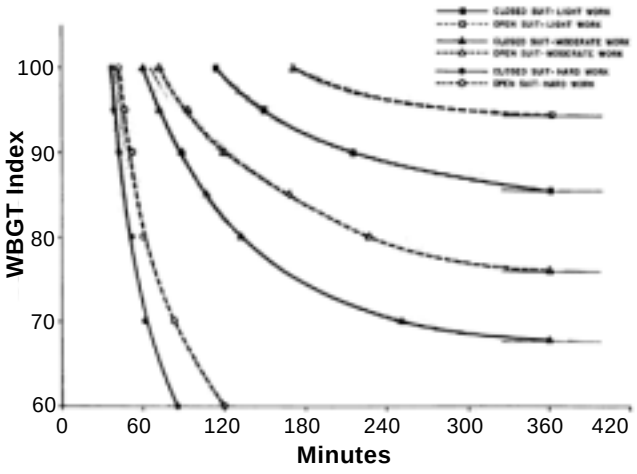

Fig. 1-7. Predicted time to 50% heat casualties when chemical, biological, radiological (CBR) protective suits are worn. When wearing CBR protective clothing, whether the protective ensemble is worn fully closed (MOPP 4) or open (MOPP 1, with the mask but with the neck and wrist areas not buttoned closed and even with sleeves rolled up above the wrists), the mismatch between the heat produced during hard work and the heat that can be lost by the body to the environment is very severe at WBGT > ~ 75°F. There is only a marginal reduction of heat stress by exposing so much area to possible CBR agents: about 12 minutes during hard work at 75°F in the predicted time to 50% heat casualties (ie, heat exhaustion collapse of 25% of the troops, with another 25% at high risk of it). Even at WBGT of 60°F, predicted time to 50% risk of unit heat casualties is only about 90 minutes in MOPP 4, and 120 minutes in MOPP 1 at sustained hard work (eg, > 450 kcal/h). At moderate work levels (~ 300 kcal/h), the difference in MOPP levels is predicted to result in an offset of about 10 Fahrenheit degrees in WBGT, with 50% risk of heat casualties occurring in 4 hours (240 min) at about 78°F WBGT in open suit, and about 68°F WBGT in MOPP 4. At light work, there should be little risk of heat casualties at WBGT < 85°F in MOPP 4, or < 95°F in MOPP 1. Finally, after 6 hours of exposure, *if* troops are kept fully hydrated, the risk of heat casualties appears to have reached a plateau at light and moderate work levels. The shaded areas on the graph represent an estimate (and only an estimate) of variability around the curve. The straight lines on both sides of the last four data points suggest the asymptotic nature of the curves at those points. Reprinted with permission from Joy RJT, Goldman RF. A method of relating physiology and military performance: A study of some effects of vapor barrier clothing in a hot climate. *Mil Med.* 1968;133:469.

open suit. At very light work (M = 125 kcal/h), predicted tolerance time is about 2 hours at a WBGT of 100°F (38°C) with closed suits, and about 3 hours with open suits; such an extreme WBGT might occur in a buttoned-up armored fighting vehicle sitting in the sun with its engine running. Combat-ready tank crews in MOPP 4, serving as subjects in the field research that led to introduction of air conditioning for the latest US tank,[53] had to be helped out of the tank after 80 minutes of rest interrupted by several short firing missions, during which they had to hand-crank the gun turret into position. Each crew member had lost more than 3 L of sweat during the 80 minutes, as WBGT rose above 90°F inside the tank.

The predicted tolerance times shown in Figure 1-7 for wearing chemical–biological protective clothing in the heat can be compared with those shown in Table 1-7, which is compiled from various research done in the United States, the United Kingdom, and USSR.[54–59]

Although three different temperature indices were used in Figures 1-5 through 1-7 (ET, WD, and WBGT, respectively), they are all comparable at higher levels. Thus, the data from all three figures

TABLE 1-7

SAFE EXPOSURE PERIODS FOR MODERATE WORK* WHILE WEARING CHEMICAL PROTECTIVE UNIFORM SYSTEMS

Air Temperature (°F)	Safe Wear Time (h) (closed suit)†
< 30	8
30–50	5
50–60	3
60–70	2
70–80	1.5
80–85	1.0
85–90	0.5
> 90	0.25

*250 kcal/h

†MOPP 4 (mission-oriented protective posture gear): wearing gas masks with protective hoods, gloves, and overboots, with all openings sealed to provide as complete encapsulation as possible against chemical and biological agents

Adapted with permission from Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, eds. *Protecting Personnel in Hazardous Waste Sites.* Stoneham, Mass: Butterworth Publishers; 1985: 246.

represent a spectrum of temperature conditions from ET levels of 80°F to 95°F, WD levels from 91°F to 122°F, and WBGT levels while wearing chemical–biological protection, of 60°F to 100°F (ie, a range of index temperatures from about 20°C to 50°C). The various discomfort and tolerance times presented cover exposures lasting from 20 minutes to more than 4 hours. Major difficulties in maintaining control of activity, food and water intake, and the like, during longer periods seriously limit the reliability of data from more extended studies (eg, Project CANE [Combined Arms in a Nuclear Environment][60]). Thus the factors leading to heat illness cannot be as clearly delineated in historical events, such as the military medical reports and afteraction reports that follow, as they have been in controlled clinical investigations.

Whayne's Analyses of Military Medical Reports

The military medical reports of heat casualties incurred during various wars provide, at best, a very limited look at the true effects of heat on military operations. There are few or no reliable data prior to the 1800s, and the data reported even in the latest large-scale military operation in the heat, the Persian Gulf War (1990/91), appear to be subject to the same problem as earlier data: failures in differential diagnosis of heat illness among heat exhaustion collapse, heatstroke, and "ill-defined effects of heat" confound the data. An even more serious problem is that the number of cases of heat illness treated at a medical treatment facility, and thus recorded, probably under-represents by at least 3-fold the number of soldiers who were treated at forward aid stations or in the field.[61]

On 9 June 1951, Colonel Tom F. Whayne delivered a lecture entitled "History of Heat Trauma as a War Experience" as part of the Medical Service Officer Basic Course at the Army Medical Service Graduate School, Army Medical Center (now Walter Reed Army Medical Center), Washington, DC. In his lecture, Colonel Whayne analyzes the available medical information on the effects of heat on military operations of US fighting forces from the Revolutionary War to the end of World War II. The text of the lecture has been available only as a mimeographed handout distributed to a relatively few students, or bound, with other lectures delivered during the course, in a volume housed in the main library at Walter Reed Army Institute of Medicine, Washington, DC.[62] The editors of this textbook believe that the Whayne lecture deserves a wider readership; it is reprinted in its entirety as Appendix 1 to Volume 3 of *Medical Aspects of Harsh Environments*.

Readers should keep in mind, however, that medical reports are unable to portray even the major losses of military effectiveness that occurred among troops who did not receive, or may not have needed, treatment for heat problems. Whayne presents numerous tables and graphs to bolster his analyses and repeatedly makes two points: (1) the races respond differently to heat, and (2) the effects of heat on soldiers are not severe enough to compromise the particular military operations selected. A few relevant excerpts from the Whayne lecture follow.

American Civil War

> [Whayne's] Figure 3 presents the admission rates for sunstroke per thousand mean strength per year for white and colored troops of the United States Army (Union forces). For the year 1861, only the months of May and June were recorded but these were calculated as an annual rate. Even when reduced to the proper proportions, the admission rate for that year was higher than for the two succeeding years … probably a reflection of a lack of acclimatization or physical conditioning and, unquestionably, to the added stress of combat during hot weather. The rates for 1862 and 1863 are appreciably lower and are probably to be expected for a seasoned army operating for the most part in Maryland, Virginia and Pennsylvania. The higher rate for 1864 is probably associated with penetration of Northern forces further to the South. …
>
> Data was recorded on admissions for the colored troops only from 1864. … [T]he colored troop rate was lower than for the white troops … a phenomenon that appears … in all of the wars in which American forces took part, with the possible exception of World War II.
>
> As one considers the magnitude of these admission rates, however, it is obvious that "sunstroke" was not a medical or tactical problem of great significance. … The maximum rate recorded (1864) is only approximately four per thousand per year and could have had little influence in the overall prosecution of the war if, indeed, these data represent the true picture. The annual admission rate for the total period was 3.1, which included both white and colored troops.[63(p7)]

Spanish–American War

> [H]eat trauma came under the diagnosis of "heatstroke."…
>
> . . . .

The total number of cases, white and colored, for the entire Army was only 748. The total number of deaths was nine. The admission rate for 1898 was 3.68, and for 1899, 1.93 or a total for the two years of 2.95. No deaths occurred in 1899, but for 1898 the case fatality rate was 1.65. …

For the white troops, the rates per thousand per year were 3.79 for 1898 and 2.04 for 1899, while for the colored troops these rates were 1.62 and 0.43 respectively. When the case fatality rate is calculated for the year 1898, however, the white rate was 1.5 as compared to the colored rate of 8.3. … As for the Civil War, it is again demonstrated that within the limitations of the diagnosis for heat stroke, heat trauma was not of great medical or tactical importance.

In neither the Civil War nor the Spanish–American War have we any estimate as to mild casualties from the effects of heat, or of the role of high temperature in lowering efficiency and physical and mental effectiveness in such a way as to interfere with optimum military performance.[63(pp7–11)]

World War I

Effects of "excessive heat" presumably covered the total range of heat injury. … The data … is more complete and its analysis provides some estimate of the cause of the malady. [There were 3,623 cases of "excessive heat" among white enlisted men, a rate of 1.00/1,000/y, with 3 deaths, giving a case fatality rate of 0.08; for black troops, there were 210 cases, for a rate of 0.73/1,000/y, with a case fatality rate of 0.475.—R.F.G.]

. . . .

[T]he relationship between the admission rates and case fatality rates already commented upon holds true in World War I. Whereas white admission rates were about one, colored admission rates were 0.73. Conversely, the white case fatality rate was 0.08, whereas the colored case fatality rate was 0.475. Here again, we note a ratio of approximately one white death to six colored deaths on a percentage basis. [Of the total 3,880 cases of excessive heat, 3,200 of them occurred in the United States (the Zone of the Interior), which pointedly illustrates heat injury as a Zone of the Interior malady.—R.F.G.]

. . . .

Total days lost from disease or injury is a measure of its military cost. During World War I in the total Army ... including officers and enlisted men—there were 31,532 days lost. For white enlisted men, days lost were 28,093; for colored, 2,166. … Among the enlisted men in the U.S., 22,107 days were lost by white enlisted men and 1,863 by colored …, again demonstrating the prevalence of heat injury in the Zone of the Interior. The over-all non-effective rate from heat injury for the total war period was 0.02. Days lost per case … for white enlisted men [was] 7.75; and for colored enlisted men, 10.3. … [T]he time lost per case for colored soldiers is consistently longer than for white soldiers and may corroborate the impression gained from case fatality rates that the severity of the injury in colored men affected may be greater. …

[A]dmission rates [including World War II] as between troops in the United States and overseas … [show] that, except for the Civil War, the over-all admission rates for the total war period in the United States are reasonably close. [They were 2.96 for the Civil War, 1.63 for the Spanish–American War, 1.61 for World War I, and 1.85 for World War II.—R.F.G.]

Based on available data, heat injury in the United States Army was not an important cause for loss of time and had no great medical or tactical significance up to the time of World War II. … [E]xcept for the Spanish–American War, admission rates were relatively high in the Zone of the Interior and low … overseas.[63(pp11–13)]

World War II

Three categories—heat exhaustion, heatstroke, and ill-defined effects of heat—were used for all heat injuries during World War II.

The year 1940—a … peacetime year prior to World War II—shows annual heat injury admission rates per thousand for the Army in the United States of 0.5, … in Panama, 1.4, and … in the Philippines, 1.3. Line officers believed in "water discipline" and … that the drinking of water during work in the heat was harmful. …

. . . .

[I]n some of the training in the tropics prior to the war, men were required to wear wool shirts because it was thought that they were cooler than cotton. …

There was a desperate urgency to train men for operations … in many of the hottest regions on earth, … about which opinion and tradition had fostered the impression that the white man could not endure. …

. . . .

[H]ow the American soldier … performed … is, he did well after he had learned several fundamental rules and after those who were responsible for his

training … [understood] the proper corrective measures: … acclimatization, proper use of water, proper intake of salt, control of activity, physical fitness, adequate sleep and rest, adequate nutrition, proper clothing, previous and associated illnesses, and appropriate education.

[D]efects of these data … are apparent from the records.

1. We have data only on cases severe enough to be admitted to a medical installation. These are inadequate indices … because they fail … to record unknown numbers of men who were not sufficiently incapacitated to report to sick call, but were not well enough to perform their duties efficiently. Some of these men were confined to quarters, others stuck it out, while others, because of injury or intercurrent disease, were carded for these causes, and mild heat injury was not recorded. …

2. Criteria for diagnosing heat injury were not generally … understood. …

3. Heat casualty has a seasonal incidence; … calculation of annual admission rates, which includes the cool months, may not reflect the magnitude of the problem.

4. Study of heat injuries on the basis of theaters, rather than on the cost … in small units, gives a false picture of the potentialities of heat trauma as a tactical military problem.

Figure 10 shows admissions and admission rates for effects of heat other than burns and sunburn in the United States Army. …

Heat casualties in WW II were more frequent in the United States than in any other theater, and casualties were most frequent among men who were overweight or obese, in the older age groups, and for those whose service in the Army had been of short duration. … While the highest rate was reached in 1942, proportionately there was a greater number of cases for the year 1943 when training activities were the heaviest. … [63(pp13–15)]

. . . .

Heat casualties in this country begin to be a problem in May and slacken off … by October. [In my experience, whether dealing with hazardous-waste site workers, football players, firefighters, the military, or civilian populations, the first few weeks of exposure to hot conditions generate the majority of heat problems, especially the initial few days following the first day of a heat wave.—R.F.G.][63(p17)]

. . . .

It was in the desert area and more specifically, in the Persian Gulf Command … that most of the overseas heat casualties occurred. The annual admission rate for 1943 reached 20.78 per thousand. The total case load was 1,102 and in July and August of that year the incidence rates reached 57.26 per thousand per year and 88.58 per thousand per year, respectively. This was indeed a medical problem of some significance and a potentially hazardous military tactical problem. In 1944, a striking decrease occurred. The annual rate per 1000 mean strength was 4.03; the highest monthly rate was 26.73 for July. The total case load for the year was only 183 cases.

This remarkable decrease … was achieved even though the summer of 1944 was as hot as the summer of 1943, and even though the Command broke all records for moving supplies. The experience of the Persian Gulf Command was especially important, since it was the hottest theater in which American troops functioned for long periods, and it was a dry heat rather than a humid heat.

It was the final consensus of the Command that once proper living conditions were instituted, proper working hours adhered to, and troops

FIGURE 10 [FROM WHAYNE LECTURE]

ADMISSIONS FOR EFFECTS OF HEAT (OTHER THAN BURNS AND SUNBURN) IN THE US ARMY BY NATURE OF TRAUMATISM AND BY AREA, 4-YEAR PERIOD, 1942–1945

Nature of Traumatism	Total Army	United States	Overseas
		(Number)	
Heat Exhaustion	18,128	12,151	5,977
Heatstroke	1,676	1,315	361
Other Effects of Heat	15,558	13,814	1,744
Total Effects of Heat	35,362	27,280	8,082
Annual rate per 1,000 mean strength:			
Heat Exhaustion	.71	.82	.56
Heatstroke	.07	.09	.03
Other Effects of Heat	.61	.94	.16
Total Effects of Heat	1.39	1.85	.75

Reprinted from Whayne TF. History of heat trauma as a war experience. Lecture delivered 9 June 1951. In: US Army Medical Service Graduate School. *Notes: Medical Service Company Officer Course 8-0-1 (b)*. Vol 2. Washington, DC: US Army Medical Service Graduate School; 1951: 15. Walter Reed Army Institute of Medicine, Washington, DC: Library call number RC971/.U5/v.2.

> handled in a way most likely to protect them from heat injury, then effective and efficient work could be maintained without significant injury from heat in spite of the very hot summers. This experience was duplicated in [China, Burma, and India,] a hot, humid region.[63(p25)]

Military medical reports from other nations, as well as from subsequent US wars, add little to the information above, which Colonel Whayne so superbly detailed in his lecture.

Afteraction Reports

Another source of information is the ex post facto reviews of various military activities. Some were written by participants, others by concerned reviewers trying to explain or understand what happened, and why. In modern terminology, these are called "afteraction reports." They are usually far less detailed than the type of analysis presented by Colonel Whayne, but highlight the impressions of participants or narrators who wrote shortly after the event.

American Revolutionary War

One of the earliest American military experiences indicated an effect of heat on military operations. British infantrymen, wearing their tall, heavy, black headgear and carrying full packs on a hot summer day, attempted to take a field fortification, erected on top of a hill, by the direct assault common to that era: advancing in line to a drumbeat, pausing only to level and fire their heavy muskets. The advance was broken three times by the American militiamen holding the position, despite frantic efforts of the British officers to steady the troops, who were carrying 80 pounds, uphill, on a hot June day. After General Gage ordered the British to doff their helmets and packs, they drove the Americans off. The extent to which relief from heat stress, versus the Americans' running out of ammunition, contributed to the British taking Bunker Hill in Boston, Massachusetts, cannot be determined, but this unusual order from a British general officer in that era certainly reflects his estimate of the effects of heat trauma on his troops. The attackers lost 1,054 officers and men out of a total of 2,500; the defenders lost 441 in killed, wounded, and prisoners.

War of 1812: Napoleon in Russia

The War of 1812–1813 took a terrible toll on Napoleon's forces in the Russian campaign. Almost all military historians are aware of the effects of the severe cold, as reported by his chief surgeon, Larrey (only about 10,000 of the 100,000 troops who left Moscow reached the Niemen River en route back to France).[64] Far fewer are aware of Larrey's reports on the effects of the unseasonably warm weather during the advance to Moscow with about 250,000 men. Napoleon began his advance into Russia when he crossed the Niemen River, the boundary between the French and Russian empires, on 24 June 1812. By the end of the first month of the campaign, after winning several minor battles and detaching more than 100,000 troops to guard his return line, Napoleon's army had lost well over half its men from heat, drought, lack of food, desertion, and disease—although the significant fighting (at the great battles of Smolensk and Borodino) was still to come. On 28 July Napoleon halted the remaining 175,000 troops near Vitebsk, a 20-day march from Moscow, and waited for Czar Alexander to sue for peace. The weather turned "stifling hot" and the exhausted, half-starved troops lacked water, fighting each other to drink from muddy puddles during the 20-day wait. Then, as Napoleon moved on to Smolensk with only 145,000 troops left, vodka contributed to the problems of young soldiers, weakened by hunger, heat, and fatigue, with only limited amounts of muddy water to drink. Arriving in Viazma on 28 August, the men continued to fight over water in mud puddles. Napoleon reached Borodino on 15 September with only 130,000 men.[64,65]

As is always the case with battles fought earlier than the 20th century, it is difficult or impossible to sort troop losses according to battle, disease, heat or cold, desertion or detachment; nevertheless, it is clear that heat, and especially the lack of drinking water, was a problem. Complicating the problem was, doubtless, the reduction in French recruiting standards, as continuing wars reduced the available supply of sturdy farmers and increased recruitment from the cities. The minimum standard height for the French military was 5 feet 5 inches in 1776, and 5 feet 4 inches in 1792, but was reduced to just under 5 feet in 1813.[66] This reduction in fitness may have been part of the basis for Napoleon's dictum that the essentials of the fighting man comprise his arms and ammunition, trenching tool, knapsack, and 4 days' rations[67]; note that the water bottle (canteen) was not included, although the British had called for one beginning in about 1655 for campaigns in the West Indies.[68]

American Civil War (1861–1864)

> [1861.] On the morning of July 21st, ... we halted in the shade, as the day, even thus early, promised to

> be one of the hottest of the season. While observing the troops passing … I perceived that our troops marched at double quick, and some at full run, while many, overcome by the heat, threw away their blankets and haversacks. I expressed my opinion to the General, that owing to their rapid movement, the men would be exhausted before they arrived on the scene of action. … [H]e directed the men not to run; but, as the Officers behind … constantly repeated the command to close up, the troops were kept at the run a great part of the way. The weather was excessively hot, and, as one of the causes of the Bull Run failure, I desire to express my belief that the exhaustion of our forces, by the long and forced march, contributed as much as anything else to the disasters of the day. … [O]n several occasions where our men faltered … or did not pursue an advantage … it was manifestly owing to complete exhaustion rather than any want of spirit or courage … [T]heir failure … was from inability for further exertion.[69(p3)]

> [1864.] During a march many cases of sunstroke come under the hands of the regimental surgeons. I have seen about forty cases of different grades of severity, from slight dizziness, with inability to walk straight, to violent … convulsions and almost immediate death. … Cases of heat apoplexy have also occurred during marches on hot sultry summer nights.[70(p199)]

> [1864. On 26 June,] a very large number … of men in the command judged by the Medical Officers incapable of performing a forced march … were sent to hospital. … At four o'clock PM … [t]he Corps was pushed on so rapidly that the twenty ambulances following each division were very speedily filled with exhausted men, and straggling took place by the roadside to a far greater extent than is usual even in day marches, when hot sun combines with fatigue to overcome the men.[71(p187)]

> [1864. On 25 July, t]he day was oppressively hot, so much so, that although the men had only marched a couple of miles, a very large number were utterly exhausted … . Many were insensible; some in convulsions; four I saw dead. … [A]bout an hour and a half elapsed before all the cases of sunstroke could be carried to the rear.[72(p188)]

Official and Anecdotal Reports

Crimean War

During the Crimean War, attention focused on the heart and lung lesions that resulted from the combination of heavy loads being carried and the shift from sturdy yeomen to less physically fit urban recruits. The load carried by French troops was reduced by 12 pounds after the war because of the "high incidence of emphysema of the lungs."[73(p8)] A British Royal Commission also addressed these issues, pointing out that it was not just the net exertion, but the fact that the exertions are maintained, not with open necks and rolled sleeves, nor in specially adapted costume like the sportsman, but at the utmost possible disadvantage as regards the weight carried and the entire arrangement of dress and equipment.[74(p7)]

World War I

The huge load carried during World War I was also linked to cardiac overstrain in soldiers, and in particular in Macedonia where men weakened by malaria and from marching in irregular, mountainous terrain developed heart lesions to such an extent that the cumulative effects of marching while weakened by malaria were studied in the field. During 4 weeks of marching, some 9% of the troops engaged became heat casualties, but of the individuals afflicted with malaria, no less than 22% were afflicted with heat effects; thus, although they formed less than 11% of the total personnel involved, the men with malaria contributed 25% of the total casualties.[75(p9)] Lothian, writing in 1921, reports that

> apart from actual acute heat effects, such as heatstroke and derangement of the heat regulating system, such hurtful conditions may cause a lot of inefficiency not perhaps so noticeable at the time, but tending to cumulative injury as well as immediate exhaustion.[75(p9)]

Arab–Israeli War (1956)

Perhaps the most numerous loss of troops during a 24-hour period from the effects of heat on military operations was experienced by the Egyptians on 2 November, the sixth day of the Arab–Israeli War of 1956. A new Egyptian commander reached the 2,500- to 3,000-man garrison holding the two main ridges east of Abu Agueila in the Sinai just before it was surrounded by Israeli forces. Cut off from food and water, the commander announced that it was every man for himself. Starting that night, the Egyptians fled into the desert in a vain attempt to escape to El Arish, 52 miles across the desert dunes. The number of deaths from heat and dehydration, rather than from the knives of Bedouins seeking loot, is undocumented, but only about 700 Egyptians were eventually captured by the Israelis.[76(p139)]

The British in Kuwait: Operation Vantage

A nonclassified summary of a report detailed the health and efficiency of troops on "Operation Vantage," a 1961 British expedition into Kuwait. Despite humidities of 6% to 11%, the WBGT index between the hours of 1130 and 1430 approximated 31°C, with an ambient temperature of 45°C and globe temperatures from 50°C to 55°C; inside armored vehicles the conditions were considerably worse (WBGT to 38°C, T_a to 50°C with peaks to 70°C, and wet bulb temperatures in the 30°C range). There were 132 heat casualties treated at a base hospital, indicative of a total of approximately 400 to 500 heat casualties during the first 47 days; the maximum number of heat casualties reached the base hospital during the first 5 days of the expedition. Water requirements averaged 4 to 6 gallons per day, or about 125 tons of water per day for 5,000 troops.[61]

Vietnam War

A letter from Vietnam in May 1970 shows the continuing effects of heat on military operations:

> Along the Cambodian border troop units are being relocated and shuffled to new areas; often without any or enough canteens. This battalion had been moved a couple of days earlier from their old base in the delta where they had been fighting in relatively easy rice paddies (dry season) and were now required to hack their way through thick jungle with machetes, which made a big difference in terms of the energy required. Short (ie, 1–3 day) patrols need continuous air re-supply, especially water, but air assets are in critical supply and working continuously. Yet in the space of a couple of hours I saw 5 men of one squad dusted off back to the relocated base camp and several others from that one company dusted off for heat casualties—all within the space of a couple hours on May 6th. The temperature was hotter than anything I'd ever experienced outside of a foundry or hay mow; in the adjacent FSB [fire support base] someone reported 126°F. As I departed, I heard there were more heat casualties on the way in and I was worried about their supply of intravenous fluids.
>
> These heat casualties were promptly cared for by the competent medics and doctor by fanning, I.V.s, oral fluids and rest. About [one third] had *severe* cramps and [two thirds] had severe weakness, palpitations, fever and near collapse; the latter's skin was hot and dry and red but no temps were taken.
>
> So I can personally confirm that heat stress influenced this mission (company sized patrol, grunts only) and may have prevented its accomplishment. I will attempt to get some information that is better than this anecdotal material.[77]

EFFECTS OF DEHYDRATION ON MILITARY OPERATIONS

US Army, Texas, 1877

One of the best descriptions of severe dehydration in troops appears in the report of Company A, Tenth Cavalry, which lost its way and spent 3.5 days without water during July 1877, in an arid area of Texas 140 miles from Fort Concho. The terrain—dry soil with an occasional, stunted, mesquite bush—offered no shelter from the sun, and the heat was excessive. Company A started at noon with only a full canteen each. The next day, *coup de soleil* had prostrated two men and all were suffering from the lack of water; many were faint and exhausted, with some falling from their saddles. The second day, the captain decided to return to base, supposedly some 75 to 100 miles away. Marching in the midday heat, the men's mouths were so dry that they could not tell if they had anything in them. Their tongues were swollen; brown sugar would not dissolve in their mouths, and they could not swallow it.

> Vertigo and dimness of vision affected all; they had difficulty in speaking, voices weak and strange sounding. … [T]hey were also very feeble and had a tottering gait. Many were delirious.[78(p195)]

As their horses died, the men drank the blood. They also drank their own urine, sweetening it with sugar. They were oppressed with dyspnea and a feeling of suffocation, but they breathed as little as possible and through the nose, with closed lips

> covered with a whitish, dry froth. … Their fingers and palms looked shriveled and pale; some who had removed their boots suffered from swollen feet and legs.[78(p195)]

The third day, part of the unit reached base camp:

> both officers and men were almost helpless … and the … water did not greatly benefit any of them this day.[78(p196)]

A few men set out with extra canteens to backtrack and find stragglers and those who had been sent to

find water. Fortunately, the next morning another unit with Indian scouts arrived at the base camp and helped in the rescue. The sufferers had an irresistible desire to drink, but their stomachs would not retain water; it was vomited up, as was food.[78]

Command and Control

As early as 1912 it had been suggested that even when water is readily available, troops working in the heat tend not to replace water as quickly as it is lost. In 1947, Adolph and his colleagues[79] termed this deficit "voluntary dehydration." The greatest deficits occur on the first active day in the heat, particularly during the first 12 hours. As shown by a study done in the United Kingdom in 1968,[80] the problem is universal in nature, with the same temporal issues: the 12-hour debts (as a percentage of initial body weight) on the first active day in the heat were 2.4% in Tripolitania, 2.6% in Swaziland, and 3.4% in Bahrain, but only 0.8% in Malaya. After 24 hours these deficits were 0.9% in Tripolitania, 1.7% in Swaziland, 3.4% in Bahrain, and 1.6% in Malaya. The deficits were almost completely abolished after 48 hours. Part of the problem may be the difficult logistical challenge of supplying adequate water to troops in the field: approximately 4 gallons per man per day in Kuwait[61]; the same amount was also needed in Singapore.[81]

Dehydration, expressed as the percentage of loss from initial, fully hydrated body weight, is a major contributor to problems during military operations in the heat. Beyond a level of about 2% dehydration, the rate of body temperature rise is accelerated with each additional percentage of dehydration, although the final temperature reached may not be very different.[82] Emphasis on "water discipline" (ie, training troops to perform in hot conditions with ever-decreasing ingestion of water) has proven to be a serious mistake. The United States,[83] the United Kingdom,[84] Israel,[85] and South Africa[86] have done independent but essentially equivalent studies of men working in the heat. Troop units, assigned to complete an approximately 10-mile march across hot, desert terrain, were split into three groups, based on their water intake:

- one third were allotted just one or two canteens for the mission;
- another third were given two canteens at the start, which would be refilled whenever requested during the march; and
- the remaining third were weighed on a scale before the march and during each rest break, and were required to ingest enough water at each break to return to their premarch body weight.

The results were the same in all these countries' studies: few who had only two canteens of water to drink were able to complete the march, most of those with ad libitum access to water failed to avoid dehydration and had difficulty completing the march, and those whose hydration was maintained at original levels by command control had minimal problems. The conclusion in all these studies was that thirst is an inadequate stimulus to maintain full hydration, and maintenance of normal body water levels is a major factor in reducing heat stress.

Note that the average soldier has only about 5 liters of blood, and that this circulatory system fluid must

1. transfer oxygen from the lungs to the working muscles, brain, and other vital organs;
2. transfer heat from the working muscles to the skin; and
3. at least initially, provide all the water used as sweat for evaporative cooling, at sustainable rates of 1 L/h, and short-term rates up to 3 L/h.

The need to provide adequate quantities of drinking water, and to reinforce its intake by making it palatable (slightly acidic) and cool (about 70°F), as well as by command control to ensure adequate intakes, must be a hallmark in prevention of heat illness. It is essential to solve the logistical problems of delivering 125 tons of water per 5,000 man-units per day, and to solve the problems of troops trying to ingest at least 1 L/h while wearing protective respirators. Dehydration by 5%, a shortfall of only 3.5 liters of water for the average 70-kg man, is considered a limit to mission effectiveness. Doctrine published as recently as 1942 in Field Manual 31-25, *Desert Operations*, stated:

> Restricted water consumption must become a habit. Training must condition troops to live on a limited water ration and must develop such self-discipline in the use of water as will assure the maintenance of combat efficiency on the limited water supply available.[87]

This concept of water discipline *does not work*. However, the doctrine in this field manual against drinking alcoholic beverages is still valid.

The foregoing is not to deny that thirst can be a major problem in the heat, but that thirst, in itself, is not necessarily an adequate stimulus to avoid de-

hydration. As the explorer Hedin wrote of his experience crossing a desert in 1899, "the first few days the tortures of thirst are so poignant that you are on the brink of losing your senses."[88(p228)] MacDougal[89] reported that one day's deprivation of water so disrupted men's mental balance that although suffering from thirst, these men forded waist-deep streams to continue on and die in the desert. Larrey, describing Napoleon's 1798 campaigns into Egypt and Syria, reported that

> [w]ithout food or water the army corps entered the dry deserts bordering Libya, and arrived only with the greatest difficulty on the fourth day of the march at the first place in Egypt which offered supplies. Never has an army undergone such great vicissitudes or such painful privations. Struck by the burning rays of the sun, marching always afoot on still more burning sand, … the most vigorous soldiers, consumed by thirst and overwhelmed by heat, succumbed under the weight of their arms.[64]

However, it is difficult to recognize other adverse effects of heat on Napoleon's military operation, which was considered a successful campaign.

CONCLUSIONS

Viewed from the numbers and nature of heat injury reported in the military medical literature, it appears that the major effect of heat on large-scale military operations is the need to supply such large quantities of potable water to troops engaged in active operations under hot conditions (up to 125 tons of water per day per 5,000 men)[61]; this may compete with delivery of other essential military supplies. Other effects of heat on individual units can prevent them from accomplishing their assigned missions. However, as suggested by Napoleon's campaigns in Egypt, effects of "heat stress" on large-scale operations are difficult to identify, particularly given the vastly improved capabilities of modern military communications and other command and control capabilities. Any such heat stress effects tend to be diffused and have limited impact during operations by larger than company-sized military units. Rotation of reserves or replacement of units seriously degraded by heat appear to allow large-scale military operations to continue with only barely detectable effects of heat.

When ample supplies of drinking water are available, the real impact of heat on military operations is on the effectiveness of unseasoned units (ie, units during their first 3–5 days in country) with limited manpower reserves, who are attempting to carry out missions that involve sustained, moderate-to-heavy levels of activity under operational conditions where such "agents of heat" are present as high temperature, high humidity, low air motion, heavy loads, protective clothing, or personnel protective items such as body armor, chemical–biological warfare masks, and so forth.

Many afteraction reports document the adverse impact of heat on military operations. The reports included in this chapter suggest these effects, as do books by military historians such as *The Soldier's Load and the Mobility of a Nation*, by S. L. A. Marshall.[28] However, Ogburn's *The Marauders*[90] (which reports on the World War II operation by American troops in the China–Burma–India theater, who were led by Merrill and cut off behind Japanese lines in Burma) indicates what well-led, motivated troops can accomplish despite heat, difficult terrain, and lack of resupply. This supports the World War II conclusions reported by Whayne on the extent to which leadership can overcome the adverse effects of heat on military operations, even under severe heat stress.

Because the effects of environmental heat are dramatically amplified by increasing activity, heat favors defensive over offensive operations. Any requirement for wearing body armor, chemical-protective ensembles, or both, must consider their effects in increasing heat stress. However, heat has usually had little medical or tactical significance on the overall outcome of military operations; the primary adverse effects of heat on military operations are at the small-unit level. Such effects can be avoided to a great extent by proper doctrine, training, and leadership. The Israeli Army policy that heat illness is a court martial offense—not for the heat casualty but for the unit leader—provides an insightful comment on the occurrence of heat illness during military operations.

Now that we have previewed heat illness—both as it affects military operations and in terms of the classic epidemiological triad (agent, disease, and host factors)—the stage is set for the problem to be considered in greater depth in the ensuing chapters.

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Chapter 1: ATTACHMENT

THE ORIGINS OF THE US ARMY RESEARCH INSTITUTE OF ENVIRONMENTAL MEDICINE AND ITS RESEARCH ON EXTREME ENVIRONMENTS

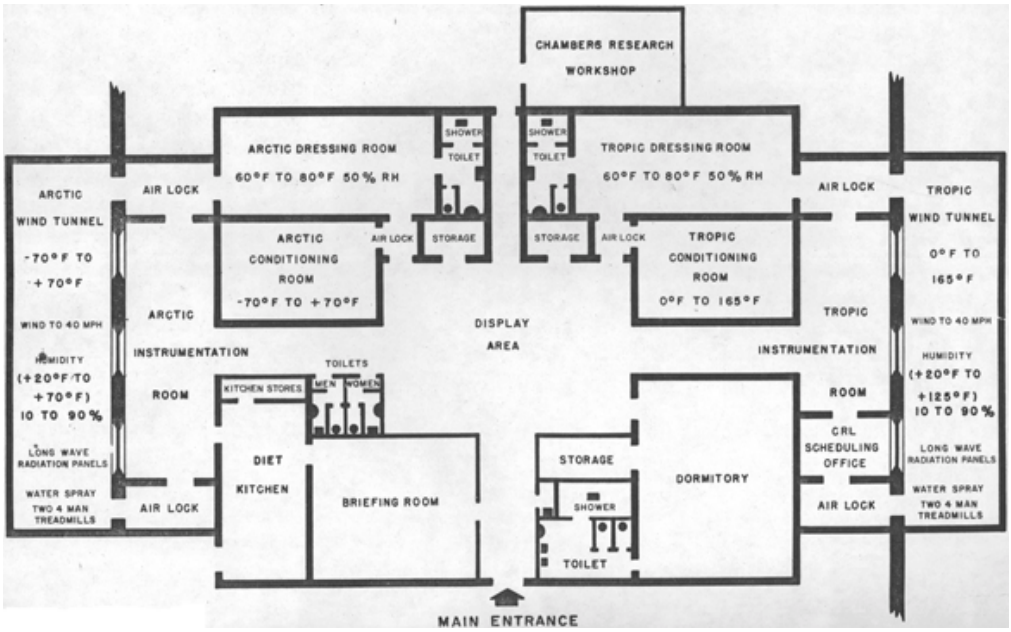
Until World War II there was little government interest or support in the United States for environmental research on the effects of heat, cold, or altitude. Then these effects became of critical government concern, with US military pilots flying high-altitude missions; tank crews fighting in the desert; infantry living and fighting in the jungle; the mountain division in Northern Italy; troops in winter in Alaska to repel Japanese attacks on the Aleutian Island chain; and naval crews sailing in tropical seas trying to work in confined gun turrets, boiler rooms, and the like. Fortunately, the Harvard Fatigue Laboratory had been set up as an industry-funded, nonprofit laboratory in 1927 to study fatigue and discomfort of workers, and ways to improve productivity. Conceived as part of Harvard Medical School, its first 3 years were spent establishing normal physiological values for healthy adult workmen. These data provided baselines for studies of the effects of environment on workers, but the subsequent "applied" work was relegated to a basement of the Harvard Business School, where some rooms were set aside for the laboratory. The Chairman of the Board of the Harvard Fatigue Laboratory, L. J. Henderson, was renowned for his studies on respiratory physiology; one focus of the laboratory was on Peruvian altitude studies. Another was on heat stress: in Panama in 1931; in sharecroppers in Mississippi in 1939; and throughout the construction of Boulder Dam in the Nevada desert. Researchers associated with the Harvard Fatigue Laboratory before World War II included such luminaries as Beane, Brouha, Belding, Darling, Dill (who published his monograph on "Life, Heat and Altitude" there), Folke, Forbes, Graybiel, Horvath, Johnson, Robinson, Talbott, Turrell, Sargent, and Scholander. Frank Consolazio, who later headed a Surgeon General nutrition group, started as a technician at the Harvard Fatigue Laboratory.

Attachment Fig. 1. Left to right: James Bogart, a technician in the Physiology Branch of the Military Ergonomics Division; the water "immersion" (nose only) manikin; Harwood (Woody) Belding, PhD, Professor at the University of Pittsburgh (Pennsylvania) School of Public Health, the inventor of these manikins; his manikin, Chauncy; Ralph F. Goldman, PhD, Founder and Director of the Military Ergonomics Division, who conceived of the cotton skin to make a "sweating" manikin, and also conceived of the need for a walking manikin to study the effects of the "pumping" of clothing by body motion; a "sweating" copper manikin; J. Robert Breckenridge, MS, head of the Biophysics Branch of the Military Ergonomics Division of the US Army Research Institute of Environmental Medicine (USARIEM), Natick, Mass. This photograph was taken in 1972, on the occasion of USARIEM's purchase of Belding's manikin, Chauncy; the other two manikins, which had been used at Wright Patterson Air Force Base, were on permanent loan from the US Air Force.

By 1942, the US military had set up a number of laboratories to deal with the problems of environmental extremes. Researchers from the Harvard Fatigue Laboratory, many of them in uniform, provided the cadre for these laboratories. The US Navy set up a laboratory at Pensacola, Florida, and the Army Air Corps set up the Aero-Medical Research Laboratory near Dayton, Ohio, at what became Wright-Patterson Air Base during the war. The Armored Medical Research Laboratory was established at Fort Knox, Kentucky, by the Combat Armored Divisions to study the specific problems of that combat arm, with Dr William Beane as its commander. There was no Medical Research and Development Command (until 1958), so medical and medical service corps officers were detailed to the Fort Knox laboratory. Doctors Ashe, Eichna, Horvath, Keats, and Shelley carried out classic studies on heat effects and acclimatization, perhaps stimulated by stories that German General Erwin Rommel, who was dominating tank warfare in North Africa, had done studies on tank crewmen in a climate chamber in Germany in the early 1930s.

The US Army Quartermaster General, recognizing the problems of food, personal equipment, clothing, and particularly cold weather protection (as a result of the Aleutian campaign in Alaska, where far more members of the US Army's 7th Division were injured by cold than by the enemy), established the Climatic Research Laboratory (CRL). Sited in Lawrence, Massachusetts, where Pacific Mills (a manufacturer of woolen goods) had a very large cold chamber capable of producing temperatures of –112°C, CRL's first commander was Colonel John Talbott; its first scientific director was Harwood Belding, PhD. The copper manikin that Belding developed in 1939 proved to be a key tool in studies of clothing insulation; it allowed precise measurement of clo, the unit of measurement for clothing insulation developed by A. P. Gagge,[1] and served as a prototype for similar manikins for the US Army Air Corps and the US

Attachment Fig. 2. From right to left, Mr Gerald Newcomb; R. F. Goldman, PhD; Captain John M. Witherspoon, Medical Corps, US Army, a physician assigned as medical monitor for this study; and eight test subjects wearing different rain wear, which varied in coverage, air permeability, and design. The test subject seated in the foreground at position 5 (arrow), wearing the full-coverage, impermeable, hooded rain parka, experienced the least heat stress while walking in this garment. This unanticipated, counterintuitive finding was one of the key factors leading to the development of the "pumping coefficient" for clothing and to the subsequent construction of a walking manikin. Photograph: US Army Natick Laboratories, Natick, Mass. Negative 66-6-15-218-2.



Attachment Fig. 3. Floor plan of the Climatic Research Laboratory, US Army Natick Laboratories, Natick, Massachusetts, which was occupied in 1954. Source: US Army Quartermaster Research and Development Command, Natick, Mass.

Attachment Fig. 4. Building T24 at Fort Churchill, Ontario, Canada, where scientists from the Quartermaster General's Environmental Protection Research Division (EPRD) at Natick, Massachusetts, measured heat loss during the night under arctic and subarctic conditions.[1] Among many other studies conducted at Fort Churchill was a joint nutrition study in 1956 between the EPRD and the US Army's Medical Nutrition Research Laboratory, Fitzsimons General Hospital, Denver, Colorado. (1) Goldman RF, Brebbia DR, Buskirk ER. *Heat Loss During the Night Under Subarctic Conditions.* Natick, Mass: Environmental Protection Research Division; March 1960. ERPD Technical Report EP-129. Photograph: US Army Natick Laboratories, Natick, Mass. Negative 4328-56.

Navy, as well as for sectional copper hands and feet, which were used in testing hand and footwear. Some 30 years later, when fitted with a "sweating" cotton skin by Goldman, such manikins (Attachment Figure 1) allowed Alan Woodcock's theoretical moisture permeability index (I_m) to be measured for complete clothing ensembles. (Woodcock, a Canadian, was chief of the Biophysics Division at CRL.) By the end of the 20th century, close to 100 manikins, some of which have the "walking" capability established by Goldman[2] as useful in analyzing human heat transfer, serve as fundamental tools to generate data for functional clothing design and modeling of human tolerance limits to heat and cold.

In 1951, CRL moved from Lawrence to a former Veteran's Hospital in Framingham, Massachusetts, pending completion of the US Army Quartermaster Research and Development Command's Natick Laboratories, the cornerstone of which had been laid in 1951. The Korean War maintained the Quartermaster's orientation to cold weather studies, and in late 1954, the group, now renamed the Quartermaster Research and Engineering Command, moved to Natick, Massachusetts. The climatic chamber facilities provided are shown in Attachment Figures 2 and 3. To supplement these superb research laboratory facilities, the group carried out numerous field studies in arctic, desert, and tropical climates, and, after 1968, at high terrestrial elevations. A number of studies were carried out on military operations under subarctic conditions at Fort Churchill on Hudson Bay in Ontario, Canada (Attachment Figure 4). Heat production, heat loss, and body composition changes in the cold were primary topics. A number of nutrition studies were also carried out at Fort Churchill in collaboration with the US Army Nutrition Laboratory based at Fitzsimons General Hospital, Denver, Colorado. However, the discovery of Russia's developments in chemical warfare refocused research on heat, particularly on the heat stress and performance limitations associated with wear of protective cloth-

Attachment Fig. 5. At Fort Lee, Virginia, in 1962, R. F. Goldman, PhD, and Mr Gerald Newcomb are seen with a test subject in one of the earlier studies on the effects of wearing nuclear–biological–chemical (NBC) clothing on military performance capability (see Table 1-2). Dr Howard Hembree of the Fort Lee test group is standing behind Dr Goldman; at far right, Mr Tom Dee of the Environmental Protection Research Division Field Test Division is seen talking with another subject. Photograph: US Army Natick Laboratories, Natick, Mass. Negative P-7876.

Attachment Fig. 6. (**a**) The US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts. (**b**) Edward F. Adolph, PhD (right of center), Professor of Physiology, University of Rochester Medical School, Rochester, New York, the keynote speaker at the 17 October 1968 dedication of the new, 75,000 ft^2 USARIEM building. Others in this photograph are, from left to right, Brigadier General G. F. Gerace, Commanding Officer, US Army Quartermaster Research and Development Command's Natick Laboratories; David E. Bass, PhD, Technical Director, USARIEM; Major General J. Blumberg, Commanding Officer, US Army Medical Research and Development Command; [Dr Adolph]; Colonel James E. Hansen, Medical Corps, US Army, Commanding Officer, USARIEM; and Lieutenant General Austin W. Betts, Commanding General, US Army Research and Development Command. Photograph (b): Courtesy of American Physiological Society, Bethesda, Md.

a

b

Attachment Fig. 7. The additional energy cost required to march on soft sand, found to be a multiplier (terrain coefficient) of 2.1 times the energy cost at the same march rate on a treadmill or blacktop road, is being measured in this study.[1] The military volunteer subjects are shown wearing "Max Planck" gasometers. This 7-lb gas meter measures a timed volume and the temperature of respired air, and collects an aliquot sample (0.3% of each exhalation) in the rubber bag shown hanging from the gasometer. The difference between the oxygen content of ambient air (20.93%) and the residual oxygen content subsequently measured in the sampled expired air, multiplied by the gasometer's measured respiratory volume (L/min, corrected to standard temperature and atmospheric pressure), can be directly converted to kilocalories per minute using the factor of 4.85 kilocalories per liter of oxygen consumed. This photograph helps explain why the multiplier is so high, if one notes the amount of sand lifted with each step and recalls the finding (from an earlier study[2]) that the energy cost of 1 pound of footwear is equivalent to that of 5 pounds carried on the back. (1) Soule RG, Goldman RF. Terrain coefficients for energy cost prediction. *J Appl Physiol*. 1972;32:706–708. (2) Soule RG, Goldman RF. Energy cost of loads carried on the head, hands or feet. *J Appl Physiol*. 1969;27:687–690.

ing (Attachment Figure 5). The US Army Research Institute of Environmental Medicine (USARIEM) played a major role in studies on chemical–biological protective clothing from 1958 to 1980 (see Table 1-7 in Chapter 1, Introduction to Heat-Related Problems in Military Operations).

By the 1960s there were extensive reorganizations to eliminate duplication and combine functions. The Quartermaster General's Environmental Protection Research Division (EPRD) at Natick and The US Army Surgeon General's Armored Medical Research Laboratory (AMRL) at Fort Knox had been competing for the limited research and development funding available during the Cold War. A joint decision was made that The Surgeon General would take responsibility for environmental research and combine its Fort Knox AMRL with the Quartermaster's EPRD into a single organization, USARIEM, which was formally established in 1961. Colonel Don Howie, Medical Corps, US Army, of the Medical Research and Development Command, developed plans for a USARIEM building, to consist of some 75,000 ft^2. Although occupancy had taken place some months earlier, the formal dedication was held on 17 October 1968, with Edward F. Adolph, PhD, one of the world's leaders in heat stress studies in the desert during World War II, serving as the principal speaker (Attachment Figure 6). Former Senator Leverett Saltonstall of Massachusetts, a major supporter of Natick; General William C. Westmoreland; and other dignitaries also attended.

A few years later, to supplement specific Cold, Heat, Altitude, and Exercise Divisions, USARIEM set up a Military Ergonomics Division with a broad-based, five-faceted research program,[3] which is exemplified in Attachment Figure 7:

1. define the soldier's tolerance limits to work, cold, and heat;
2. identify (based on studies by the other divisions) the physiological basis for such limits;
3. assess extension of these limits by physiological or psychological means or both (conditioning, training, acclimatization, nutrition, motivation, etc);
4. assess tolerance extension by improved clothing or equipment, or by redesigning the mission (ie, add manpower, time, work/rest cycles, etc); and
5. predict small unit performance as a function of physical, physiological, psychological, and tactical factors.

This program has been quite useful in supplying design guidance to clothing and equipment developers; guidance for preventive medical officers on thermal and work problems; planning information for tactical and logistics personnel; training information to the combat forces; and recommendations to government and industry for regulation of exposures. While not providing all the answers, this program organized the available information, identified areas in which more research was needed, and helped set relative priorities for military research in the areas of heat, cold, and work.

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Chapter 2

HUMAN ADAPTATION TO HOT ENVIRONMENTS

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INTRODUCTION

Problems due to heat stress may occur whenever the rate of heat production or heat gain from the environment is sufficiently large in relation to the body's ability to dissipate heat. Thus, sustained high-intensity physical exercise; excessive thermal insulation due to body armor or protective clothing; or thermoregulatory impairment due to fever, drugs, or dehydration may create the conditions for heat-impaired performance or heat illness, even during cool weather.

It is difficult to evaluate the effects of heat stress on the health and performance of troops; thus, the overall impact on military operations is probably much greater than generally appreciated. This is so for several reasons. First, heat illness is probably underreported. Second, in an operational setting, cumulative effects of prolonged heat exposure and combined effects of heat and other stresses are likely to be important, but such effects are difficult and costly to reproduce under controlled experimental conditions. Therefore, they have not been the subject of much experimental study. Third, troops exposed to such conditions may not appreciate the extent to which their abilities and performance are affected.

Most of the earth's hot regions are inhabited, and human physiology permits people to live and work successfully in very hot climates provided they are acclimatized (physiologically adjusted to an environment, in nature) to heat, have access to shade and sufficient supplies of potable water, and can limit their physical activity during the heat of the day. However, military operations in a hot climate must confront problems of heat stress that differ substantially from those ordinarily faced by the local inhabitants. Military operations may involve troops who were not acclimatized to heat before their deployment, and local supplies of fresh water may be insufficient for the requirements of a large military force. Moreover, because of the demands of combat or other mission requirements, troops may have to perform physical exercise during the heat of the day, or at levels that exceed established guidelines for prevention of heat casualties. The accompanying threat to the troops' health and effectiveness may be aggravated by a need to perform such exercise when they are at increased risk of heat illness because they are sleep deprived, or do not have free access to drinking water.

IMPORTANCE OF TISSUE TEMPERATURE

Extreme temperatures injure tissue directly. A protein's biological activity depends on the location of electrical charges in the molecule and on its overall configuration. Many physicochemical processes can alter a protein's configuration and charge distribution, and thus change its activity, without affecting the sequence of amino acids. Such alteration of a protein is called *denaturation*; and by inactivating a cell's proteins, denaturation injures or kills the cell. High temperature can denature proteins, and a familiar illustration of this effect is the coagulation of the albumin in the white of a cooked egg. If living tissue is heated, injury occurs at temperatures higher than about 45°C, which is also the temperature at which heating the skin causes pain. The degree of injury depends on both temperature and duration of the heating.[1]

Cold, like heat, can cause direct injury to tissue, although via different mechanisms. As a water-based solution freezes, crystals of pure ice form. Thus all the dissolved substances are left behind in the liquid that has not yet frozen, which becomes more and more concentrated as more ice forms. Freezing damages cells through two mechanisms. First, ice crystals themselves probably disrupt the cell membranes mechanically. Second, the increase in solute concentration of the cytoplasm as ice forms denatures the proteins by removing their water of hydration, by increasing the ionic strength of the cytoplasm, and by other changes in the physicochemical environment in the cytoplasm.

Mammals, including human beings, are *homeotherms*, or warm-blooded animals, and regulate their internal body temperatures within a narrow band near 37°C (Figure 2-1), despite wide variations in environmental temperature. Tissues and cells can tolerate temperatures from just above freezing to nearly 45°C—a range far wider than the limits within which homeotherms regulate body temperature. What biological advantage do homeotherms gain by maintaining such a stable body temperature?

Temperature is a fundamental physicochemical variable that profoundly affects many biological processes, both through specific effects on such specialized functions as electrical properties and fluidity of cell membranes, and through a general effect on most chemical reaction rates. Most reaction rates vary approximately as an exponential function of temperature within the physiological range, and increasing temperature by 10 Centigrade de-

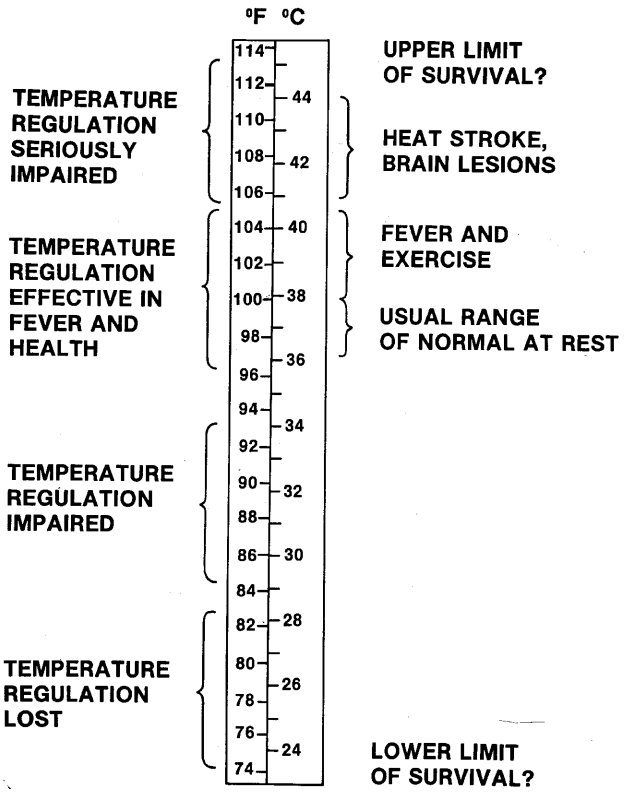

Fig. 2-1. Ranges of rectal temperature found in healthy persons, patients with fever, and persons with impairment or failure of thermoregulation. Reprinted with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA. *Medical Physiology.* Boston, Mass: Little, Brown; 1995: 588

grees increases the reaction rate by a factor of 2 to 3. For any reaction, the ratio of the reaction rates at two temperatures 10 Centigrade degrees apart is called the Q_{10} for that reaction, and the effect of temperature on reaction rate is called the Q_{10} *effect*. The concept of Q_{10} is often generalized to apply to a group of reactions that are thought of as comprising a physiological process because they share a measurable overall effect, such as oxygen consumption. The effect of body temperature on metabolic processes is clinically important in caring for patients with high fevers who are receiving fluid and nutrition intravenously, and an often-used rule states that each Centigrade degree of fever increases a patient's fluid and calorie needs by 13%.[2]

BODY TEMPERATURES AND HEAT TRANSFER IN THE BODY

The body is divided into a warm internal *core* and an outer *shell* (Figure 2-2),[3] the temperature of which is strongly influenced by the environment. Although shell temperature is not regulated within narrow limits the way internal body temperature is, thermoregulatory responses do strongly affect the temperature of the shell, and especially its outermost layer, the skin. The shell's thickness depends on the environment and the need to conserve body heat. In a warm environment, the shell may be less than 1 cm thick; but in a subject conserving heat in a cold environment, it may extend several centimeters below the skin. The internal body temperature that is regulated is the temperature of the vital organs inside the head and trunk, which together with a variable amount of other tissue, comprise the warm internal core.

Although heat is produced throughout the body, it is lost only from tissues that are in contact with the environment, mostly skin and respiratory passages. Because heat flows from warmer regions to cooler regions, the greatest heat flows within the body are those from major sites of heat production to the rest of the body, and from core to skin. Within the body, heat is transported by two means: *conduction* through the tissues; and *convection* by the blood, the process by which flowing blood carries heat from warmer tissues to cooler tissues.

Heat flow by conduction is proportional to the thermal conductivity of the tissues, the change of temperature with distance in the direction of heat flow, and the area (perpendicular to the direction of heat flow) through which the heat flows. As Table 2-1 shows, the tissues are rather poor heat conductors.

Heat flow by convection depends on the rate of blood flow and the temperature difference between the tissue and the blood supplying the tissue. Because the capillaries have thin walls and, taken together, a large total surface area, the capillary beds are the sites at which heat exchange between tissue and blood is most efficient. Because the shell lies between the core and the environment, all heat leaving the body via the skin must first pass through the shell. Thus the shell insulates the core from the environment. In a cool subject, skin blood flow is low, so that core-to-skin heat transfer is dominated by conduction; the subcutaneous fat layer adds to the insulation value of the shell, because it adds to the thickness of the shell and because fat has a conductivity only about 0.4 times that of dermis or muscle. In a warm subject, on the other hand, the

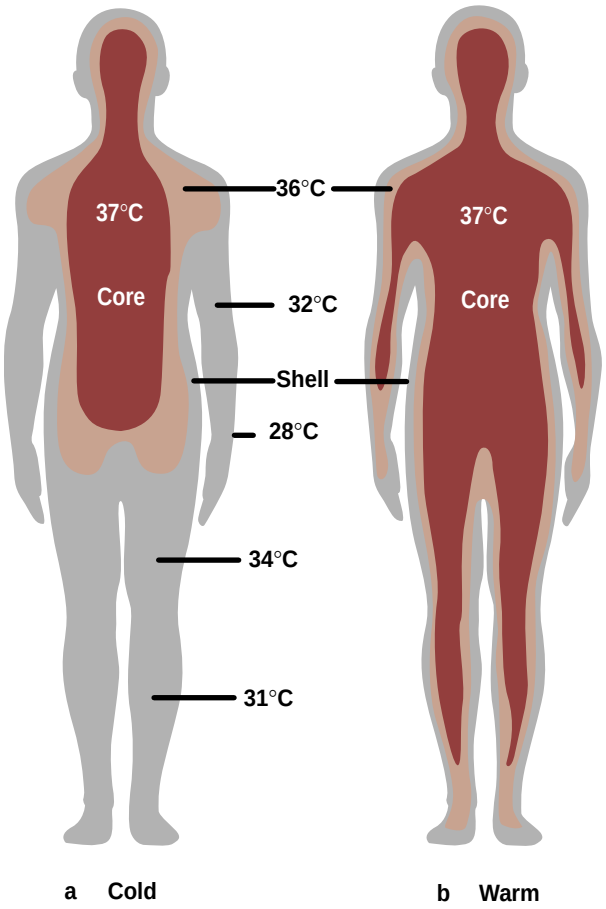

Fig. 2-2. Distribution of temperatures within the body and division of the body into core and shell during exposure to (**a**) cold and (**b**) warm environments. The temperatures of the surface and the thickness of the shell depend on the environmental temperature, so that the shell is thicker in the cold and thinner in the heat. Adapted with permission from Elizondo RS. Regulation of body temperature. In: Rhoades RA, Pflanzer RG, eds. *Human Physiology*. Philadelphia, Pa: Saunders College Publishing; 1989: 823–840.

TABLE 2-1

THERMAL CONDUCTIVITIES AND RATES OF HEAT FLOW*

	Conductivity kcal/(s•m•°C)	Rate of Heat Flow kcal/h	Watts
Copper	0.092	33,120	38,474
Epidermis	0.00005	18	21
Dermis	0.00009	32	38
Fat	0.00004	14	17
Muscle	0.00011	40	46
Water	0.00014	51	59
Oak (across grain)	0.00004	14	17
Dry air	0.000006	2.2	2.5
Glass fiber insulation	0.00001	3.6	4.2

*Through slabs of different materials 1 m^2 in area and 1 cm thick, with a difference in temperature of one Centigrade degree between the two faces of the slab

Adapted with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology*. Boston, Mass: Little, Brown; 1995: 590.

shell is relatively thin, and thus provides little insulation. Furthermore, a warm subject's skin blood flow is high, so that heat flow from the core to the skin is dominated by convection. In these circumstances the subcutaneous fat layer—which affects conduction but not convection—has little effect on heat flow from core to skin.

Core Temperature

Core temperature varies slightly from one site to another, depending on such local factors as metabolic rate and blood supply and the temperatures of neighboring tissues. However, the notion of a single uniform core temperature is a useful approximation because temperatures at different places in the core are all similar to the temperature of the central blood, and they tend to change together. Sites where core temperature is measured clinically include the mouth, the tympanic membrane, the rectum, and occasionally, the axilla. No site is ideal in every respect, and each has certain disadvantages and limitations (Exhibits 2-1 and 2-2).

The value of 98.6°F that is often given as the normal level of body temperature may suggest that body temperature is regulated so precisely that it is not allowed to deviate even a few tenths of a degree. In fact, 98.6°F is simply the Fahrenheit equivalent of 37°C; and, as Figure 2-1 indicates, body temperature does vary. The effects of heavy exercise and fever, for example, are quite familiar. In addition, variation among individuals and such factors[4] as time of day (Figure 2-3), phase of the menstrual cycle,[5,6] and acclimatization to heat can cause differences of up to about one Centigrade degree in core temperature in healthy subjects at rest. The thermoregulatory system receives information about the level of core temperature provided by temperature-sensitive neurons and nerve endings in the abdominal viscera, great veins, spinal cord, and especially the brain.[7,8] Later in the chapter we

EXHIBIT 2-1

MEASURING BODY CORE TEMPERATURE

Any measurement that is used as an index of core temperature should not be biased by environmental temperature. Because the tongue is richly supplied with blood, oral temperature under the tongue is usually similar to blood temperature and is 0.3°C to 0.4°C below rectal temperature[1]; but cooling of the face, neck, or mouth can make oral temperature misleadingly low.[2] Oral temperature should not be used to assess a patient with a suspected heat illness because such a patient may hyperventilate, thus cooling the mouth.

In 1959, Benzinger introduced tympanic temperature as an index of internal temperature for research in thermal physiology[3] and later also advocated its use as a clinical tool.[4] As Benzinger demonstrated, tympanic temperature responds more rapidly than rectal temperature to body cooling or heating[5]; and for this reason it has certain advantages over rectal temperature as a research tool. However, Benzinger did not merely say that tympanic temperature responds more rapidly than rectal temperature; he called it "cranial" temperature[5,6] and claimed that it represented hypothalamic temperature. He claimed further that the tympanum and hypothalamus share "a common blood supply … from the internal carotid artery,"[7(p139)] although, in fact, the blood supply of the tympanum is chiefly through branches of the external carotid artery. It would be easy to conclude that Benzinger believed tympanic temperature to be superior to core temperature measured anywhere outside the head (eg, in the esophagus or the heart or great vessels) as a representative of hypothalamic temperature. However, he evidently never claimed that tympanic temperature is superior in this regard to any temperature other than rectal temperature. Nevertheless, later authors[8] have concluded that tympanic temperature does indeed represent hypothalamic temperature better than other internal temperature measurements do—without, however, adducing any intracranial temperature measurements to support their conclusion. (Measurements in a surgical patient, in fact, showed that esophageal temperature followed changes in brain temperature more closely than did tympanic temperature.[9]) As a research tool in thermal physiology, tympanic temperature is now considerably less widely used than esophageal temperature because tympanic temperature is sensitive to skin temperature of the head and neck,[2] and thus may be biased substantially by ambient temperature. Benzinger himself recognized this problem and stressed that in environments cooler than 30°C, the ear should be insulated from the environment—preferably with the palm of the subject's hand.[5] However, his recommendation has frequently been ignored. Moreover, since most of the tympanum's blood supply comes through branches of the external carotid artery, thus following a somewhat superficial course, it is not clear how wide an area should be insulated, and there is no general agreement on this point.

Infrared sensing devices for measuring tympanic temperature, which eliminate the need for direct contact with the tympanum, have become available in recent years and have been marketed for clinical use. Tympanic temperature has come to enjoy a fair degree of popularity because these devices give a reading quickly and are easy to use. However, these devices are ordinarily used with no provision for insulating the ear from the ambient air, so tympanic temperature may be seriously biased by ambient temperature and is unsuitable for evaluating a patient suspected of having a heat illness.[10] (For a more extensive critique of tympanic temperature, see Brengelmann.[11])

The rectum is a few tenths of a Centigrade degree warmer than other core sites.[1] The rectum is well insulated from the environment, so rectal temperature is independent of environmental temperature and is the most reliable clinical index of body temperature.

If a patient holds his or her upper arm firmly against the chest so as to close the axilla, its temperature will gradually approach core temperature. Probably the chief advantage of measuring axillary temperature is that disinfecting the thermometer is less critical than when temperature is measured in the mouth or rectum. However, it may take 30 minutes or more for axillary temperature to come reasonably close to core temperature, so axillary temperature may be misleadingly low if insufficient time is allowed or if the patient does not keep his or her arm firmly against the chest. Axillary temperature has all but fallen into disuse.

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(1) Cranston WI, Gerbrandy J, Snell ES. Oral, rectal and oesophageal temperatures and some factors affecting them in man. *J Physiol (Lond)*. 1954;126:347–358. (2) McCaffrey TV, McCook RD, Wurster RD. Effect of head skin temperature on tympanic and oral temperature in man. *J Appl Physiol*. 1975;39:114–118. (3) Benzinger TH. On physical heat regulation and the sense of temperature in man. *Proc Natl Acad Sci U S A*. 1959;45:645–659. (4) Benzinger TH. Clinical temperature. New physiological basis. *JAMA*. 1969;209:1200–1206. (5) Benzinger TH, Taylor GW. Cranial measurements of internal temperature in man. In: Hardy JD, ed. *Temperature, Its Measurement and Control in Science and Industry. Vol 3, Part 3, Biology and Medicine*. New York, NY: Reinhold; 1963: 111–120. (6) Benzinger TH, Kitzinger C, Pratt AW. The human thermostat. In: Hardy JD, ed. Part 3. *Biology and Medicine.* In: Herzfeld CM, ed. *Temperature: Its Measurement and Control in Science and Industry.* Vol 3. New York, NY: Reinhold; 1963: 637–665. (7) Benzinger TH. The human thermostat. *Sci Am*. 1961;204:134–147. (8) Cabanac M, Caputa M. Open loop increase in trunk temperature produced by face cooling in working humans. *J Physiol (Lond)*. 1979;289:163–174. (9) Shiraki K, Sagawa S, Tajima F, Yokota A, Hashimoto M, Brengelmann GL. Independence of brain and tympanic temperatures in an unanesthetized human. *J Appl Physiol*. 1988;65:482–486. (10) Roberts WO. Assessing core temperature in collapsed athletes: What's the best method? *The Physician and Sportsmedicine*. 1994;22(8):49–55. (11) Brengelmann GL. Dilemma of body temperature measurement. In: Shiraki K, Yousef MK, eds. *Man in Stressful Environments: Thermal and Work Physiology*. Springfield, Ill: Charles C Thomas; 1987: 5–22.

EXHIBIT 2-2

BRAIN TEMPERATURE

A few investigators believe in the existence in humans of a physiological process called "selective brain cooling" that keeps the brain cooler than the trunk core during hyperthermia.[1,2] A similar process is known to occur in panting animals that possess carotid retes or other specialized vascular structures that provide for heat exchange between carotid arterial blood on its way to the brain, and cool venous blood returning from the respiratory passages, where evaporative cooling takes place. However, panting is not an important heat-loss mechanism in humans, and humans have no such specialized vascular structures for heat exchange. These investigators therefore propose that selective brain cooling in humans depends on venous blood that has been cooled by evaporation of sweat on the skin of the head, and then drains into the cranium[1–3] to exchange heat at several sites, particularly the cavernous sinus.[1,2] The evidence for selective brain cooling in humans is based largely on measurements of tympanic temperature, taken as representing brain temperature. In fact, because fanning to cool the face was found to lower tympanic temperature, fanning the face has been recommended as a way to protect the brains of patients with hyperthermia from thermal injury.[4] However, humans have no known heat-exchange mechanism that can cool the brain's blood supply more than a few hundredths of a Centigrade degree.[5] Interpretation of tympanic temperature as either core temperature or brain temperature is fraught with problems. Moreover, reports that the difference between esophageal and tympanic temperatures can be eliminated by suitable construction and placement of the tympanic temperature probe[6] imply that the notion of significant selective brain cooling in humans rests on a measurement artifact.

(1) Cabanac M. Keeping a cool head. *News Physiol Sci*. 1986;1:41–44. (2) Cabanac M, Caputa M. Natural selective cooling of the human brain: Evidence of its occurrence and magnitude. *J Physiol (Lond)*. 1979;286:255–264. (3) Cabanac M, Brinnel H. Blood flow in the emissary veins of the human head during hyperthermia. *Eur J Appl Physiol*. 1985;54:172–176. (4) Cabanac M. Face fanning: A possible way to prevent or cure brain hyperthermia. In: Khogali M, Hales JRS, eds. *Heat Stroke and Temperature Regulation*. Sydney, Australia: Academic Press; 1983: 213–221. (5) Wenger CB. More comments on "Keeping a cool head." *News Physiol Sci*. 1987;2:150. (6) Sato KT, Kane NL, Soos G, Gisolfi CV, Kondo N, Sato K. Reexamination of tympanic membrane temperature as a core temperature. *J Appl Physiol*. 1996;80:1233–1239.

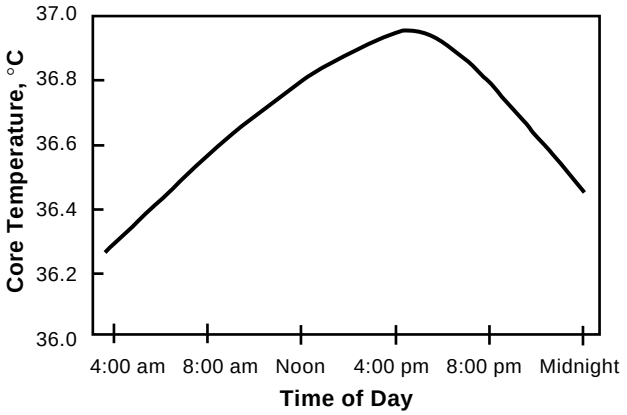

Fig. 2-3. Effect of time of day on internal body temperature of healthy resting subjects. Reprinted with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA. *Medical Physiology.* Boston, Mass: Little, Brown; 1995: 591. Original data sources: (1) Mackowiak PA, Wasserman SS, Levine MM. A critical appraisal of 98.6°F, the upper limit of normal body temperature, and other legacies of Carl Reinhold August Wunderlich. *JAMA*. 1992;268:1578–1580. (2) Stephenson LA, Wenger CB, O'Donovan BH, Nadel ER. Circadian rhythm in sweating and cutaneous blood flow. *Am J Physiol.* 1984;246:R321–R324.

discuss how the thermoregulatory system processes this information and uses it to maintain core temperature within a narrow range.

Skin Temperature

Skin temperature is important in heat exchange and thermoregulatory control. Most heat is exchanged between the body and the environment at the skin surface. Skin temperature is much more variable than core temperature and is affected by thermoregulatory responses such as skin blood flow and sweat secretion; by the temperatures of underlying tissues; and by environmental factors such as air temperature, air movement, and thermal radiation. Skin temperature, in turn, is one of the major factors determining heat exchange with the environment. For these reasons, skin temperature provides the thermoregulatory system with important information about the need to conserve or lose body heat. Many bare nerve endings just under the skin are very sensitive to temperature. Depending on the relation of discharge rate to temperature, these nerve endings are classified as either warm or cold receptors[7,9] (Figure 2-4). From the relative densities of cold- and warm-sensitive spots in human skin,[10] cold receptors appear to be roughly 10-fold as nu-

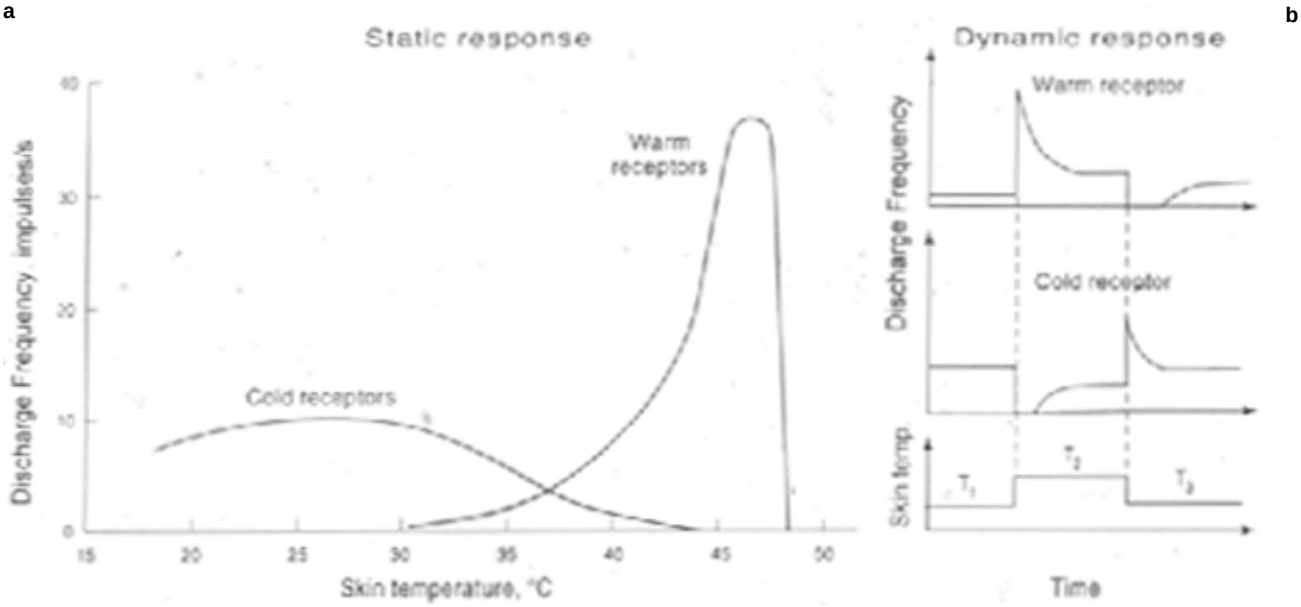

Fig. 2-4. Responses of cold- and warm-sensitive nerve fibers in the skin. Static response (**a**) is the discharge frequency when skin temperature is stable. Dynamic response (**b**) is the discharge frequency following a change in skin temperature. Adapted with permission from Hensel H, Kenshalo DR. Warm receptors in the nasal region of cats. *J Physiol (Lond).* 1969;204:109.

merous as warm receptors because, as a rule, a single cold or warm fiber innervates a single cold- or warm-sensitive spot.[11] With heating of the skin, warm receptors respond with a transient burst of activity, whereas cold receptors respond with a transient suppression; the reverse happens with cooling. These transient responses at the beginning of heating or cooling give the central integrator almost immediate information about changes in skin temperature, and may explain, for example, the intense, brief sensation of being chilled that occurs during a plunge into cold water.

Skin temperature usually is not uniform over the body surface, so a mean skin temperature ($\bar{T}_{sk}$) is frequently calculated from skin temperatures measured at several selected sites, usually weighting the temperature measured at each site according to the fraction of body surface area that it represents. It would be prohibitively invasive and difficult to measure shell temperature directly. Instead, therefore, skin temperature also is commonly used along with core temperature to calculate a mean body temperature and to estimate changes in the amount of heat stored in the body.

BALANCE BETWEEN HEAT PRODUCTION AND HEAT LOSS

All animals exchange energy with the environment. Some energy is exchanged as mechanical work, but most is exchanged as heat—by conduction, convection, and radiation; and as latent heat through evaporation or (rarely) condensation of water (Figure 2-5). If the sum of energy production and energy gain from the environment does not equal energy loss, the extra heat is "stored" in, or lost from, the body. This is summarized in Equation 1, the heat balance equation:

$$M = E + R + C + K + W + S \quad (1)$$

where M is metabolic rate; E is rate of heat loss by evaporation; R and C are rates of heat loss by radiation and convection, respectively; K is the rate of heat loss by conduction (only to solid objects in practice, as explained later); W is rate of energy loss as mechanical work; and S is rate of heat storage in the body, which takes the form of changes in tissue temperatures.[12,13]

The term M is always positive, but the other terms in Equation 1 may be either positive or negative. E, R, C, K, and W are positive if they represent energy losses from the body, and negative if they represent

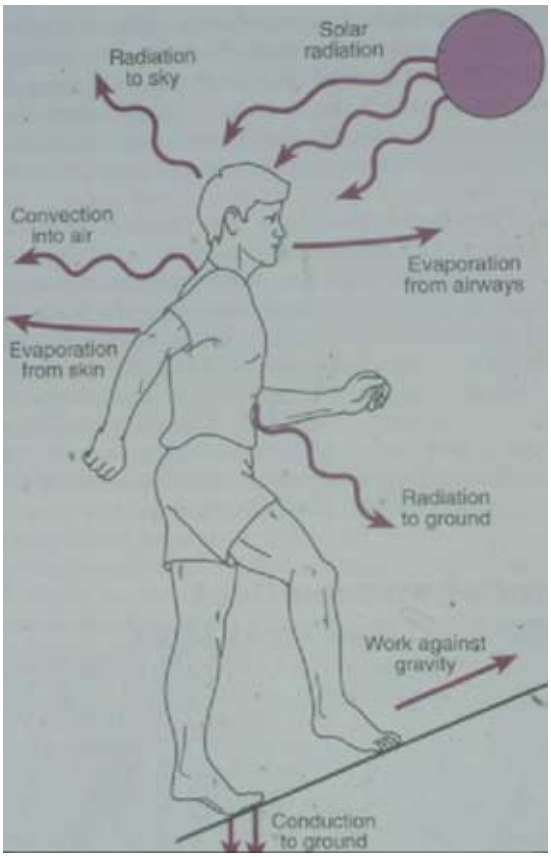

Fig. 2-5. Exchange of energy with the environment. This hiker gains heat from the sun by radiation, and loses heat by conduction to the ground through the soles of his feet, by convection into the air, by radiation to the ground and sky, and by evaporation of water from his skin and respiratory passages. In addition, some of the energy released by his metabolic processes is converted into mechanical work, rather than heat, since he is walking uphill. Reprinted with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology*. Boston, Mass: Little, Brown; 1995: 592.

energy gains. When S = 0, the body is in heat balance and body temperature neither rises nor falls. When the body is not in heat balance, its mean tissue temperature increases if S is positive, and decreases if S is negative. This commonly occurs on a short-term basis and lasts only until the body responds to changes in its temperature with thermoregulatory responses sufficient to restore balance; but if the thermal stress is too great for the thermoregulatory system to restore balance, the body will continue to gain or lose heat, until either the stress diminishes so that the thermoregulatory system can again restore the balance, or death occurs (Exhibit 2-3).

EXHIBIT 2-3

UNITS FOR MEASURING QUANTITY OF HEAT

The International Union of Physiological Sciences endorses the International System of Units (Système Internationale, SI) for expressing physiological quantities. In this system, quantity of heat is expressed in joules, the unit of work, and rate of heat production or heat flow is expressed in watts, the unit of power (1 W = 1 J/s). In traditional physiological usage, however, heat is expressed in kilocalories (kcal), which are still used widely enough that it is useful to be familiar with them. A kilocalorie (1 kcal = 4186 J) is the quantity of heat that will raise the temperature of 1 kg of pure water by one Centigrade degree, and is identical to the calorie (often spelled with a capital C) used to express the energy value of foods. The word "calorie," however, is a potential source of confusion because the same word was used in chemistry and physics to refer to a unit only 0.001 as large (sometimes called a small calorie), which is the quantity of heat that will raise the temperature of 1 g of pure water by one Centigrade degree.

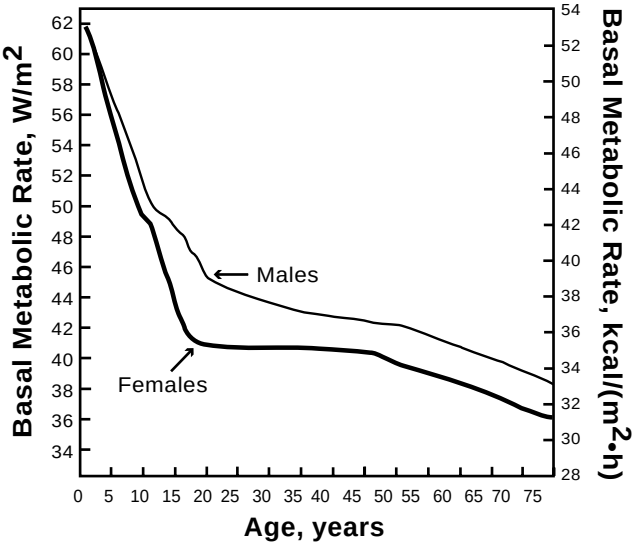

Fig. 2-6. Effects of age and gender on basal metabolic rate of normal subjects, expressed as the ratio of energy consumption to body surface area. Original data source: Fleish PA. La métabolisme basal standard et sa détermination au moyen du "metabocalculator." *Helv Med Acta.* 1951; 18:23–44.

TABLE 2-2

ILLUSTRATIVE VALUES FOR THERMAL PHYSIOLOGY

Measurement	SI* Units	Traditional Heat Units
Energy equivalent of oxygen for a mixed diet	20.2 kJ/L	4.83 kcal/L
Heat of evaporation of water	2.43 kJ/g	0.58 kcal/g
For a "Typical," Healthy, Lean, Young Man:		
Mass	70 kg	
Body surface area	1.8 m^2	
Mean specific heat of the body†	3.39 kJ/(kg • °C)	0.81 kcal/(kg • °C)
Volume specific heat of blood	3.85 kJ/(L • °C)	0.92 kcal/(L • °C)
Maximum rate of O_2 consumption	3.5 L/min	
Metabolic rate at rest‡	45 W/m^2	52.3 kcal/(m^2 • h)
Core-to-skin conductance with minimal skin blood flow‡	9 W/(m^2 • °C)	10.5 kcal/(m^2 • °C • h)

*Système Internationale (in which heat is expressed in units of work)
†Calculated for a body composition of 16% bone, 10% fat, and 74% lean soft tissue (ie, nonfatty tissue, neither bone nor tooth)
‡Per square meter of body surface area
Adapted with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology*. Boston, Mass: Little, Brown; 1995: 611.

Heat Production

Metabolic energy is required for active transport via membrane pumps, for muscular work, and for chemical reactions such as formation of glycogen from glucose and proteins from amino acids, whose products contain more energy than the materials that entered into the reaction. Most of the energy used in these processes is transformed into heat within the body. The transformation may be almost immediate, as with energy used in active transport or with heat produced as a by-product of muscular contraction. In other processes the conversion of energy to heat is delayed, as when the energy that was used to form glycogen or protein is released as heat when glycogen is converted back into glucose, or protein back into amino acids.

Metabolic Rate and Sites of Heat Production at Rest

Metabolic rate at rest varies with body size and is approximately proportional to body surface area. In a fasting young man it is about 45 W/m^2 (Figure 2-6) (81 W or 70 kcal/h for 1.8 m^2 body surface area [Table 2-2]), corresponding to an O_2 consumption of about 240 mL/min). At rest the trunk viscera and brain account for about 70% of energy production, even though they comprise only about 36% of body mass (Table 2-3). All the heat required to maintain heat balance at comfortable environmental temperatures is supplied as a by-product of metabolic processes that serve other functions, although in the cold, supplemental heat production may be elicited to maintain heat balance.

Factors other than body size that affect metabolism at rest include gender, age, hormones, and digestion. A nonpregnant woman's metabolic rate is 5% to 10% lower than that of a man of the same age and body surface area, probably because the female

TABLE 2-3

RELATIVE MASSES AND RATES OF METABOLIC HEAT PRODUCTION OF VARIOUS BODY COMPARTMENTS

	Body Mass (%)	Heat Production (%)		
		Rest	Severe*	Exercise
Brain	2	16		1
Trunk Viscera	34	56		8
Muscle and Skin	56	18		90
Other	8	10		1

*Intense or heavy
Adapted with permission from Wenger CB, Hardy JD. Temperature regulation and exposure to heat and cold. In: Lehmann JF, ed. *Therapeutic Heat and Cold*. Baltimore, Md: Williams & Wilkins; 1990: 156.

body includes a higher proportion of fat, a tissue with a low metabolic rate. (However, the growing fetus's energy requirements increase a pregnant woman's measured metabolic rate.)

Catecholamines and thyroxine are the hormones with the largest effect on metabolic rate. Catecholamines stimulate many enzyme systems, thus increasing cellular metabolism; and hypermetabolism occurs in some cases of pheochromocytoma, a secreting tumor of the adrenal medulla. Thyroxine magnifies the metabolic response to catecholamines and stimulates oxidation in the mitochondria. Hyperthyroidism may double the metabolic rate in severe cases, although an increase to 45% above normal is more typical; and metabolic rate is typically 25% below normal in hypothyroidism but may be 45% below normal with total lack of thyroxine.

Metabolic rate at rest increases after a meal as a result of the *thermic effect of food* (or "specific dynamic action," the older term). The increase varies according to the composition of the meal and the physiological state, including the level of nutrition, of the subject.[14] In a well-nourished subject the increase is typically 10% to 20%. The effect lasts several hours and appears to be associated with processing the products of digestion by the liver.

Measurement of Metabolic Rate

Heat exchange with the environment can be measured directly with a human calorimeter,[15] a specially constructed insulated chamber that allows heat to leave only in the air ventilating the chamber or, often, in water flowing through a heat exchanger in the chamber. From accurate measurements of the flow of air and water, and their temperatures as they enter and leave the chamber, we can compute the subject's heat loss by conduction, convection, and radiation; and from measurements of the moisture content of air entering and leaving the chamber, we can also determine heat loss by evaporation. *Direct calorimetry*, as this technique is called, is simple in concept but difficult and costly in practice. Therefore metabolic rate is often estimated by *indirect calorimetry*[16] based on measurements of O_2 consumption, because virtually all energy available to the body depends ultimately on reactions that consume O_2.

Consumption of 1 liter of O_2 is associated with release of 21.1 kJ (5.05 kcal) if the fuel is carbohydrate, 19.8 kJ (4.74 kcal) if the fuel is fat, and 18.6 kJ (4.46 kcal) if the fuel is protein. For metabolism of a mixed diet, an average value of 20.2 kJ (4.83 kcal) per liter of O_2 is often used (see Table 2-2). The ratio of CO_2 produced to O_2 consumed in the tissues, called the r*espiratory quotient* (RQ), is 1.0 for oxidation of carbohydrate, 0.71 for oxidation of fat, and 0.80 for oxidation of protein. In a steady state in which CO_2 is exhaled at the same rate that it is produced in the tissues, RQ is equal to the respiratory exchange ratio, R; and the accuracy of indirect calorimetry can be improved by also determining R, and either estimating the amount of protein oxidized—usually small compared with fat and carbohydrate—or calculating it from urinary nitrogen excretion.

Skeletal Muscle Metabolism and Muscular Work

Even during very mild exercise the muscles are the chief source of metabolic heat, and during heavy exercise they (together with the skin) may account for up to 90% of the heat production (see Table 2-3). A healthy but sedentary young man performing moderately intense exercise may increase his metabolic rate to 600 W (in contrast to about 80 W at rest); and a trained athlete performing intense exercise, to 1400 W or more. Exercising muscles may be nearly one Centigrade degree warmer than the core because of their high metabolic rate. Blood is warmed as it perfuses these muscles, and the blood, in turn, warms the rest of the body and raises core temperature. Like engines that burn fossil fuels, muscles convert most of the energy in the fuels that they consume into heat rather than mechanical work.

When adenosine 5'-diphosphate (ADP) is phosphorylated to form adenosine 5'-triphosphate (ATP), 58% of the energy released from the fuel is converted into heat, and only about 42% is captured in the ATP that is formed. Then when ATP is hydrolyzed during a muscle contraction, some of the energy in the ATP is converted into heat rather than into mechanical work. The efficiency of this process varies enormously, and is zero in isometric contraction, in which a muscle's length does not change while it develops tension, so that the muscle does no work even though it consumes metabolic energy. Finally, some mechanical work is converted by friction into heat within the body—as, for example, happens to the mechanical work done by the heart in pumping blood. At best, no more than one quarter of the metabolic energy released during exercise is converted into mechanical work outside the body, and the remaining three quarters or more is converted into heat within the body[17] (Exhibit 2-4).

EXHIBIT 2-4

ENERGY CONSUMPTION AND HEAT PRODUCTION DURING PERFORMANCE OF MILITARY TASKS

Many military tasks require high levels of power output, and are associated with correspondingly high rates of metabolic heat production. Table 3-2 in Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues lists metabolic rates required by men wearing the battle dress uniform (BDU) to perform 28 military occupational tasks. The added weight and stiffness of special protective clothing increase the energy cost of performing a task, and wearing the full ensemble of nuclear biological chemical protective clothing (including overgarment, boot, gloves, gas mask, and hood) over BDUs increases the rate of oxygen consumption by an average of about 10%.[1]

Of the military tasks with a high energy demand, walking and running—with or without an external load—are probably among those that are most suitable for prediction of energy requirement. For walking speeds of 2.5 km/h or greater, and light-to-moderate loads that are distributed so that their center of gravity is near the body's center of gravity, the following equation[2] predicts the metabolic power requirements for walking as a function of body weight, speed, grade, carried load, and surface:

$$M = \eta\,(W + L)\,\{2.3 + 0.32\,(V - 2.5\ \text{km/h})^{1.65} + G\,[0.2 + 0.7\,(V - 2.5\ \text{km/h})]\}$$

where *M* represents metabolic rate, kcal/h; η represents the terrain factor, defined as 1 for treadmill walking; *W* represents body weight in kilograms; *L* represents external load in kilograms; *V* represents walking speed in kilometers per hour; and *G* represents % grade.

Some values of the terrain factor, η, are 1.0 for blacktop surface, 1.1 for dirt road, 1.2 for light brush, 1.5 for heavy brush, 1.8 for swampy bog, and 2.1 for loose sand.[3]

Exhibit Table 1 contains some illustrative predictions for metabolic rates of a 70-kg subject walking at several speeds and grades on blacktop with no external load:

EXHIBIT TABLE 1
PREDICTED METABOLIC RATES OF A 70-KG SOLDIER WALKING AT SELECTED COMBINATIONS OF SPEED AND GRADE

	Speed			
Grade	**4 km/h (2.5 mph)**	**5 km/h (3.1 mph)**	**6 km/h (3.7 mph)**	**7 km/h (4.4 mph)**
0%	204 kcal/h	263 kcal/h	338 kcal/h	429 kcal/h
2%	379 kcal/h	536 kcal/h	709 kcal/h	898 kcal/h

Adding an external load, or substituting a less advantageous surface for blacktop, will increase the energy requirements proportionately. The cumulative effect of seemingly small changes in speed, grade, load, and terrain can impose a huge physiological burden on the body's capacity to support physical exercise and dissipate heat.

(1) Patton JF, Murphy M, Bidwell T, Mello R, Harp M. *Metabolic Cost of Military Physical Tasks in MOPP 0 and MOPP 4.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1995. USARIEM Technical Report T95-9. (2) Givoni B, Goldman RF. Predicting metabolic energy cost. *J Appl Physiol*. 1971;30:429–433. (3) Soule RG, Goldman RF. Terrain coefficients for energy cost prediction. *J Appl Physiol*. 1972;32:706–708.

Heat Exchange With the Environment

Convection, radiation, and evaporation are the dominant means of heat exchange with the environment. Both the skin and the respiratory passages exchange heat with the environment by convection and evaporation, but only the skin exchanges heat by radiation. In some animal species, panting is an

important thermoregulatory response, which can produce high rates of heat loss. In humans, however, respiration usually accounts for only a minor fraction of total heat exchange and is not predominantly under thermoregulatory control, although hyperthermic subjects may hyperventilate.

Convection is transfer of heat due to movement of a fluid, either liquid or gas. In thermal physiology the fluid is usually air or water in the environment, or blood inside the body, as discussed earlier. Fluids conduct heat in the same way as solids do, and a perfectly still fluid transfers heat only by conduction. Because air and water are not good conductors of heat, perfectly still air or water are not very effective in heat transfer. Fluids, however, are rarely perfectly still, and even nearly imperceptible movement produces enough convection to cause a large increase in the rate of heat transfer. Thus, although conduction plays a role in heat transfer by a fluid, convection so dominates the overall heat transfer that we refer to the entire process as convection. The conduction term, K, in Equation 1 is therefore restricted to heat flow between the body and other solid objects, and usually represents only a small part of the total heat exchange with the environment.

Convective heat exchange between the skin and the environment is proportional to the difference between skin and ambient air temperatures, as expressed by Equation 2:

$$C = h_c \bullet A \bullet (\overline{T}_{sk} - T_a) \qquad (2)$$

where A is the body surface area, $\overline{T}_{sk}$ and T_a are mean skin and ambient temperatures, respectively, and h_c is the convective heat transfer coefficient.

The term h_c includes the effects of all the factors besides temperature and surface area that affect convective heat exchange. For the whole body, the most important of these factors is air movement, and convective heat exchange (and thus h_c) varies approximately as the square root of the air speed (Figure 2-7) unless air movement is very slight.

Every surface emits energy as electromagnetic radiation with a power output that depends on its area, its temperature, and its emissivity (e), a number between 0 and 1 that depends on the nature of the surface and the wavelength of the radiation. (For purposes of this discussion the term "surface" has a broader meaning than usual, so that, for example, a flame and the sky are both surfaces.) The emissivity of any surface is identical to its absorptivity (ie, the fraction of incoming radiant energy that the surface absorbs rather than reflects). Such radiation,

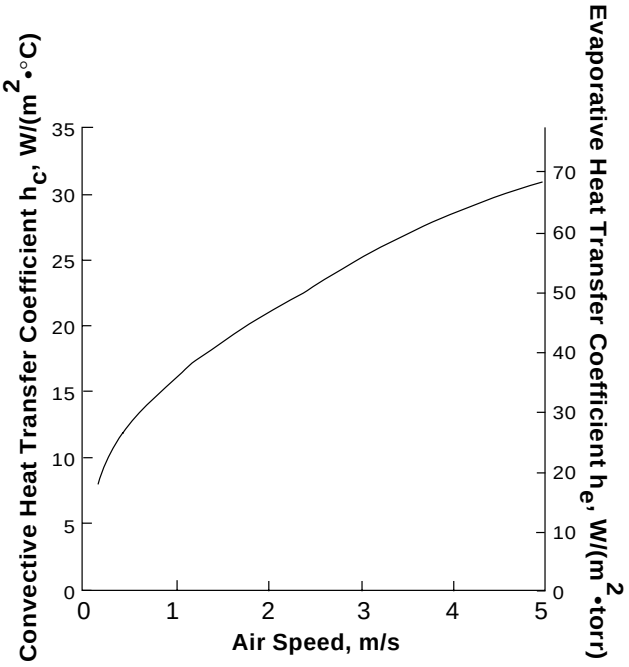

Fig. 2-7. The convective ($h_{c)}$ and evaporative (h_e) heat transfer coefficients for a standing human as a function of air speed. The coefficients h_c and h_e increase with air speed in the same way, and $h_e = h_c \bullet 2.2°C/mm\ Hg$. Thus with suitable scaling of the vertical axes, as in this figure, the curves for h_c and h_e overlie each other. The horizontal axis can be converted into English units by using the relation 5 m/s = 16.4 ft/s = 11.2 mph.

called thermal radiation, has a characteristic distribution of energy as a function of wavelength, which depends on the temperature of the surface. For a surface that is not hot enough to glow this radiation is in the infrared part of the spectrum, and at ordinary tissue and environmental temperatures virtually all of the emitted energy is at wavelengths longer than 3 microns. Most surfaces except polished metals have emissivities near 1 in this range, and thus both emit and absorb radiation at nearly the theoretical maximum efficiency. As a surface's temperature increases, however, the average wavelength of its thermal radiation decreases, and most of the energy in solar radiation is in the near infrared and visible range, for which light surfaces have lower absorptivities than dark ones.

If two surfaces exchange heat by thermal radiation, radiation travels in both directions; but because each surface emits radiation with an intensity that depends on its temperature, the net heat flow is from the warmer to the cooler body. Radiative heat exchange between two surfaces is, strictly, proportional to the difference between the fourth

powers of the surfaces' absolute temperatures. However, if the difference between $\overline{T}_{sk}$ and the temperature of the radiant environment (T_r) is much smaller than the absolute temperature of the skin, R is nearly proportional to ($\overline{T}_{sk} - T_r$). Some parts of the body surface (eg, inner surfaces of the thighs and arms) exchange heat by radiation with other parts of the body surface, so that the body exchanges heat with the environment as if it had an area smaller than its actual surface area. This smaller area is called the *effective radiating surface area* (A_r), and depends on the posture, being greatest, or closest to the actual surface area, in a "spread eagle" posture, and least in someone who is curled up. Radiative heat exchange can be represented by Equation 3:

$$(3) \qquad R = h_r \bullet e_{sk} \bullet A_r \bullet (\overline{T}_{sk} - T_r)$$

where h_r is the radiant heat transfer coefficient, 6.43 W/(m^2 • °C) at 28°C; and e_{sk} is the emissivity of the skin.

When a gram of water is converted into vapor at 30°C, it absorbs 2,425 J (0.58 kcal; see Table 2-2), the *latent heat of evaporation*, in the process. When the environment is hotter than the skin—as it usually is when the environment is warmer than 36°C—evaporation is the body's only way to lose heat, and must dissipate not only the heat produced by the body's metabolism, but also any heat gained from the environment by R and C (from Equation 1). Most water evaporated in the heat comes from sweat; but even in the cold, water diffuses through the skin and evaporates. Evaporation of this water is called *insensible perspiration*,[9,18] and occurs independently of the sweat glands. E is nearly always positive (representing loss of heat from the body); but it is negative in unusual circumstances, such as in a steam room, where water vapor condensing on the skin gives up heat to the body.

Evaporative heat loss from the skin is proportional to the difference between the water vapor pressure at the skin surface and the water vapor pressure in the ambient air. These relations are summarized in Equation 4:

$$(4) \qquad E = h_e \bullet A \bullet (P_{sk} - P_a)$$

where P_{sk} is the water vapor pressure at the skin surface, P_a is the ambient water vapor pressure, and h_e is the evaporative heat transfer coefficient.

Because water vapor, like heat, is carried away by moving air, air movement and other factors affect E and h_e in just the same way that they affect C and h_c. If the skin surface is completely wet, the water vapor pressure at the skin surface is the saturation water vapor pressure (Figure 2-8) at skin temperature, and evaporative heat loss is E_{max}, the maximum possible for the prevailing skin temperature and environmental conditions. This situation is described in Equation 5:

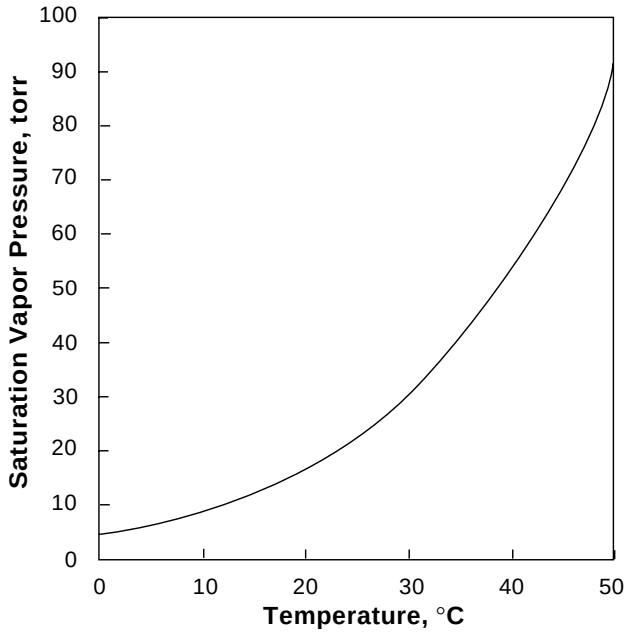

Fig. 2-8. The saturation vapor pressure of water as a function of temperature. For any given temperature, the water vapor pressure is at its saturation value when the air is "saturated" with water vapor (ie, the air holds the maximum amount possible at that temperature, or the relative humidity is 100%).

$$(5) \qquad E_{max} = h_e \bullet A \bullet (P_{sk,sat} - P_a)$$

where $P_{sk,sat}$ is the saturation water vapor pressure at skin temperature, and h_e is the evaporative heat transfer coefficient.

When the skin is not completely wet, it is impractical to measure the actual average water vapor pressure at the skin surface. Therefore a coefficient called skin *wettedness* (w)[19] is defined as the ratio E/E_{max}, with $0 \le w \le 1$. Skin wettedness depends on the hydration of the epidermis and the fraction of the skin surface that is wet. We can now rewrite Equation 4 as Equation 6:

$$(6) \qquad E = h_e \bullet A \bullet w \bullet (P_{sk,sat} - P_a)$$

Wettedness depends on the balance between secretion and evaporation of sweat. If secretion ex-

ceeds evaporation, sweat accumulates on the skin and spreads out to wet more of the space between neighboring sweat glands, thus increasing wettedness and E; and if evaporation exceeds secretion, the reverse occurs. If sweat rate exceeds E_{max}, then once wettedness becomes 1, the excess sweat drips from the body because it cannot evaporate.

Note that P_a, on which evaporation from the skin directly depends, is proportional to the actual moisture content in the air. By contrast, the more familiar quantity, relative humidity (rh), is the ratio between the actual moisture content in the air and the maximum moisture content that is possible at the temperature of the air. It is important to recognize that rh is only indirectly related to evaporation from the skin. For example, in a cold environment, P_a will be low enough that sweat can easily evaporate from the skin even if rh = 100%.

Clothing reduces heat exchange between the body and its environment through several mechanisms. By impeding air movement, clothing reduces h_c and h_e at the skin, thereby reducing heat exchange by convection and evaporation. In addition, clothing resists conduction of heat, and is at least a partial barrier to radiative heat exchange and passage of water vapor. For all of these reasons, clothing creates a microenvironment that is closer to skin temperature than is the environment outside the clothing. Furthermore, since the body is a source of water vapor, the air inside the clothing is more humid than outside. The conditions inside this microenvironment—air temperature, water vapor pressure, and temperature of the inner surface of the clothing—are what determine heat gain or heat loss by unexposed skin. These conditions in turn are determined by the conditions outside the clothing, the properties of the clothing, and the rate at which the body releases heat and moisture into this microenvironment. Therefore, the level of physical activity determines both (*a*) the appropriate level of clothing for the environmental conditions and (*b*) the degree of heat *strain* (ie, physiological change produced by a disturbance) that results from wearing clothing that is too warm for the conditions, as protective clothing often is.

Although clothing reduces heat exchange between covered skin and the environment, it has little effect on heat exchange of exposed skin. Therefore—especially when the clothing is heavy and most of the skin is covered—exposed skin may account for a fraction of the body's heat loss that far exceeds the exposed fraction of the body's surface. Thus in the cold, the head may account for half of the heat loss from the body[20]; and in someone exercising while wearing nuclear, biological, and chemical (NBC) protective clothing without gas mask and hood, donning the mask and hood while continuing to exercise may lead to a dramatic increase in heat strain.[21]

Heat Storage

Heat storage is a change in the body's heat content. The rate of heat storage is the difference between heat production/gain and heat loss (see Equation 1), and can be determined from simultaneous measurements of metabolism by indirect calorimetry and heat gain or loss by direct calorimetry. Because heat storage in the tissues changes their temperature, the amount of heat stored is the product of body mass, the body's mean specific heat, and a suitable mean body temperature (T_b). The body's mean specific heat depends on its composition, especially the proportion of fat, and is about 3.39 kJ/(kg • °C) [0.81 kcal/(kg • °C)] (see Table 2-2) for a typical body composition of 16% bone, 10% fat, and 74% lean soft tissue (ie, tissue that is neither bone nor tooth, and is not fatty). Empirical relations of T_b to core temperature (T_c) and $\overline{T}_{sk}$, determined in calorimetric studies, depend on ambient temperature, with T_b varying from $0.67 \bullet T_c + 0.33 \bullet \overline{T}_{sk}$ in the cold to $0.9 \bullet T_c + 0.1 \bullet \overline{T}_{sk}$ in the heat.[19] The shift from cold to heat in the relative weighting of T_c and $\overline{T}_{sk}$ reflects the accompanying change in the thickness of the shell (see Figure 2-2).

HEAT DISSIPATION

Figure 2-9 shows rectal and mean skin temperatures, heat losses, and calculated shell conductances for nude resting men and women at the end of 2-hour exposures in a calorimeter to ambient temperatures from 23°C to 36°C. Shell conductance represents the sum of heat transfer by two parallel modes (ie, conduction through the tissues of the shell, and convection by the blood); it is calculated by dividing heat loss through the skin (HF_{sk})—(ie, total heat loss less heat loss through the respiratory tract)—by the difference between core and mean skin temperatures, as shown in Equation 7:

$$C = HF_{sk} / (T_c - \overline{T}_{sk}) \quad (7)$$

where C is shell conductance, and T_c and $\overline{T}_{sk}$ are core

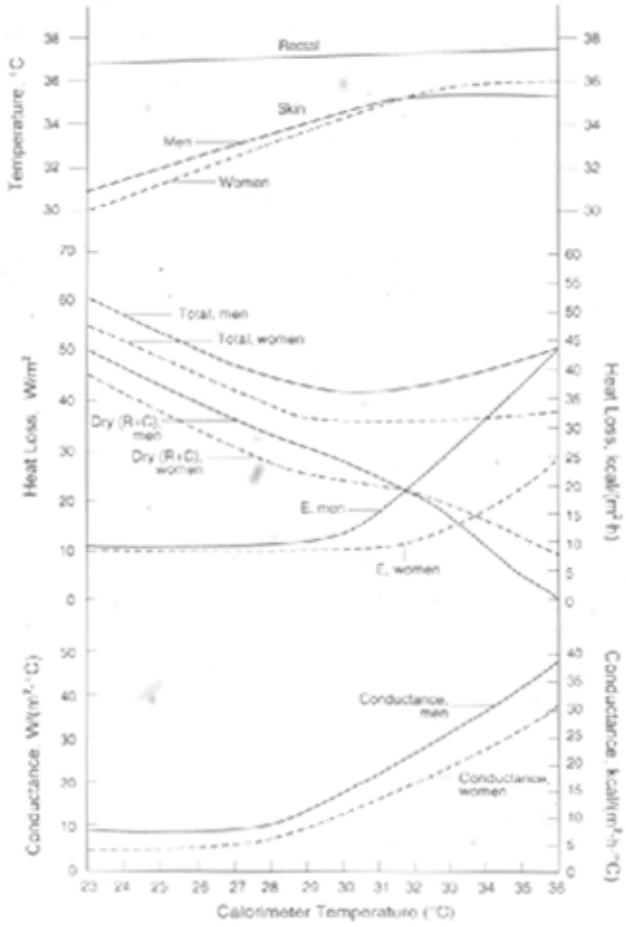

Fig. 2-9. Average values of rectal and mean skin temperatures, heat loss, and core-to-skin thermal conductance for nude resting men and women near steady state after 2 hours at different environmental temperatures in a calorimeter. (All energy-exchange quantities in this figure have been divided by body surface area, to remove the effect of individual body size.) Total heat loss is the sum of dry heat loss (by radiation [R] and convection [C]) and evaporative heat loss (E). Dry heat loss is proportional to the difference between skin temperature and calorimeter temperature, and it decreases with increasing calorimeter temperature. Adapted (data correction) with permission from Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology*. Boston, Mass: Little, Brown; 1995: 596. Data source: Hardy JD, DuBois EF. Differences between men and women in their response to heat and cold. *Proc Natl Acad Sci U S A.* 1940;26:389–398.

and mean skin temperatures, respectively.

At ambient temperatures below 28°C, these subjects' conductance is minimal because their skin blood flow is quite low. Because the minimum attainable level of conductance depends chiefly on the subcutaneous fat layer, the women's thicker layer allows them to attain a lower conductance than men. At about 28°C, conductance begins to increase, and above 30°C, conductance continues to increase and sweating begins. For these nude subjects, the range 28°C to 30°C is the zone of *thermoneutrality;* that is, the range of comfortable environmental temperatures in which thermal balance is maintained without either shivering or sweating.[12] In this zone, heat loss is matched to heat production by controlling conductance, and thus $\overline{T}_{sk}$, R, and C.

Evaporation

As we saw in Figure 2-9, evaporative heat loss is nearly independent of ambient temperature below 30°C, and is 9 to 10 W/m^2. This corresponds to evaporation of about 13 to 15 $g/(m^2 \bullet h)$, of which about half is lost through breathing and half as insensible perspiration. This heat loss is not under thermoregulatory control. To achieve heat balance at higher ambient temperatures, the subjects in Figure 2-9 depend more and more on evaporation of sweat, which in humans can dissipate large amounts of heat.

There are two histological types of sweat glands, *eccrine* and *apocrine*. In humans, apocrine glands are found mostly in the axilla, inguinal region, perianal skin, and mammary areolae, and less consistently on other parts of the trunk and the face.[22] Eccrine sweat is essentially a dilute electrolyte solution, but apocrine sweat also contains fatty material. Eccrine sweat glands are widely distributed and are the more important type in human thermoregulation, and functionally active eccrine glands number about 2 to 3 million.[23] They are controlled through postganglionic sympathetic nerves, which release acetylcholine[23] rather than norepinephrine. A healthy man unacclimatized to heat can secrete up to 1.5 liters of sweat per hour. Although the number of functional sweat glands is fixed before the age of 3 years,[23] the secretory capacity of individual glands can change, especially with endurance exercise training and heat acclimatization; and a man who is well acclimatized to heat can secrete more than 2.5 L/h.[24,25] Such rates cannot be maintained, however, and the maximum daily sweat output is probably about 15 L.[26]

Sodium concentration of eccrine sweat ranges from less than 5 to 60 mEq/L[27] (vs 135–145 mEq/L in plasma); but even at 60 mEq/L, sweat is one of the most dilute body fluids. To produce sweat that is hypotonic to plasma, the glands reabsorb sodium from the sweat duct by active transport. As sweat

rate increases, the rate at which the glands reabsorb sodium increases more slowly, so that sodium concentration in the sweat increases.

Skin Circulation and Dry (Convective and Radiative) Heat Exchange

Heat produced within the body must be delivered to the skin surface to be eliminated. When skin blood flow is minimal, core-to-skin thermal conductance (ie, the conductance of the shell) is typically 5 to 9 W per Centigrade degree per square meter of body surface (see Figure 2-9). A lean resting subject with a surface area of 1.8 m^2, minimal whole-body conductance of 16 W/°C [ie, 8.9 W/(°C • m^2) x (1.8 m^2)] and a metabolic heat production of 80 W, requires a temperature difference between core and skin of five Centigrade degrees (ie, 80 W ÷ 16 W/°C) to allow the heat produced inside the body to be conducted to the surface. In a cool environment, T_{sk} may easily be low enough for this to occur. However, in an ambient temperature of 33°C, $\overline{T}_{sk}$ is typically about 35°C; and without an increase in conductance, core temperature would need to rise to 40°C—a high although not yet dangerous level—for the heat to be conducted to the skin. But if the rate of heat production were increased to 480 W by moderate exercise, the temperature difference between core and skin would have to rise to 30°C—and core temperature to well beyond lethal levels—to allow all the heat produced to be conducted to the skin. In such circumstances a large increase in conductance is needed for the body to reestablish thermal balance and continue to regulate its temperature; and this is accomplished by increasing skin blood flow.

Role of Skin Blood Flow in Heat Transfer

If we assume that blood on its way to the skin remains at core temperature until it reaches the skin, comes to skin temperature as it passes through the skin, and then stays at skin temperature until it returns to the core, we can compute the rate of heat flow (HF_b) due to convection by the blood as seen in Equation 8:

$$(8) \quad HF_b = SkBF \bullet (T_c - T_{sk}) \bullet 3.85\ kJ/(L \bullet °C)$$

where *SkBF*, the rate of skin blood flow, is expressed in L/s rather than the more usual L/min, to simplify computing HF in W (ie, J/s); and *3.85 kJ/(L • °C) [0.92 kcal/(L • °C)]* = volume specific heat of blood[28] (see Table 2-2).

Conductance due to convection by the blood (C_b) is calculated as seen in Equation 9:

$$(9) \quad C_b = HF_b/(T_c - T_{sk}) = SkBF \bullet 3.85\ kJ/(L \bullet °C)$$

Of course, heat continues to flow by conduction through the tissues of the shell, so that total conductance is the sum of conductance due to convection by the blood plus that due to conduction through the tissues; and total heat flow is given by Equation 10:

$$(10) \quad HF = (C_b + C_0) \bullet (T_c - T_{sk})$$

in which C_0 is thermal conductance of the tissues when skin blood flow is minimal, and thus is due predominantly to conduction through the tissues.

The assumptions on which Equation 8 depend represent the conditions for maximum efficiency of heat transfer by the blood, and are somewhat artificial. In practice, blood also exchanges heat with the tissues through which it passes going to and from the skin. Heat is exchanged with these other tissues most easily when skin blood flow is low, and in such cases heat flow to the skin may be much less than that predicted by Equation 8. However, Equation 8 is a reasonable approximation in a warm subject with moderate-to-high skin blood flow. It is not possible to measure whole-body skin blood flow directly, but it is estimated to reach nearly 8 L/min during maximal cutaneous vasodilation.[29,30] Maximal cutaneous vasodilation does not occur during heavy exercise,[31] but skin blood flow still may reach several liters per minute during heavy exercise in the heat.[29] If SkBF = 1.89 L/min (0.0315 L/s), then, according to Equation 9, skin blood flow contributes about 121 W/°C to the conductance of the shell. If conduction through the tissues contributes 16 W/°C, total shell conductance is 137 W/°C; and if T_c = 38.5°C and T_{sk} = 35°C, then this will produce a core-to-skin heat transfer of 480 W, the heat production in our earlier example of moderate exercise. Thus even a moderate rate of skin blood flow can have a dramatic effect on heat transfer.

In a person who is not sweating, raising skin blood flow brings skin temperature nearer to blood temperature, and lowering skin blood flow brings skin temperature nearer to ambient temperature. In these conditions the body controls dry (convective and radiative) heat loss by varying skin blood flow and thus skin temperature. Once sweating begins, skin blood flow continues to increase as the person becomes warmer, but now the tendency of an increase in skin blood flow to warm the skin is approxi-

mately balanced by the tendency of an increase in sweating to cool the skin. Therefore, after sweating has begun, further increases in skin blood flow usually cause little change in skin temperature or dry heat exchange, and serve primarily to deliver to the skin the heat that is being removed by evaporation of sweat. Skin blood flow and sweating thus work in tandem to dissipate heat under such conditions.

Sympathetic Control of Skin Circulation

Blood flow in human skin is under dual vasomotor control.[8,30,32] In most of the skin the vasodilation that occurs during heat exposure depends on sympathetic nervous signals that cause the blood vessels to dilate, and this vasodilation can be prevented or reversed by regional nerve block.[33] Because it depends on the action of nervous signals, such vasodilation is sometimes referred to as active vasodilation. Active vasodilation occurs in almost all the skin except in the so-called acral regions—hands, feet, lips, ears, and nose.[34] In the skin areas where active vasodilation occurs, vasoconstrictor activity is minimal at thermoneutral temperatures; and as the body is warmed, active vasodilation does not begin until close to the onset of sweating.[30,35] Thus skin blood flow in these areas is not much affected by small temperature changes within the thermoneutral range.[34] The neurotransmitter or other vasoactive substance responsible for active vasodilation in human skin has not been identified.[36] However, because sweating and vasodilation operate in tandem in the heat, some investigators[30,37] have proposed that the mechanism for active vasodilation is somehow linked to the action of sweat glands.

Reflex vasoconstriction, which occurs in response to cold and also as part of certain nonthermal reflexes such as baroreflexes, is mediated primarily through adrenergic sympathetic fibers, which are distributed widely over most of the skin.[36] Reducing the flow of impulses in these nerve fibers allows the blood vessels to dilate. In the acral regions[30,36] and in the superficial veins,[30] vasoconstrictor fibers are the predominant vasomotor innervation, and the vasodilation that occurs during heat exposure is largely a result of the withdrawal of vasoconstrictor activity.[34] Blood flow in these skin regions is sensitive to small temperature changes even in the thermoneutral range, and may be responsible for "fine tuning" heat loss to maintain heat balance in this range.

THERMOREGULATORY CONTROL

In control theory, the words *regulation* and *regulate* have meanings distinct from those of *control*. A control system acts to minimize changes in the *regulated* variable (eg, core temperature) that are produced by disturbances from outside the system (eg, exercise or changes in the environment) by making changes in certain other variables (eg, sweating rate, skin blood flow, metabolic rate, and thermoregulatory behavior), which are called *controlled* variables. Human beings have two distinct subsystems to regulate body temperature: behavioral thermoregulation and physiological thermoregulation. Physiological thermoregulation is capable of fairly precise adjustments of heat balance but is effective only within a relatively narrow range of environmental temperatures. On the other hand, behavioral thermoregulation, through the use of shelter and space heating and clothing, enables humans to live in the most extreme climates on earth, but it does not provide fine control of body heat balance.

Behavioral Thermoregulation

Behavioral thermoregulation is governed by thermal sensation and comfort. Sensory information about body temperatures is an essential part of both behavioral and physiological thermoregulation. The distinguishing feature of behavioral thermoregulation is the direction of conscious effort to reduce discomfort. Warmth and cold on the skin are felt as either comfortable or uncomfortable, depending on whether they decrease or increase the physiological strain.[38] Thus a shower temperature that feels pleasant after strenuous exercise may be uncomfortably cold on a chilly morning. Because of the relation between discomfort and physiological strain, behavioral thermoregulation, by reducing discomfort, also acts to minimize the physiological burden imposed by a stressful thermal environment. For this reason the zone of thermoneutrality is characterized by thermal comfort as well as by the absence of shivering and sweating.

The processing of thermal information in behavioral thermoregulation is not as well understood as it is in physiological thermoregulation. However, perceptions of thermal sensation and comfort respond much more quickly than either core temperature or physiological thermoregulatory responses to changes in environmental temperature,[39,40] and thus appear to anticipate changes in the body's ther-

mal state. Such an anticipatory feature presumably reduces the need for frequent small behavioral adjustments.

Physiological Thermoregulation

Physiological thermoregulation operates through graded control of heat-production and heat-loss responses. Familiar nonliving control systems, such as most refrigerators and heating and air-conditioning systems, operate at only two levels because they act by turning a device on or off. In contrast, most physiological control systems produce a response that is graded according to the disturbance in the regulated variable. In many physiological systems, changes in the controlled variables are proportional to displacements of the regulated variable from some threshold value, and such control systems are called *proportional control* systems.

The control of heat-dissipating responses is an example of a proportional control system.[9] Figure 2-10 shows how reflex control of sweating and skin blood flow depends on body core and skin temperatures. Each response has a core temperature threshold, a temperature at which the response starts to increase; and these thresholds depend on mean skin temperature. Thus at any given skin temperature, the change in each response is proportional to the change in core temperature; and increasing the skin temperature lowers the threshold level of core temperature and increases the response at any given core temperature. In humans, a change of one Centigrade degree in core temperature elicits about nine times as great a thermoregulatory response as a change in mean skin temperature of one Centigrade degree.[8] (Besides its effect on the reflex signals, skin temperature has a local effect that modifies the blood vessel and sweat gland responses, as discussed later.)

Integration of Thermal Information

The central nervous system integrates thermal information from core and skin. Receptors in the body core and the skin transmit information about their temperatures through afferent nerves to the brainstem, and especially the hypothalamus, where much of the integration of temperature information occurs.[41] The sensitivity of the thermoregulatory responses to core temperature allows the thermoregulatory system to adjust heat production and heat loss to resist disturbances in core temperature. Their sensitivity to mean skin temperature allows the system to respond appropriately to mild heat or cold exposure with little change in body core temperature, so that environmentally induced changes in body heat content occur al-

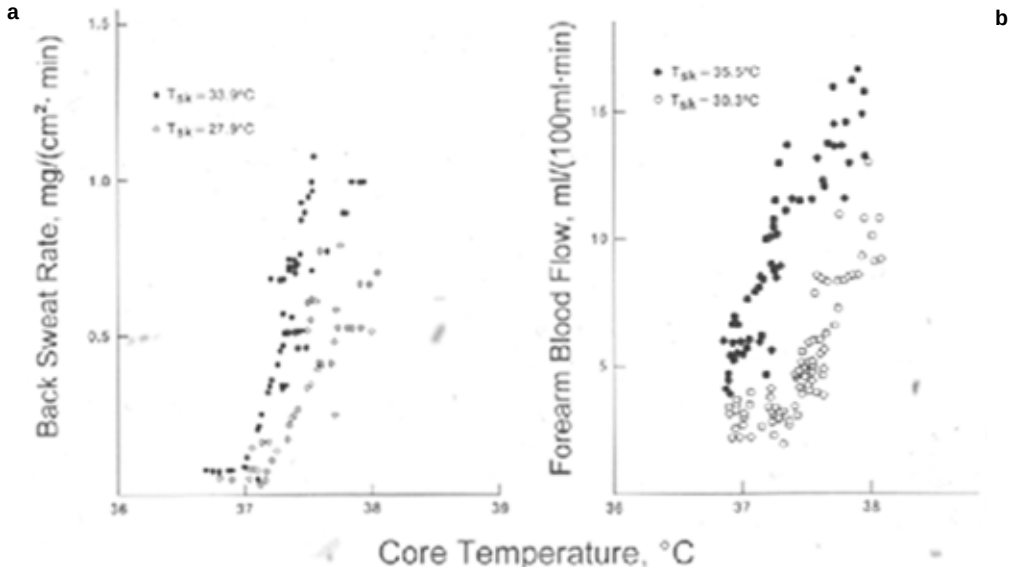

Fig. 2-10. The relations of (**a**) back (scapular) sweat rate and (**b**) forearm blood flow to core temperature and mean skin temperature ($\overline{T}_{sk}$). In the experiments shown, core temperature was increased by exercise. Adapted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise–heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes.* Indianapolis, Ind: Benchmark Press (now Cooper Publishing Group, Carmel, Ind); 1988: 101.

most entirely in the peripheral tissues (see Figure 2-2). For example, when someone enters a hot environment, his or her skin temperature rises and may elicit sweating even if there is no change in core temperature. On the other hand, an increase in heat production due to exercise elicits the appropriate heat-dissipating responses through a rise in core temperature.

Core temperature receptors involved in the control of thermoregulatory responses are concentrated especially in the hypothalamus,[42] but temperature receptors in other core sites, including the spinal cord and medulla, also participate.[42] The anterior preoptic area of the hypothalamus contains many neurons that increase their firing rate either in response to warming or in response to cooling, and temperature changes in this area of only a few tenths of a Centigrade degree elicit changes in the thermoregulatory effector responses of experimental mammals. Thermal receptors have been reported elsewhere in the core, including the heart, pulmonary vessels, and spinal cord; but the thermoregulatory role of core thermal receptors outside the central nervous system is not known.[8]

Let us consider what happens when a disturbance—say, an increase in metabolic heat production due to exercise—upsets the thermal balance. Heat is stored in the body, and core temperature rises. The thermoregulatory controller receives information about these changes from the thermal receptors, and responds by calling forth appropriate heat-dissipating responses. Core temperature continues to rise, and these responses continue to increase until they are sufficient to dissipate heat as fast as it is being produced, thus restoring heat balance and preventing further increases in body temperatures. The rise in core temperature that elicits heat-dissipating responses sufficient to reestablish thermal balance during exercise is an example of a *load error*[9]; a load error is characteristic of any proportional control system that is resisting the effect of some imposed disturbance or "load." Although the disturbance in this example was exercise, parallel arguments apply if the disturbance is a change in the environment, except that most of the temperature change will be in the skin and shell rather than in the core.

Relation of Effector Signals to Thermoregulatory Set Point

Both sweating and skin blood flow depend on core and skin temperatures in the same way, and changes in the threshold for sweating are accompanied by similar changes in the threshold for vasodilation.[4] We may therefore think of the central integrator (Figure 2-11) as generating one thermal command signal for the control of both sweating and skin blood flow. This signal is based on the information about core and skin temperatures that the integrator receives, and on the thermoregulatory *set point*.[4] We may think of the set point as the target level of core temperature, or the setting of the body's "thermostat." In the operation of the thermoregulatory system, it is a reference point that determines the thresholds of all the thermoregulatory responses.

Nonthermal Influences on Thermoregulatory Responses

Each thermoregulatory response may be affected by other inputs besides body temperatures and factors that affect the thermoregulatory set point. Nonthermal factors may produce a burst of sweating at the beginning of exercise,[43,44] and the involvement of sweating and skin blood flow in emotional responses is familiar to everyone.

Of the thermoregulatory responses that are important during heat stress, skin blood flow is most affected by nonthermal factors because of its involvement in reflexes that function to maintain cardiac output, blood pressure, and tissue oxygen delivery during heat stress, postural changes, and hemorrhage, and sometimes during exercise, especially in the heat.

Physiological and Pathological Changes to the Thermoregulatory Set Point

Several physiological and pathological influences change the thermoregulatory set point. Fever elevates core temperature at rest, heat acclimatization decreases it, and time of day and phase of the menstrual cycle change it in a cyclical fashion.[4–6] Core temperature at rest varies with time of day in an approximately sinusoidal fashion, reaching a minimum at night, several hours before awaking, and a maximum—which is one half to one Centigrade degree higher—in the late afternoon or evening (see Figure 2-3). Although this pattern coincides with patterns of activity and eating, it is independent of them, occurring even during bed rest and fasting. This pattern is an example of a *circadian* rhythm (ie, a rhythmic pattern in a physiological function with a period of about 1 day). During the menstrual cycle, core temperature is at its lowest point just before ovulation; over the next few days it rises one-half to one Centi-

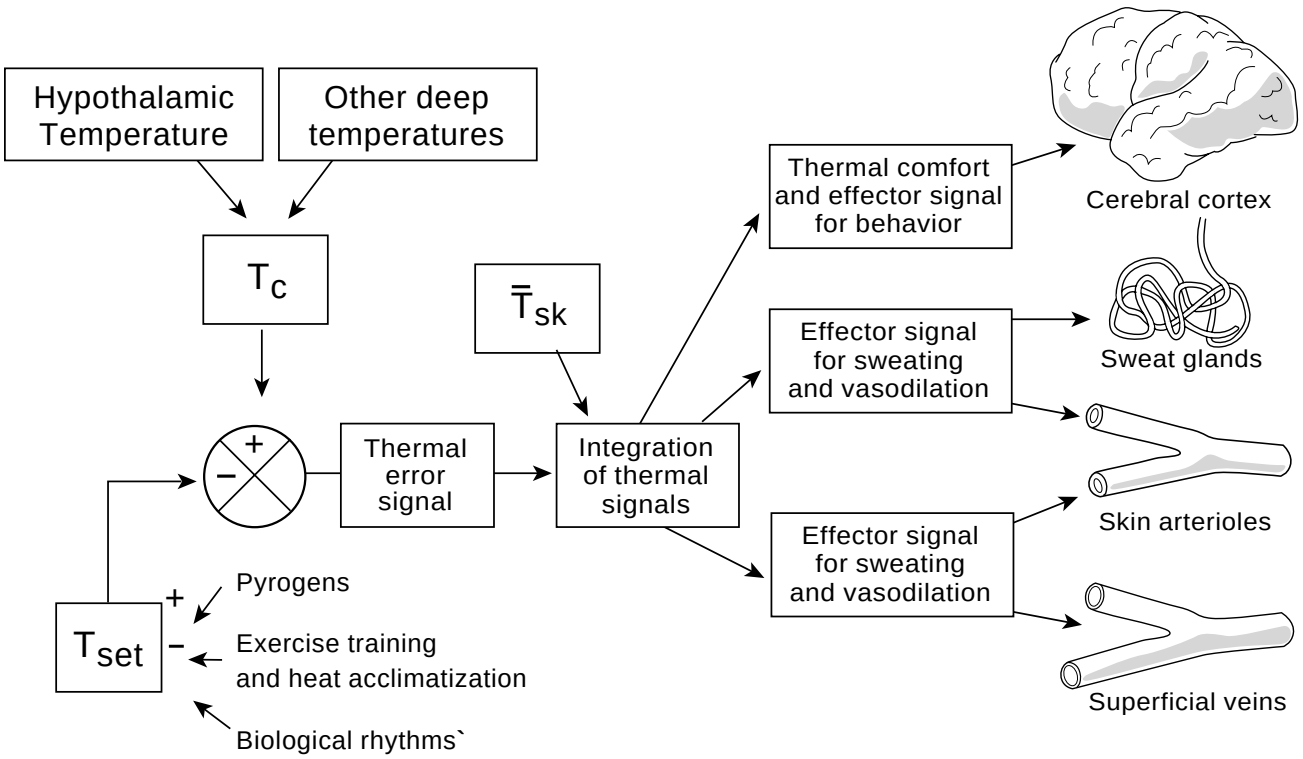

Fig. 2-11. Schematic diagram of the control of human thermoregulatory responses. The signs by the inputs to T_{set} indicate that pyrogens raise the set point, and heat acclimation lowers it. Core temperature, T_c, is compared with the set point, T_{set}, to generate an error signal, which is integrated with thermal input from the skin to produce effector signals for the thermoregulatory responses. Adapted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: 97–151.

grade degree and remains elevated for most of the luteal phase. Each of these factors—fever, heat acclimatization, the circadian rhythm, and the menstrual cycle—affects core temperature at rest by changing the thermoregulatory set point, thus producing corresponding changes in the thresholds for all the thermoregulatory responses.

Peripheral Modification of Skin Vascular and Sweat Gland Responses

The skin is the organ most directly affected by environmental temperature, and skin temperature affects heat loss responses not only through the reflex actions shown in Figure 2-10 but also through direct effects on the effectors themselves. Local temperature changes act on skin blood vessels in at least two ways. First, local cooling potentiates (and heating weakens) the constriction of blood vessels in response to nervous signals and vasoconstrictor substances.[36] Second, in skin regions where active vasodilation occurs, local heating dilates the blood vessels (and local cooling constricts them) through a direct action that is independent of nervous signals.[45,46] This effect is especially strong at skin temperatures above 35°C[46]; and when the skin is warmer than the blood, increased blood flow helps to cool the skin and protect it from heat injury.

The effects of local temperature on sweat glands parallel those on blood vessels, so that local heating magnifies (and local cooling reduces) the sweating response to reflex stimulation or to acetylcholine,[37] and intense local heating provokes sweating directly, even in sympathectomized skin.[47] During prolonged (several hours) heat exposure with high sweat output, sweat rates gradually diminish, and the sweat glands' response to locally applied cholinergic drugs is reduced also. The reduction of sweat gland responsiveness is sometimes called *sweat gland "fatigue."* Wetting the skin makes the stratum corneum swell, mechanically obstructing the sweat duct and causing a reduction in sweat

secretion, an effect called *hidromeiosis*.[48] The glands' responsiveness can be at least partly restored if the skin is allowed to dry (eg, by increasing air movement[49]), but prolonged sweating also causes histological changes, including depletion of glycogen, in the sweat glands.[50]

THERMOREGULATORY RESPONSES DURING EXERCISE

Vigorous exercise can increase oxygen consumption and heat production within the body 10-fold or more, depending on the individual's aerobic fitness. Unless exercise is very brief, it is soon accompanied by increases in the heat-dissipating responses—skin blood flow and sweating—to counter the increase in heat production. Although hot environments also elicit heat-dissipating responses, exercise ordinarily accounts for the greatest demands on the thermoregulatory system for heat dissipation, and exercise provides an important example of how the thermoregulatory system responds to a disturbance in heat balance.

Exercise and thermoregulation impose competing demands on the circulatory system. Exercise requires large increases in blood flow to exercising muscle, and the thermoregulatory responses to exercise require increases in skin blood flow. Muscle blood flow is several times as great as skin blood flow during exercise, but the increase in skin blood flow involves disproportionately large demands on the cardiovascular system, as discussed below. Moreover, if the water and electrolytes lost through sweating are not replaced, the resulting reduction in plasma volume will eventually create a further challenge to cardiovascular homeostasis.

Restoration of Heat Balance During Exercise

Exercise increases heat production so that it exceeds heat loss and causes core temperature to rise. The increase in core temperature, in turn, elicits heat-loss responses, but core temperature continues to rise until heat loss has increased enough to match heat production, so that heat balance is restored and core temperature and the heat-loss responses reach new steady state levels. Because the heat-loss responses are proportional to the increase in core temperature, the increase in core temperature at steady state is proportional to the rate of heat production, and thus to the metabolic rate.

A change in ambient temperature changes the levels of sweating and skin blood flow that are needed to maintain any given rate of heat dissipation. However, the change in ambient temperature is accompanied by a skin temperature change that elicits, via both direct and reflex effects, much of the required change in these responses. For any given rate of heat production, there is a range of environmental conditions (sometimes called the "prescriptive zone"; see Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues) within which ambient temperature changes elicit the necessary changes in heat-dissipating responses almost entirely through the effects of skin temperature changes, with virtually no effect on core temperature at steady state.[51] (The limits of this range of conditions depend on the rate of heat production, and on such individual factors as skin surface area and state of heat acclimatization.) Within this range, core temperature reached during exercise is nearly independent of ambient temperature; and for this reason it was once believed that the increase in core temperature during exercise is caused by an increase in the thermoregulatory set point,[52] just as during fever. As stated previously, however, the increase in core temperature with exercise is an example of a load error rather than an increase in set point. Note the following differences between fever and exercise (Figure 2-12):

- First, although heat production may increase substantially (through shivering) when core temperature is rising early during fever, it does n ot need to stay high to maintain the fever, but in fact returns nearly to prefebrile levels once the fever is established. During exercise, however, an increase in heat production not only causes the elevation in core temperature but is necessary to sustain it.
- Second, while core temperature is rising during fever, rate of heat loss is, if anything, lower than before the fever began; but during exercise, the heat-dissipating responses and the rate of heat loss start to increase early and continue increasing as core temperature rises. (Although in this chapter the term "fever" is used to mean specifically an elevation in core temperature due to pyrogens and occurring in connection with infection or other inflammatory process, some authors use "fever" more loosely to mean any significant elevation of core temperature.)

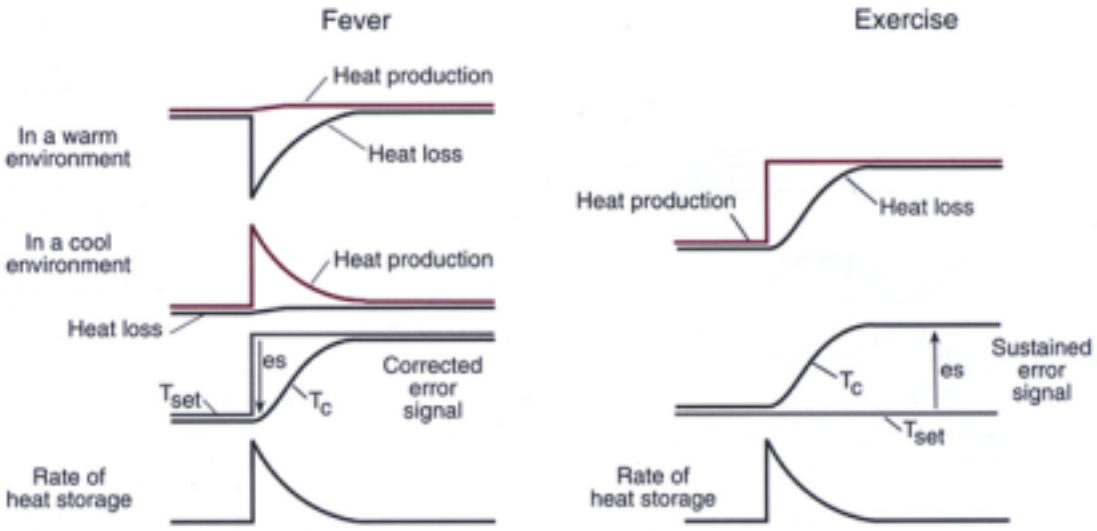

Fig. 2-12. Thermal events during the development of fever (left) and the increase in core temperature (T_c) during exercise (right). The error signal, es, is the difference between T_c and the set point, T_{set}. At the start of a fever, T_{set} has risen, so that T_{set} is higher than T_c, and es is negative. At steady state, T_c has risen to equal the new level of T_{set} and es is corrected (ie, it returns to zero). At the start of exercise, $T_c = T_{set}$ so that es = 0. At steady state, T_{set} has not changed but T_c has increased and is greater than T_{set}, producing a sustained error signal, which is equal to the load error. The error signal (or load error) is here represented with an arrow pointing down for $T_c < T_{set}$, and with an arrow pointing up for $T_c > T_{set}$. Adapted with permission from Stitt JT. Fever versus hyperthermia. *Fed Proc.* 1979;38:43.

Challenge of Exercise in the Heat to Cardiovascular Homeostasis

As pointed out earlier, skin blood flow increases during exercise in order to carry all of the heat that is produced to the skin. In a warm environment, where the temperature difference between core and skin is relatively small, the necessary increase in skin blood flow may be several liters per minute.

Impairment of Cardiac Filling

Whereas the work of supplying the skin blood flow required for thermoregulation in the heat may represent a heavy burden for a patient with cardiovascular disease,[53] in healthy subjects the primary cardiovascular burden of heat stress results from impairment of venous return.[29,30,54] As skin blood flow increases, blood pools in the large, dilated cutaneous vascular bed, thus reducing central blood volume and cardiac filling (Figure 2-13). Because stroke volume is decreased, a higher heart rate is required to maintain cardiac output. These effects are aggravated by a decrease in plasma volume if the large amounts of salt and water lost in the sweat are not replaced. Because the main cation in sweat is sodium, disproportionately much of the body water lost in sweat is at the expense of extracellular fluid, including plasma, although this effect is mitigated if the sweat is dilute.

Compensatory Cardiovascular Responses

Several reflex adjustments help to maintain cardiac filling, cardiac output, and arterial pressure during exercise and heat stress. The cutaneous veins constrict during exercise; and because most of the vascular volume is in the veins, constriction makes the cutaneous vascular bed less compliant and reduces peripheral pooling. Splanchnic and renal blood flow are reduced in proportion to the intensity of the exercise or heat stress. This reduction of blood flow has two effects. First, it allows a corresponding diversion of cardiac output to skin and exercising muscle. Second, because the splanchnic vascular beds are very compliant, a decrease in their blood flow reduces the amount of blood pooled in them[29,30] (see Figure 2-13), helping to compensate for decreases in central blood volume caused by reduced plasma volume and blood pooling in the skin. Because of the essential thermoregulatory function of skin blood flow during exercise and heat stress, the body preferentially compromises splanchnic and renal flow to maintain cardiovascular homeostasis.[55] Above a certain level of cardiovascular strain, however, skin blood flow, too, is compromised.

Despite these compensatory responses, heat stress markedly increases the thermal and cardiovascular strain that exercise produces in subjects

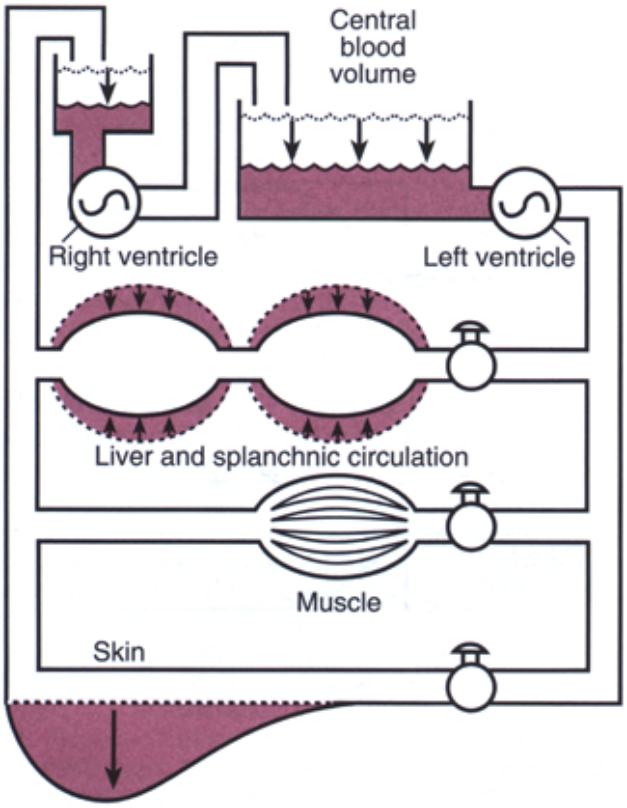

Fig. 2-13. Schematic diagram of the effects of skin vasodilation on peripheral pooling of blood and the thoracic reservoirs from which the ventricles are filled, and also the effects of compensatory vasomotor adjustments in the splanchnic circulation. The valves drawn at the right sides of liver/splanchnic, muscle, and skin vascular beds represent the resistance vessels that control blood flow through those beds. Arrows show the direction of the changes during heat stress. Adapted with permission from Rowell LB. Cardiovascular aspects of human thermoregulation. *Circulation Res*. 1983;52:367–379.

who are unacclimatized to heat. A comparison of responses on the first day of exercise on hot days with those on cool days shows some effects of unaccustomed environmental heat stress on the responses to exercise (Figure 2-14[56]). On the first day in the heat, heart rate during exercise reached a level about 40 beats per minute higher than in the cool environment, to help compensate for the effects of impaired cardiac filling and to maintain cardiac output; and rectal temperature during exercise rose one Centigrade degree higher than in the cool environment. Other effects of exercise–heat stress may include headache, nausea and vomiting secondary to splanchnic vasoconstriction, dizziness, cramps, shortness of breath, dependent edema, and orthostatic hypotension.

During prolonged exercise there is a gradual "drift" in several cardiovascular and thermoregulatory responses. This may include a continuous rise in heart rate, accompanied by a fall in stroke volume and reductions in aortic, pulmonary arterial, and right ventricular end-diastolic pressures.[57] Rowell named these changes "cardiovascular drift," and thought of them as appearing as early as after 15 minutes of exercise.[57] He and Johnson[57,58] empha-

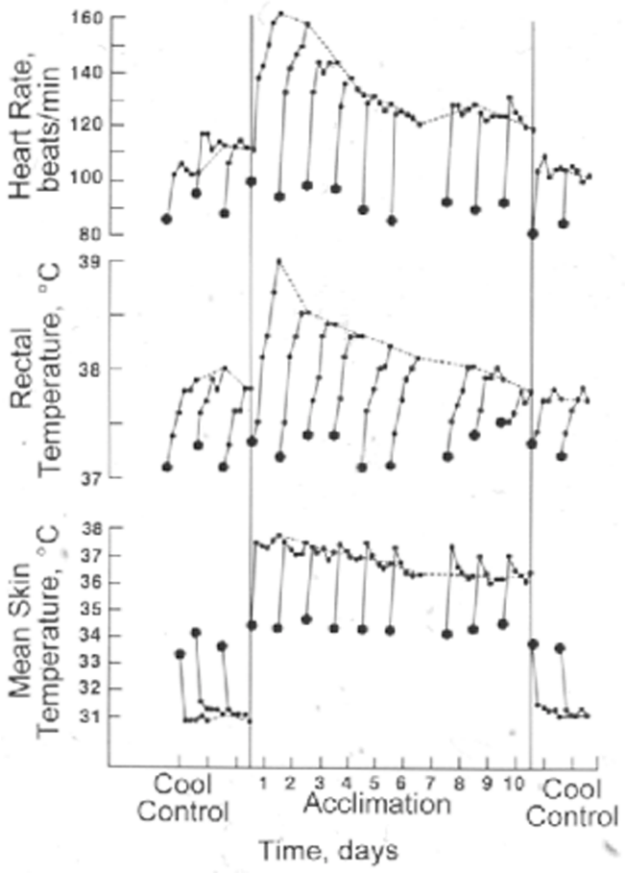

Fig. 2-14. Change in the responses of heart rate, rectal temperature, and mean skin temperature during exercise in a 10-day program of acclimation to dry heat (50.5°C, 15% relative humidity [rh]), together with responses during exercise in a cool environment before and after acclimatization. (The "cool control" conditions were 25.5°C, 39% rh.) Each day's exercise consisted of five 10-minute treadmill walks at 2.5 mph (1.12 m/s) up a 2.5% grade. Successive walks were separated by 2-minute rest periods. Large circles show values before the start of the first exercise period each day, small circles show values at the ends of successive exercise periods, and dotted lines connect final values each day. Adapted with permission from Eichna LW, Park CR, Nelson N, Horvath SM, Palmes ED. Thermal regulation during acclimatization in a hot, dry (desert type) environment. *Am J Physiol*. 1950;163:588.

sized the role of thermoregulatory increases in skin blood flow in producing cardiovascular drift. However, later authors[59–61] have described, as part of the picture of cardiovascular drift, an upward creep in core temperature, which may begin only after a period of apparent thermal steady state (eg, after 30–60 min of exercise). In some of these studies, most but not all of the changes in cardiovascular and thermoregulatory responses could be prevented by replacing fluid lost in sweat, suggesting that these changes were mostly secondary to changes in plasma volume and osmolality due to sweating. Other factors that may affect cardiovascular and thermoregulatory function during prolonged exercise include changes in myocardial function, changes in baroreceptor sensitivity or peripheral α-adrenergic receptor responsiveness (see Tibbits[62] and Raven and Stevens[63] for a discussion of these effects), or an upward adjustment of the thermoregulatory set point,[64] presumably due to some sort of inflammatory response and perhaps elicited by products of muscle injury. These effects have not been investigated extensively, and little is known about the underlying physiological or pathological mechanisms. Some of these effects have been reported only after several hours of exercise or near exhaustion, and little is known about the conditions of exercise duration and intensity required to produce them and their persistence after the end of exercise. Although their functional significance is, as yet, only poorly understood, these changes may be important in limiting performance during prolonged strenuous activity, such as forced marches.

FACTORS THAT ALTER HEAT TOLERANCE

Heat Acclimatization

Prolonged or repeated exposure to stressful environmental conditions elicits significant physiological changes, called *acclimatization,* which reduce the physiological strain that such conditions produce. (The nearly synonymous term, *acclimation,* is often applied to such changes produced in a controlled experimental setting.[12]) Figure 2-14 illustrates the development of these changes during a 10-day program of daily treadmill walks in the heat. Over the 10 days, heart rate during exercise decreased by about 40 beats per minute, and rectal and mean skin temperatures during exercise decreased more than 1°C. Because skin temperature is lower after heat acclimatization than before, dry (nonevaporative) heat loss is less (or, if the environment is warmer than the skin, dry heat gain is greater). To compensate for the changes in dry heat exchange, evaporative heat loss—and thus sweating—increases. The three classic signs of heat acclimatization are

- lower heart rate,
- lower core temperature, and
- higher sweat rate during exercise–heat stress.

Other changes include

- an increased ability to sustain sweat production during prolonged exercise-heat stress, which is essential to increasing tolerance time;
- decreased solute concentrations in sweat;
- redistribution of sweating from trunk to limbs;
- increases in total body water and changes in its distribution;
- metabolic and endocrine changes; and
- other poorly understood changes that protect against heat illness.

The overall effect of heat acclimatization on performance can be quite dramatic, so that acclimatized subjects can easily complete exercise in the heat, which previously was difficult or impossible. Figure 3-22 in Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues, in this textbook graphically shows the day-to-day improvement in performance during a 7-day program of heat acclimation.

At any given air temperature, increasing the humidity impedes evaporation of sweat (see Equation 6). To allow sweat to evaporate rapidly enough to maintain heat balance, the wetted area of skin must increase. The distribution of sweating may change to allow more of the skin surface area to be wetted, but wetter skin also favors development of hidromeiosis, limiting tolerance time by hampering maintenance of high sweat rates. Although heat acclimatization in a dry environment confers a substantial advantage in humid heat,[65,66] acclimatization in humid heat produces somewhat different physiological adaptations, corresponding to the characteristic physiological and biophysical challenges of humid heat.

Acquisition and Loss

A degree of heat acclimatization is produced either by heat exposure alone or by regular strenu-

ous exercise, which raises core temperature and provokes heat-loss responses. Indeed, the first summer heat wave produces enough heat acclimatization that after a few days most people notice an improvement in their feelings of energy and general well-being. However, the acclimatization response is greater if heat exposure and exercise are combined, causing a greater rise of internal temperature and more profuse sweating. Up to a point, the degree of acclimatization acquired is proportional to the daily heat stress and the amount of sweat secreted during acclimatization,[67] but full development of exercise–heat acclimatization does not require continuous heat exposure.

Continuous, daily 100-minute periods of heat exposure with exercise are widely considered sufficient to produce an optimal heat acclimatization response in dry heat. However, this notion is based chiefly on one study,[68] in which subjects' responses were evaluated only during 100-minute heat exposures, which provide little information about their ability to sustain heat-loss responses over time. An adequate assessment of heat tolerance may, in fact, require an exposure lasting several hours. For example, Strydom and Williams[69] compared responses of two groups of subjects during 4 hours of exercise in humid heat. Although the groups' responses were indistinguishable during the first hour, the responses of the more heat-tolerant group were clearly different from those of the less heat-tolerant group during the third and fourth hours.

Several factors affect the speed of development of heat acclimatization. However, most of the improvement in heart rate, skin and core temperatures, and sweat rate typically is achieved during the first week of daily exercise in a hot environment, although there is no sharp end to the improvement.[70] Heart rate shows the most rapid reduction,[71–73] most of which occurs in 4 to 5 days.[71] After 7 days, the reduction in heart rate is virtually complete and most of the improvement in skin and core temperatures has also occurred[72,74]; and the thermoregulatory improvements are generally believed to be complete after 10 to 14 days of exposure.[75] The improved sweating response[71,74] and ease of walking[72,74] reported during heat acclimatization may take 1 month to develop fully, and resistance to heatstroke may take up to 8 weeks.[76] Experimental heat acclimation (physiological adjustment to an environment, in a controlled setting) develops more quickly in warm weather,[66] probably because subjects are already partly acclimatized.

High aerobic fitness hastens development of acclimatization.[72,77] Aerobic exercise elevates core temperature and elicits sweating even in a temperate environment, and aerobic training programs involving exercise at 70% of maximal oxygen uptake ($\dot{V}O_2max$) or more[78,79] produce changes in the control of sweating similar to those produced by heat acclimatization. There has, however, been much disagreement as to whether or not aerobic training in a temperate environment induces true heat acclimatization. In a critical review of the evidence and arguments on both sides of the issue, Gisolfi and Cohen[80] concluded that exercise training programs lasting 2 months or more in a temperate environment produce substantial improvement in exercise heat tolerance. However, exercise training alone has not been shown to produce a maximal state of exercise–heat tolerance.

The benefits of acclimatization are lessened or undone by sleep loss, infection, and alcohol abuse[71,81]; salt depletion[71]; and dehydration.[71,82,83] Heat acclimatization gradually disappears without periodic heat exposure, although partial losses due to a few days' lapse are easily made up.[81] The improvement in heart rate, which develops more rapidly, also is lost more rapidly than are the thermoregulatory improvements.[68,77,84,85] However, there is much variability in how long acclimatization persists. In one study, for example, acclimatization almost completely disappeared after 17 days without heat exposure[86]; but in another study, approximately three quarters of the improvement in heart rate and rectal temperature was retained after 18 days without heat exposure.[77] Physically fit subjects retain heat acclimatization longer[65,66]; and warm weather may[66] or may not[85] favor persistence of acclimatization, although intermittent exposure to cold seems not to hasten the loss of heat acclimatization.[87]

Changes in Thermoregulatory Responses

After acclimatization, sweating during exercise starts earlier and the core temperature threshold for sweating is lowered. Acclimatization also increases the sweat glands' response to a given increment in core temperature and also their maximum sweating capacity. These latter changes reflect changes in the individual glands rather than in the nervous systems signals to the glands, because after acclimatization the glands also produce more sweat when stimulated with methacholine,[88,89] which mimics the effect of acetylcholine.

In an unacclimatized person, sweating is most profuse on the trunk; but during acclimatization in humid heat, the fraction of sweat secreted on the

limbs increases,[90–93] enabling an acclimatized person to make better use of the skin surface for evaporation and achieve higher rates of evaporative heat loss. During a heat stress lasting several hours, sweat rates that were initially high tend gradually to decline as the heat stress continues. Although several mechanisms may contribute to the decline, much of the decline is due to hidromeiosis, associated with wetness of the skin, and the decline is most pronounced in humid heat. After acclimatization to humid heat, this decline of sweat rate occurs more slowly[67] (Figure 2-15), so that higher sweat rates can be sustained and tolerance time is prolonged. This effect of acclimatization appears to act directly on the sweat glands themselves, and during acclimatization to dry heat it can be produced selectively on one arm by keeping that arm in a humid microclimate (eg, inside a plastic bag).[94]

Because heat acclimatization is an example of a set-point change,[4,95] thresholds for sweating and cutaneous vasodilation both are reduced in such a way that vasodilation and the onset of sweating accompany each other after acclimatization in the same way as before,[96] and heat transfer from core to skin is maintained at the lower levels of core and skin temperature that prevail after acclimatization. These changes by themselves say nothing about the effect of acclimatization on the levels of skin blood flow reached during exercise–heat stress. In many studies[56,97] (especially those using dry heat), heat acclimatization was found to widen the core-to-skin temperature gradient, presumably allowing heat balance to be reached with a lower level of skin blood flow and a lesser cardiovascular strain. Even in relatively dry heat, however, acclimatization to heat does not always widen the core-to-skin temperature gradient.[72]

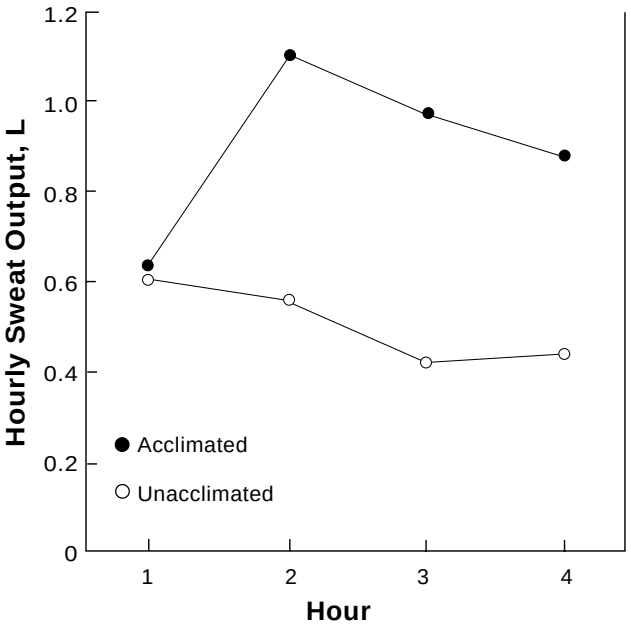

Fig. 2-15. Sweat rates during 4 hours' exercise (bench stepping, 35-W mechanical power) in humid heat (33.9°C dry bulb, 89% relative humidity, 35 mm Hg ambient vapor pressure) on the first and last days of a 2-week program of acclimatization to humid heat. Adapted with permission from Wyndham CH, Strydom NB, Morrison JF, et al. Heat reactions of Caucasians and Bantu in South Africa. *J Appl Physiol.* 1964;19:601.

Nonthermoregulatory Changes

On the first day of exercise in the heat, heart rate reaches much higher levels than in temperate conditions (see Figure 2-14), and stroke volume is lower. Thereafter, heart rate decreases and stroke volume usually, but not always, increases. Orthostatic tolerance also improves with heat acclimatization.[95] Several mechanisms participate in these changes, but their relative contributions are not known and probably vary. Plasma volume at rest expands during the first week of acclimatization and contributes to the reduction in heart rate and circulatory strain; however, if acclimatization continues, plasma volume at rest returns toward control levels after 1 or 2 weeks,[74,98–100] whereas the improvements in cardiovascular function persist. In addition, it is likely that a decrease in peripheral pooling of blood helps to support cardiovascular function in acclimatized subjects. When a decrease in skin blood flow (which is allowed by a widened core-to-skin temperature gradient) occurs, it presumably decreases peripheral pooling of blood. In addition, an increase in venous tone might substantially decrease pooling of blood, since venoconstriction can mobilize up to 25% of the blood volume.[98] The information available about such changes,[101–103] however, is very limited and far from conclusive.

Heat acclimatization increases total body water, but there is much variability both in the total increase and in its distribution among the various fluid compartments.[95] Much of the increase is accounted for by an expansion of plasma volume at rest, which develops rapidly at first and continues more slowly for about a week. The resulting increase in blood volume ranges from 12% to 27%.[104] The mechanisms responsible for this expansion are unclear, but may include an increase in extracellular fluid—ranging from 6% to 16%[104]—due to salt

retention, and a net fluid shift from interstitial space to plasma due to an increase in the mass of protein in the plasma.[105,106]

At the start of acclimatization, secretion of adrenocorticotropic hormone (ACTH) increases in response to the circulatory strain caused by heat stress. The adrenal cortex responds to ACTH by increasing secretion of cortisol and aldosterone. If salt intake is insufficient to replace losses in sweat, the resulting sodium depletion also acts via the renin–angiotensin system to increase aldosterone secretion. Cortisol and aldosterone both contribute to sodium retention: by the kidneys within a few hours, and by the sweat glands after 1 to 2 days. Exercise and heat stress also elicit secretion of aldosterone[107,108] through the renin–angiotensin system. Within a few days the sodium-conserving effects of aldosterone secreted via this pathway are sufficient to restore and maintain sodium balance, and ACTH secretion returns to normal. Depending on sodium intake, the kidneys may eventually "escape" the effects of aldosterone and excrete as much sodium as needed to maintain sodium balance. The sweat glands, however, do not escape but continue to conserve sodium as long as acclimatization persists.

An unacclimatized person may secrete sweat with a sodium concentration as high as 60 mEq/L, corresponding to 3.5 grams of NaCl per liter, and can lose large amounts of salt in the sweat (Figure 2-16). With acclimatization, the sweat glands conserve sodium by secreting sweat with a sodium concentration as low as 5 mEq/L.[27] Acclimatized men in whom sodium conservation is maximally developed can sweat up to 9 L/d and stay in salt balance on 5 grams of NaCl per day.[109,110] Maximal development of sodium-conserving capacity was accomplished with a program that combined gradual reduction of dietary sodium intake with daily exercise in the heat. However, most whites who are not secreting large volumes of sweat and are in salt balance with an intake of 10 grams of NaCl per day (a typical intake for a western diet) have high concentrations of sodium in the sweat.[111] If suddenly required to secrete large volumes of sweat, they may undergo a substantial net loss of sodium before their mechanisms for sodium conservation become fully active. Therefore, subjects who are exercising in a hot environment and are either unacclimatized or not consuming a normal diet should receive 10 grams of supplemental salt per day unless water is in short supply.[111] However, salt supplements are not recommended for acclimatized subjects performing heavy exercise in the heat if they are eating a normal diet and are not salt depleted.

Fig. 2-16. Salt deposited on a soldier's uniform by evaporation of sweat. Photograph: Courtesy of Robert E. Burr, MD, Natick, Massachusetts.

The mineralocorticoids, aldosterone and desoxycorticosterone, have been administered to subjects just before or during heat acclimatization programs.[98,104,112,113] Mineralocorticoid administration produced some responses characteristic of heat acclimatization, but neither produced a state equivalent to what the subjects attained as a result of undergoing heat acclimatization nor reduced the time necessary to reach that state. However, because of the way these studies were designed, their results do not support definite conclusions about the role of endogenous aldosterone in heat acclimatization.[95]

Effects on Heat Disorders

The harmful effects of heat stress operate through cardiovascular strain, fluid and electrolyte loss, and, especially in heatstroke, tissue injury whose mechanism is not well understood but evidently involves more than just high tissue temperatures. The topic is also discussed in Hubbard[114] and in Chapter 5, Pathophysiology of Heatstroke, in this textbook.

Heat syncope is a temporary circulatory failure due to pooling of blood in the peripheral veins and the resulting decrease in diastolic filling of the heart. Although a large increase in thermoregulatory skin blood flow is the direct cause of the peripheral pooling, an inadequate baroreflex response is probably an important contributing factor. Heat acclimatization rapidly reduces susceptibility to heat syncope, as expected from the improvement in orthostatic tolerance,[101,115,116] noted earlier.

Like heat syncope, heat exhaustion is believed to result from a decrease in diastolic filling. However, dehydration with resulting hypovolemia has a major role in the development of heat exhaustion; the baroreflex responses usually are strong enough to prevent syncope, and also account for much of the symptomatology. Little is known about the effect of acclimatization on susceptibility to heat exhaustion.

Heatstroke is the most severe heat disorder; and without prompt, appropriate treatment, mortality may be high. Typical victims of the exertional form, in which a high rate of metabolic heat production is a primary pathogenic factor, are athletes or military personnel—especially recruits. During World War II, the incidence of fatal heatstroke was low after 8 weeks of training,[76] by which time the recruits were well acclimatized. Much of the protective effect of acclimatization is presumably owing to thermoregulatory improvement, but acclimatization and physical conditioning may also protect in ways that are poorly understood, since rectal temperatures above 41°C have been measured in runners competing in marathons with no apparent ill effect.[117,118]

A small proportion of apparently healthy individuals cannot acclimatize to heat.[119,120] In South African gold-mining recruits (the population studied most extensively in this regard) individuals who do not acclimatize are, on average, smaller, older, and less aerobically fit than those who do.[120]

Physical Fitness, Gender, and Age

Individuals with low physical fitness tend to have reduced heat tolerance and less sensitive sweating responses. Obesity also is associated with reduced heat tolerance. To a large extent, the effect of obesity is explained by its relation to physical fitness, but other factors contribute as well.[121]

Women as a group are less tolerant to exercise–heat stress than men. However, the gender difference largely disappears when subjects are matched according to size, acclimatization, and $\dot{V}O_2max$.[121] The exertional form of heatstroke is often said to be quite rare in women,[122] and perhaps women enjoy a degree of protection against exertional heatstroke for either physiological or behavioral reasons. Women are susceptible to exertional heatstroke, however, and in active-duty soldiers (a population in which most heatstroke is of the exertional type), annual incidence rates of heatstroke in women are at least half of those in men.[123] Although the thermoregulatory set point changes with the phase of the menstrual cycle, as discussed earlier, the phase of the menstrual cycle has not been shown to affect tolerance or performance during exercise in the heat (for a review, see Stephenson and Kolka[124]). It may be, however, that studies of exercise at different phases of the menstrual cycle have not used exercise of sufficient intensity or duration to demonstrate an effect. In fact, Pivarnik and associates,[125] studying women's responses during exercise in a temperate environment (22°C), found that after 60 minutes of exercise heart rate was 10 beats per minute higher in the luteal phase than in the follicular; and that rectal temperature increased 1.2 Centigrade degrees in the luteal phase and was still rising, while it increased 0.9 Centigrade degrees in the follicular phase and was near steady state. Although they examined only one set of experimental conditions, their data, when extrapolated to warmer environments, suggest a decline in tolerance to exercise–heat stress during the luteal phase. Advancing age also is associated with a decline in heat tolerance. Most of the decline disappears, however, if effects of chronic disease, adiposity, and reduced physical fitness are eliminated.[126]

Drugs and Disease

Many drugs inhibit sweating, most prominently those used for their anticholinergic effects, such as atropine and scopolamine. Intramuscular injection of 2 mg atropine (the dose in one autoinjector for acute treatment of exposure to nerve agent) inhibits sweating sufficiently to cause a noticeable impairment of thermoregulation during walking in dry heat.[127] Some drugs used for other purposes, such as glutethimide (a sleep medicine), tricyclic antidepressants, and phenothiazines (tranquilizers and antipsychotic drugs) also have some anticholinergic action; and all of these, plus several others, have been associated with heatstroke.[128,129] A 30-mg oral dose of pyridostigmine bromide (the dose

given thrice daily for pretreatment against nerve agent) reduced thermoregulatory vasodilation during moderate exercise in a warm environment,[130] and may potentially impair thermoregulation under more severe heat-stress conditions.

Both chronic and acute disorders may reduce heat tolerance. Untreated hypertension impairs the circulatory responses to heat stress. The effect of treated hypertension on heat tolerance is not known, but there are theoretical reasons for suspecting that some drugs used to treat hypertension may impair heat tolerance.[121] Congestive heart failure substantially impairs both sweating and the circulatory responses to heat stress, and moderate heat exposure worsens the signs and symptoms of congestive heart failure.[53] Neurological diseases involving the thermoregulatory structures in the brainstem can impair thermoregulation. Although hypothermia may result, hyperthermia is more usual and typically is accompanied by loss of sweating and the circadian rhythm. Several skin diseases impair sweating sufficiently that heat exposure, especially combined with exercise, may produce dangerously high body temperatures. Ichthyosis and anhidrotic ectodermal dysplasia can profoundly limit the ability to thermoregulate in the heat. In addition, heat rash (miliaria rubra)[131] and even mild sunburn[132] impair sweating and may reduce tolerance to exercise in the heat. The thermoregulatory effects of heat rash may persist for a week or longer after the appearance of the skin has returned to normal.[131]

SUMMARY

The body may be divided into an internal core, which includes the vital organs, and a superficial shell. Tissue temperature is fairly uniform throughout the core. Core temperature is regulated by the thermoregulatory system and is relatively unaffected by changes in environmental conditions. The temperature of the shell is not uniform, and varies both from point to point within the shell and with changes in environmental conditions. Most heat exchange between the body and the environment occurs at the skin surface, by convection, radiation, and evaporation. These three modes of heat exchange depend on the temperature and degree of wetness of the skin, and on environmental conditions including air movement, the temperature and moisture content of the air, and the temperatures of radiating surfaces in the environment.

The body controls heat flow between core and skin by controlling skin blood flow. Changes in skin blood flow affect skin temperature, and thus controlling skin blood flow provides a means of influencing heat exchange with the environment by convection and radiation. However, the effect of skin blood flow on heat exchange with the environment is limited in the heat, and the body cannot dissipate heat by convection and radiation if the environment is warmer than the skin. Secretion of sweat wets the skin, and sweating increases evaporative heat loss, as long as the environmental conditions allow the sweat to evaporate. Large amounts of heat can be dissipated by evaporation of sweat: sweat rates of 1L/h (corresponding to a rate of heat loss of about 675 W) can be sustained for many hours, and higher rates can be achieved for shorter periods.

Sweating and skin blood flow are controlled via the sympathetic nervous system, and these responses are graded according to elevations in core and skin temperatures. The operation of the thermoregulatory system is governed by the thermoregulatory set point, which we may think of as the setting of the body's "thermostat." The set point varies in a cyclical fashion, with an amplitude of 0.5 to 1.0 Centigrade degrees, according to time of day and, in women, the phase of the menstrual cycle, and it is elevated during fever.

Vigorous exercise can increase heat production within the body 10-fold or more. Because of the levels of skin blood flow needed for high rates of heat dissipation in a hot environment, exercise and heat dissipation make competing demands on the cardiovascular system. Moreover, if water and electrolytes lost as sweat are not replaced, plasma volume eventually is depleted. For these reasons, heavy exercise in the heat may seriously challenge cardiovascular homeostasis.

Heat tolerance is increased by aerobic exercise training and by acclimatization to heat. Acclimatization to heat develops quickly: the effectiveness of the heat-dissipating arm of the thermoregulatory system and exercise performance in the heat show pronounced improvements within a week. Conversely, poor physical fitness and certain disease states and drugs are associated with impairment of the thermoregulatory responses.

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Chapter 3

PHYSICAL EXERCISE IN HOT CLIMATES: PHYSIOLOGY, PERFORMANCE, AND BIOMEDICAL ISSUES

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INTRODUCTION

US Army fighting doctrine states that "US Army forces must be prepared to fight and win on short notice anywhere in the world, from blistering deserts to frigid wastelands, in rain forests—and all types of terrain"[1(p14-1)] and that soldiers are the most important and most vulnerable part of the war fighting system. Troops participating in almost all military deployments (including arctic regions[2]) are likely to encounter heat stress that must be managed for successful mission accomplishment. Military operations conducted in oppressively hot climates (eg, the Pacific and North African campaigns in World War II, and the Vietnam and Persian Gulf wars) require troops to perform strenuous exercise for long hours and in conditions that will push them to their physiological limits. Humans are tropical animals and (if heat-acclimatized, given adequate shade and water, and able to limit their physical activity) can tolerate extended exposure to any climatic heat stress on earth.[3] However, some military situations, such as work in boiler rooms, firefighting, and wearing protective clothing in hot environments, involve heat stress conditions so severe they cannot be tolerated for extended periods.[4–7] In addition, mission requirements that demand intense physical activity and the degraded physical or nutritional status of the troops often make successful heat stress management during military operations very difficult.

Heat stress results from the interaction of climatic conditions, body heat production, and the wearing of clothing or equipment or both that impedes heat loss. Heat stress generates a need for sweating and circulatory responses to dissipate body heat, especially when the environment is warmer than skin, and may push the body's homeostatic systems to their limits. Substantial levels of heat stress can occur even in cool climates, especially during intense exercise or while wearing clothing whose thermal properties are inappropriate to the environment and level of metabolic heat production.[8] The problem of heat stress due to thermally inappropriate clothing is especially pronounced in nuclear, biological, chemical (NBC) warfare protective ensembles, because their high thermal insulation and low permeability to water vapor greatly impede heat loss, and thus exacerbate heat stress.[9,10]

Climatic Heat Stress

Wet bulb globe temperature (WBGT) is commonly used to quantitate environmental heat stress in military, occupational, and sports applications.[11–15] The outdoor and indoor WBGT, natural wet bulb, dry bulb, and black globe temperatures are related in the following manner[16]:

1. Outdoor WBGT = 0.7 natural wet bulb + 0.2 black globe = 0.1 dry bulb
2. Indoor WBGT = 0.7 natural wet bulb + 0.3 black globe

where *natural wet bulb* is defined as the wet bulb temperature under conditions of prevailing air movement; *black globe*, the temperature inside a blackened, hollow, thin copper globe, in which the thermometer is located in the center of the sphere; and *dry bulb* (T_{db}), the temperature of the air surrounding the body. WBGT is an empirical index of climatic heat stress, and is used to determine the permitted level of physical activity and strategies to minimize the risk of heat injury. WBGT was originally developed for resting comfort and was later adapted for light-intensity physical exercise.[17] WBGT does not include any considerations for clothing or exercise intensity (metabolic rate), so it cannot predict heat exchange between a person and the environment.[11] The risk of heat injury at any given WBGT is greater in humid conditions; therefore, different guidance tables must be used in climates with low, moderate, and high humidity.[11]

Figure 3-1 is a global map of WBGT during July, the hottest month in the northern hemisphere.[18] During July, much of North America, South America, Europe, and Asia have WBGT values above 29°C (85°F). Depending on the climate, such high WBGT values can be achieved either through high humidity,[19] as reflected in high wet bulb temperature, or through high air (dry bulb) temperature and solar load,[3] as reflected in black globe temperature.

Temperature Regulation

Body temperature is regulated through two parallel processes: behavioral temperature regulation and physiological temperature regulation (this topic is also discussed in Chapter 2, Human Adaptation to Hot Environments). Examples of behavioral temperature regulation include seeking shade, discontinuing exercise, or removing clothing or equipment, or both. Among military personnel, behavioral thermoregulatory drives are often overridden by motivation to successfully complete the mission.

Physiological temperature regulation operates through graded heat loss responses (chiefly sweating

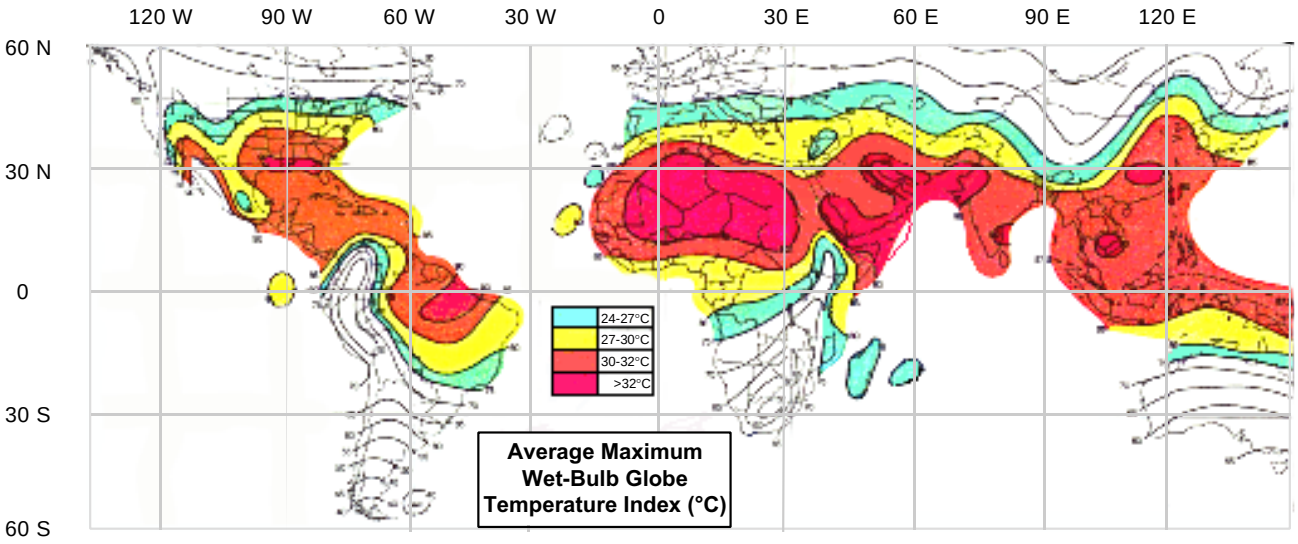

Fig. 3-1. The average wet bulb globe temperature (WBGT) index in the northern hemisphere during July. Adapted from John B. Stennis Space Center. *Global Climatology for the Wet Bulb Globe Temperature (WBGT) Heat Stress Index*. Bay St. Louis, Miss: Gulf Weather; 1989: 37, 54, 71.

and skin blood flow), which are proportional to the disturbance in core temperature and modified according to information from temperature receptors in the skin. If some environmental or metabolic heat stress, such as a higher ambient temperature or increased heat production due to exercise, upsets the body's thermal balance, then the body will store heat and the temperature of the core or skin or both, will increase. In response to these temperature increases, the body will increase its heat loss responses. Unless the heat stress exceeds the capacity of the thermoregulatory system, the heat loss responses will increase until they are sufficient to restore heat balance, so that core temperature stops increasing and reaches a new steady state level, which persists as long as the conditions of exercise and environmental heat stress continue. The body temperature increase during exercise–heat stress is an example of a load "error," which, in this case, is the core temperature increase above set point. In contrast, if environmental conditions, clothing, or equipment impose a biophysical limit for heat loss that is less than the rate of body heat production, then increases in *effector responses* (ie, increased sweating and skin blood flow) will not restore heat balance but will only result in increased physiological strain.

BODY TEMPERATURES

Body temperatures can arbitrarily be divided into deep body (ie, core) and surface (ie, skin) measurements. Deep body and surface temperatures will vary depending on their location and the perturbation of the environment and physical work task required of the soldier.

Core and Skin Temperatures

There is no one "true" core temperature because of temperature differences among different core sites. However, temperatures at all core sites are close to (within about one Centigrade degree) central blood temperature at thermal steady state. Core temperature is measured clinically in the rectum, mouth, tympanum, and auditory meatus. For research and for specialized purposes, core temperature is also measured in the esophagus and gastrointestinal tract. These sites differ in the speed with which their temperatures respond to changes in central blood temperature, and their susceptibility to being biased by extraneous influences, such as environmental temperature or breathing through the mouth. Table 3-1 summarizes the advantages and disadvantages of different core temperature measurement sites.[20]

Esophageal temperature is measured with a sensor at the level of the left atrium, where the heart and esophagus are in contact and virtually isothermal for several centimeters.[21] Esophageal temperature responds rapidly (time constant $\approx$ 1 min) and quantitatively to changes in central blood tempera-

TABLE 3-1

ADVANTAGES AND DISADVANTAGES OF CORE TEMPERATURE MEASUREMENT SITES

Anatomical Site	Advantage	Disadvantage
Esophagus	Accurate; rapid response	Affected by swallowing; can be uncomfortable
Mouth	Easy to use	Affected by drinking and mouth-breathing
Gastrointestinal Tract	Easy to use (pill form)	Affected by location of pill in GI tract (location is variable and uncertain) Affected by drinking while in stomach
Rectum	Accurate	Slow response; can be uncomfortable
Tympanum, Auditory Meatus	Easy to use; rapid response	Inaccurate; affected by ambient and skin temperatures; can be uncomfortable

Adapted from Levine L, Sawka MN, Gonzalez RR. *General Procedures for Clothing Evaluations Relative to Heat Stress.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1995. Technical Note 95-5.

ture,[22–24] and most thermal physiologists consider esophageal temperature to be the best noninvasive index of core temperature in humans.[25] Gastrointestinal tract temperature is obtained from a swallowed pill-like sensor, which telemeters a temperature signal to a receiver outside the body.[26–28] As the pill moves through the gastrointestinal tract its temperature will change with its location. Therefore, temperature measurements with the pill are, in general, somewhat more variable than are other core temperature measurements. However, this method is well suited to measuring core temperature in free-moving subjects because it requires no leads passing through body orifices, and the signal can be relayed to a monitoring station some distance away. A swallowed sensor therefore provides a useful means for monitoring workers in heat stress situations in which prediction of tolerance is difficult or unreliable.

Skin temperature is measured (*a*) to calculate the mean body temperature for heat storage determinations, (*b*) to calculate dry (radiative [R] plus convective [C]) heat exchange and skin conductance, and (*c*) to provide an index of the skin temperature input to the thermoregulatory controller. Because the skin represents the boundary between two media—tissue and the ambient air—changes in skin temperature may result from physiological adjustments (eg, cutaneous blood flow, sweat evaporation) or alterations in the environment (eg, air motion, temperature, and radiation). Skin temperature varies with ambient temperature and also according to the site where it is measured (Figure 3-2). Although a single skin temperature measurement can be useful for biophysical calculations, scientists are more often interested in the average, or mean skin temperature.[25,29] Mean skin temperature is calculated from individual local temperature measurements, usually weighted based on the percentage of body surface area that is represented by each body region at which temperature is measured. Early investigators[30,31] used 12 to 15 skin regions to calculate mean skin temperature, but, more recently, some investigators[32–34] have attempted to minimize the number of sites necessary for a valid estimate of mean skin temperature.

Exercise in Heat

When performing physical work in temperate or hot climates, a soldier's core temperature will increase. The magnitude of core temperature rise will depend on the environmental conditions, exercise task, and the soldier's biomedical state.

Influence of Metabolic Rate and Climate

Figure 3-3 illustrates that during physical exercise, core (esophageal and rectal) temperature initially increases rapidly and subsequently increases at a reduced rate until essentially steady state levels are achieved.[29] The core temperature increase represents the storage of metabolic heat, which is a by-product of skeletal muscle contraction. At the initiation of exercise, the metabolic rate increases immediately; however, the thermoregulatory effector responses for heat dissipation respond more slowly. Once these heat loss mechanisms increase sufficiently to balance metabolic heat production, a new steady state level of core temperature is achieved.

During muscular exercise, the magnitude of the

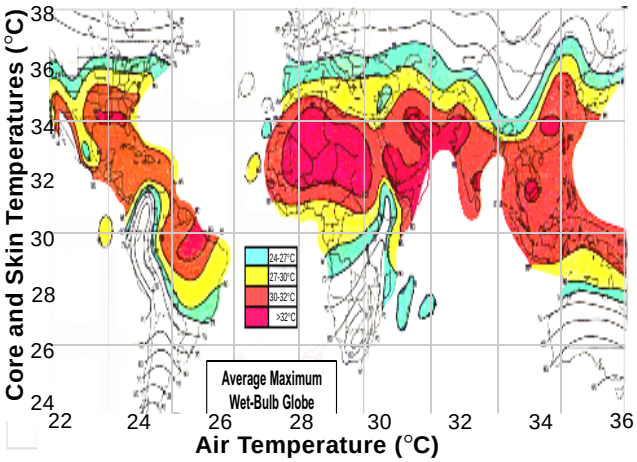

Fig. 3-2. Relationship between selected regional skin temperatures and core (rectal) temperature at rest over a range of temperate and hot climatic conditions. Adapted with permission from Hardy JD, DuBois. Basal metabolism and heat loss of young women at temperatures from 22°C to 38°C. *J Nutr*. 1941;21:383–404.

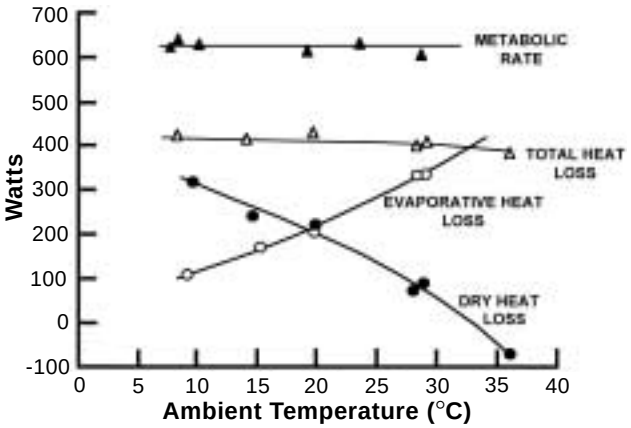

Fig. 3-4. Heat exchange during muscular exercise in a broad range of ambient temperatures. The figure shows the metabolic rate as constant at approximately 630 W. The figure also shows a fairly constant heat loss over the total temperature range. What changes is the partition between evaporative and dry heat loss. Reprinted from Nielsen M. Die Regulation der Körpertemperatur bei Muskelarbeit. *Scand Arch Physiol*. 1938;9:216.

core temperature increase at steady state is independent of climatic conditions over a fairly wide range, and is proportional to the metabolic rate.[35–37] Figure 3-4 presents heat exchange data during physical exercise (cycle ergometer at ~ 147 W mechanical power output; metabolic rate ~ 650 W) in a broad range of environmental conditions (5°C–36°C dry bulb temperatures with low humidity).[38]

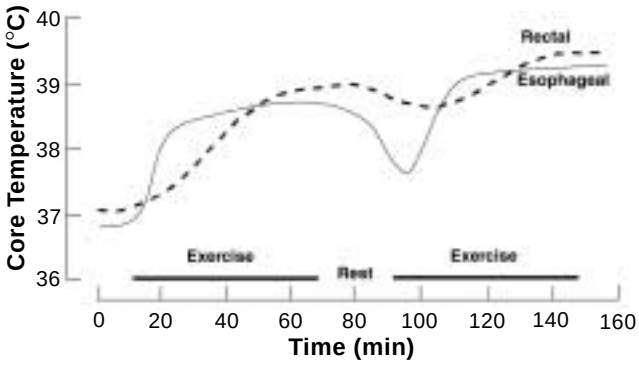

Fig. 3-3. Core (esophageal and rectal) temperature responses during exercise and recovery. Reprinted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise–heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 110.

The difference between metabolic rate and total heat loss represents the sum of energy used for mechanical work and stored as heat. Note that the total heat loss and, therefore, the heat storage and core temperature increase, are constant for each climate. The relative contributions of dry and evaporative heat exchange to the total heat loss, however, vary with climatic conditions. At 10°C, the large skin-to–ambient temperature gradient facilitates dry heat exchange, which is approximately equal to 70% of the total heat loss. As the ambient temperature increases, this gradient for dry heat exchange diminishes and there is a greater reliance on evaporative heat exchange. When the ambient temperature is equal to skin temperature, evaporative heat exchange will account for essentially all of the heat loss; and when the ambient temperature exceeds the skin temperature, there is dry heat gain to the body.

The concept that the core temperature increase during exercise is independent of the environment seems contrary to experience. Part of the reason for the discrepancy is that the feeling of being hot depends on more factors than core temperature (eg, skin temperature and cardiovascular strain). In addition, there are biophysical limits to heat exchange between the body and the environment[39,40] so that the core temperature increase during exercise is independent of the environment only within a range of conditions called the *prescriptive zone*.[35] Figure 3-5 illustrates this concept by presenting steady state

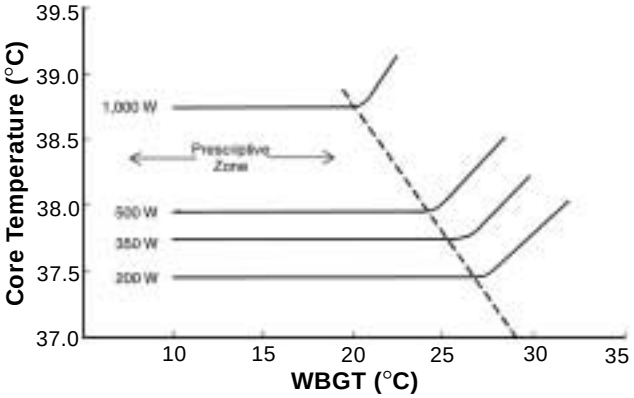

Fig. 3-5. Steady state core temperature responses of seminude subjects during exercise, as related to metabolic rate (expressed in watts [W]) and environmental heat stress (expressed as wet bulb globe temperature [WBGT] in Centigrade degrees). At all metabolic rates, the prescriptive zone extends from 10°C to the dotted line at right. Data source: Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. *J Appl Physiol*. 1963;18:53.

core temperature responses during muscular exercise performed at four metabolic intensities in a broad range of environmental conditions (with low humidity).[35] For military and other occupational tasks, metabolic rates of 250, 425, and 600 W represent light-, moderate-, and heavy-intensity exercise, respectively[41] (Table 3-2). (For comparison, highly trained athletes may be able to sustain exercise at metabolic rates as high as 1,000 W.) Note that as metabolic rate increases, the width of the prescriptive zone narrows. This occurs because as environmental temperature increases, skin temperature also increases (see Figure 3-2). As a result, the temperature gradient between core and skin is narrowed, and a greater skin blood flow is required to transfer metabolic heat to the skin. The upper end of the prescriptive zone is reached at the point at which further increases in skin temperature alone do not elicit a large enough increase in skin blood flow to maintain heat transfer, and core temperature must rise to allow sufficient heat transfer to achieve heat balance. At higher exercise intensities the skin blood flow required is greater, and the upper end of the prescriptive zone is reached at lower skin and ambient temperatures. Clothing and equipment that impede heat loss will further narrow the prescriptive zone because the insulation and moisture-transfer barriers provide additional biophysical limits to heat exchange.[39]

For any one individual, the core temperature increase during exercise within the prescriptive zone is proportional to the metabolic rate.[35–37,42] For comparisons among individuals, the association between metabolic rate and core temperature is much more variable. However, expressing exercise intensity as relative intensity (ie, as a percentage of maximal oxygen uptake [$\dot{V}O_2max$]) rather than absolute intensity, removes much of the intersubject variability[43] (Figure 3-6). ($\dot{V}O_2max$ is widely used as a measure of physical fitness level.)

Studies undertaken to clarify the relationship between relative intensity and steady state core temperature response to exercise[44,45] show a curvilinear relationship in Figure 3-7, and steady state core temperature values during exercise, at 65% and 85% $\dot{V}O_2max$, in relation to the ambient dry bulb (with < 50% relative humidity [rh]) temperature.[44] Note that at 65% $\dot{V}O_2max$, core temperature is independent of dry bulb temperature from 5°C to 20°C; at 85% $\dot{V}O_2max$, however, core temperature is strongly influenced by dry bulb temperature, illustrating again (see Figure 3-5) that the prescriptive zone is smaller at higher metabolic rates.

Because core temperature changes are related to the relative exercise intensity, it seems logical to expect that any condition that lowers maximal oxygen uptake (and thus increases relative intensity) would also elicit a greater core temperature response at a given absolute (thus higher relative) exercise intensity. Evidence from several studies in which subjects' $\dot{V}O_2max$ was reduced by hypobaric hypoxia[46,47] or carboxyhemoglobinemia[48] suggests that this is, in fact the case; but contrary results have also been reported.[49] Interpretation of such data is complicated by the fact that some experimental conditions decreased core temperature at rest, so that exercise would need to cause a greater net increase in core temperature to produce the same steady state core temperature level. Conversely, data from studies in which subjects' $\dot{V}O_2max$ was increased by autologous erythrocyte infusion suggest that interventions that increase $\dot{V}O_2max$ (thus lowering relative intensity) may reduce the core temperature increase elicited by exercise.[50–52]

Influence of Hydration

Troops exercising in the heat often incur body water deficits, which typically range from 2% to 8% of their body weight.[53,54] Such deficits develop either because (*a*) adequate amounts of potable fluid are not available or (*b*) thirst is insufficient to make them replace all the fluid lost as sweat.[55] The resulting dehydration increases physiological strain,

TABLE 3-2

TYPICAL METABOLIC RATES ASSOCIATED WITH PERFORMANCE OF SELECTED MILITARY TASKS

Work Intensity Category	Military Activity	Metabolic Rate (W)
Very Light (< 175 W)	Lying on ground	105
	Standing in foxhole	116
	Sitting in truck	116
	Guard duty	137
	Driving truck	163
Light (175–325 W)	Cleaning rifle	198
	Walking on hard surface, 1 m/s, no load	210
	Walking on hard surface, 1 m/s, 20-kg load	255
	Manual of arms	280
	Lift and carry 45-kg artillery shell, 2/min	284
	Walking on hard surface, 1 m/s, 30-kg load	292
Moderate (325–500 W)	Walking on loose sand, 1 m/s, no load	326
	Rifle fire, prone	338
	Walking on hard surface, 1.56 m/s, no load	361
	Lift and carry 45-kg artillery shell, 3/min	370
	Calisthenics	378
	Lift and carry 45-kg artillery shell, 4/min	446
	Walking on hard surface, 1.56 m/s, 20-kg load	448
	Scouting patrol	454
	Crawling, full pack	465
	Foxhole digging	465
	Field assaults	477
Heavy (> 500 W)	Walking on hard surface, 1 m/s, 30-kg load	507
	Walking on hard surface, 2 m/s, no load	525
	Emplacement digging	540
	Bayonet drill	616
	Walking on hard surface, 2 m/s, no load	637
	Walking on loose sand, 1.56 m/s, no load	642

Sources: (1) Sawka MN, Modrow HE, Kolka MA et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders.* Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. Technical Note 95-1. (2) Sawka MN, Roach JM, Young AJ, et al. *Sustaining Soldier Health and Performance During Operation Support Hope: Guidance for Small Unit Leaders.* Fort Detrick, Frederick, Md: US Army Medical Research, Development, Acquisition and Logistic Command; 1994. Technical Note 94-3.

decreases exercise performance, and can have devastating medical consequences.[56–58] Dehydration magnifies the core temperature responses to exercise in both temperate[59,60] and hot[61] environments, and this effect is observed with a fluid deficit as small as 1% of body weight.[62] As the water deficit increases, there is a concomitant graded elevation of core temperature during exercise stress.[61,63] Figure 3-8 illustrates relationships between body water loss and core temperature elevations from studies that examined several water deficit levels.[64] The magnitude of additional core temperature elevation ranges from 0.1°C to 0.23°C for every percentage of body weight lost.[53,61,63,65]

Dehydration not only elevates core temperature responses but also negates the core temperature advantages conferred by high levels of aerobic fitness and heat acclimation.[59,66,67] Figure 3-9 illustrates the effect of dehydration (5% body weight loss) on core temperature responses in the same persons when unacclimated and when acclimated to heat.[67] Heat acclimation lowered core temperature responses of euhydrated subjects; however, similar core temperature responses were observed in hypohydrated subjects regardless of acclimation state. Therefore, the core temperature penalty induced by dehydration was greater in heat-acclimated than in unacclimated persons.

Hyperhydration (greater than normal body water) has been suggested as a means to improve ther-

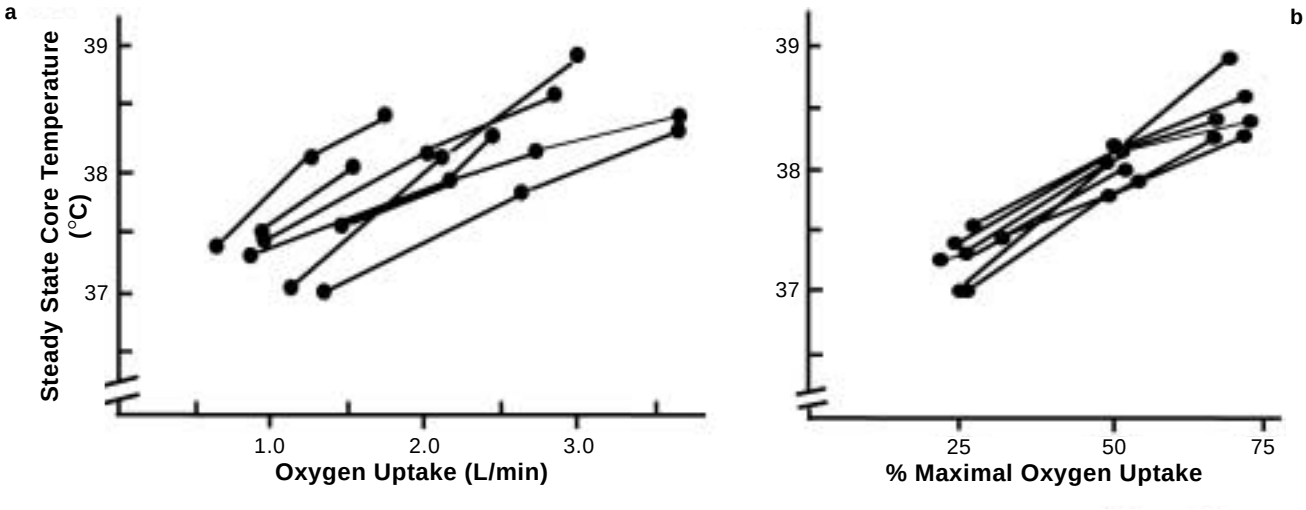

Fig. 3-6. Steady state core temperature responses during exercise as related to (**a**) absolute and (**b**) relative exercise intensities. For both categories, the dry bulb temperature (T_{db}) was approximately 21°C and the relative humidity (rh) approximately 55%. Adapted with permission from Saltin B, Hermansen L. Esophageal, rectal, and muscle temperature during exercise. *J Appl Physiol.* 1966;21:1759.

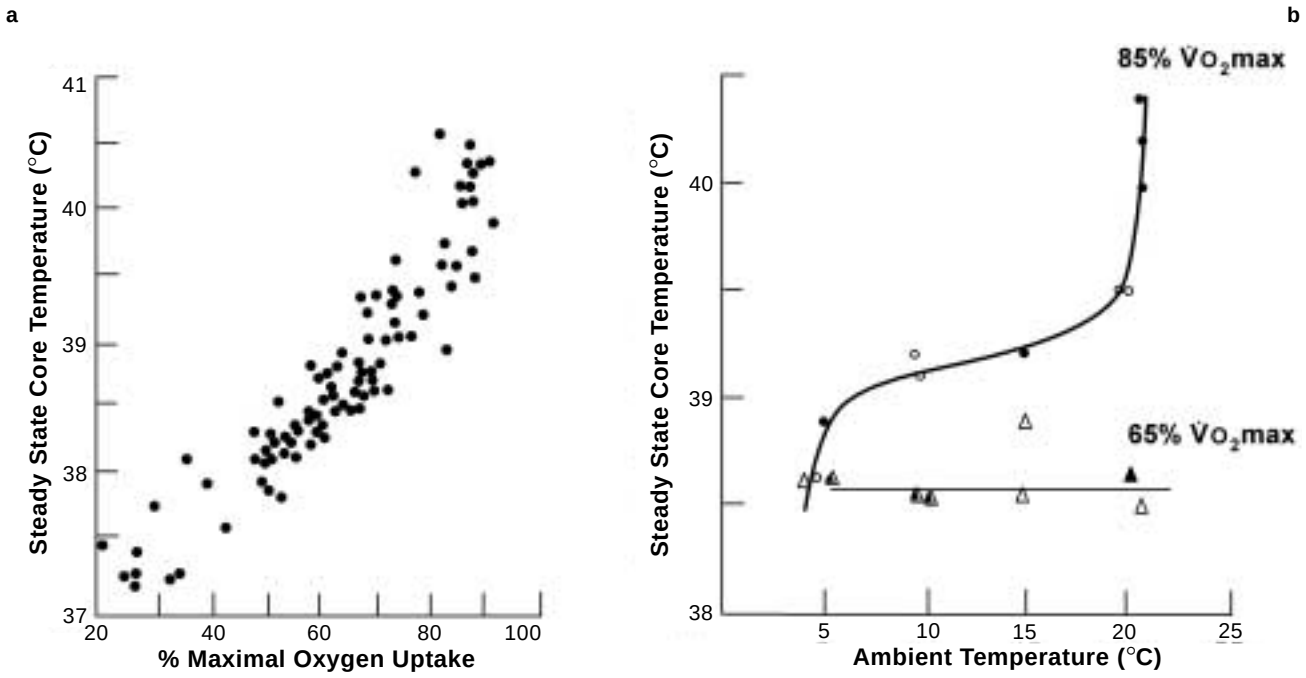

Fig. 3-7. Steady state core temperature response for given (**a**) relative exercise intensity (symbols represent data from four different datasets) and (**b**) ambient temperature (open and closed symbols in each curve represent values for two experimental subjects). $\dot{V}O_2max$: maximal oxygen uptake. (a) Adapted with permission from Davies CTM, Brotherhood JR, Zeidifard E. Temperature regulation during severe exercise with some observations on effects of skin wetting. *J Appl Physiol.* 1976;41:774. (b) Adapted with permission from Davies CTM. Influence of skin temperature on sweating and aerobic performance during severe work. *J Appl Physiol.* 1979;47:772.

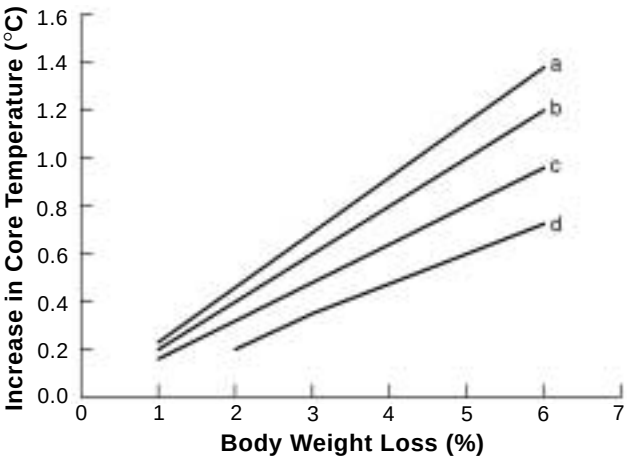

Fig. 3-8. Relationship between body water loss and steady state core temperature elevations during exercise. Data sources for curves: (a) Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol.* 1992;73:1340–1350. (b) Adolph EF, Associates. *Physiology of Man in the Desert*. New York, NY: Intersciences, Inc; 1947. (c) Sawka MN, Young AJ, Francesconi RP, Muza SR, Pandolf KB. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol.* 1985;59:1394–1401. (d) Strydom NB, Holdsworth DL. The effects of different levels of water deficit on physiological responses during heat stress. *Int Z Angew Physiol.* 1968;26:95–102. db: dry bulb temperature, $\dot{V}O_2max$: maximal O_2 uptake. Figure adapted with permission from Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise–heat exposure. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport*. Boca Raton, Fla: CRC Press; 1996: 148.

moregulation and exercise performance in the heat, compared with those that occur during euhydration. However, most studies that examined such effects of hyperhydration suffered from serious design problems (eg, control conditions that represented not euhydration but dehydration).[64] Some investigators[68–72] reported lower core temperatures during exercise after hyperhydration, while others[73–75] did not. One study[76] reported that glycerol-facilitated hyperhydration substantially lowered core temperature (0.7°C) during exercise, compared with control conditions and hyperhydration with water alone. Recent research, however, has failed to confirm that hyperhydration confers any thermoregulatory advantages during exercise–heat stress, whether hyperhydration is induced with water alone or with water plus glycerol.[77,78]

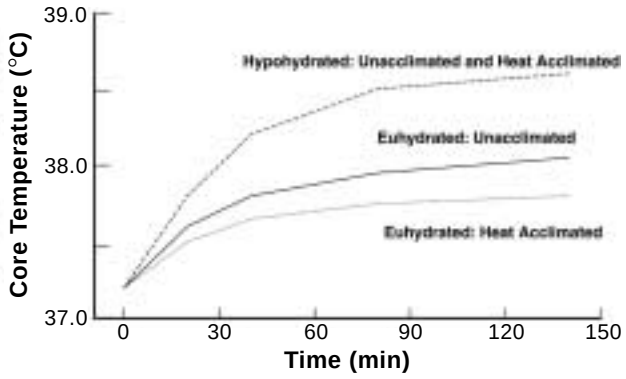

Fig. 3-9. Effect of dehydration on core temperature responses during exercise–heat stress in unacclimated and acclimated persons. Data source: Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: Effects of heat acclimation, gender, and environment. *J Appl Physiol.* 1983;55:1147–1153.

Influence of Exercise Type

Military personnel perform exercise with their legs (eg, marching, climbing), arms (eg, lifting, digging), or both. All studies discussed so far have employed leg (lower body) exercise on treadmills or cycle ergometers. Maximal-effort upper body (arm-crank) exercise elicits an oxygen uptake that is typically 70% of that obtained during maximal-effort lower body exercise.[79] If responses during upper body exercise are compared with those of the same individual during lower body exercise, it is uncertain whether the core temperature response during exercise would be determined by the relative intensity of the exercise (ie, as a fraction of the maximum intensity for the exercising muscle group) or by the absolute metabolic rate, regardless of the exercising muscle mass.[80] If the core temperature responses are determined by the relative intensity (with respect to employed musculature), upper body exercise should be expected to elicit a higher core temperature for a given metabolic rate than should lower body exercise. There has been debate over this question.[80] In fact, several early studies[81,82] reported that upper body exercise elicited lower core temperature responses than lower body exercise at the same absolute metabolic rate, but experimental design and technical problems make their conclusions tenuous.[80] Subsequent studies[83–87] have reported that upper and lower body exercises performed at the same absolute metabolic rate elicit the same core temperature responses. In addition, when upper and lower body exercises are performed at the same relative intensity, upper body exercise elicits lower core temperature responses

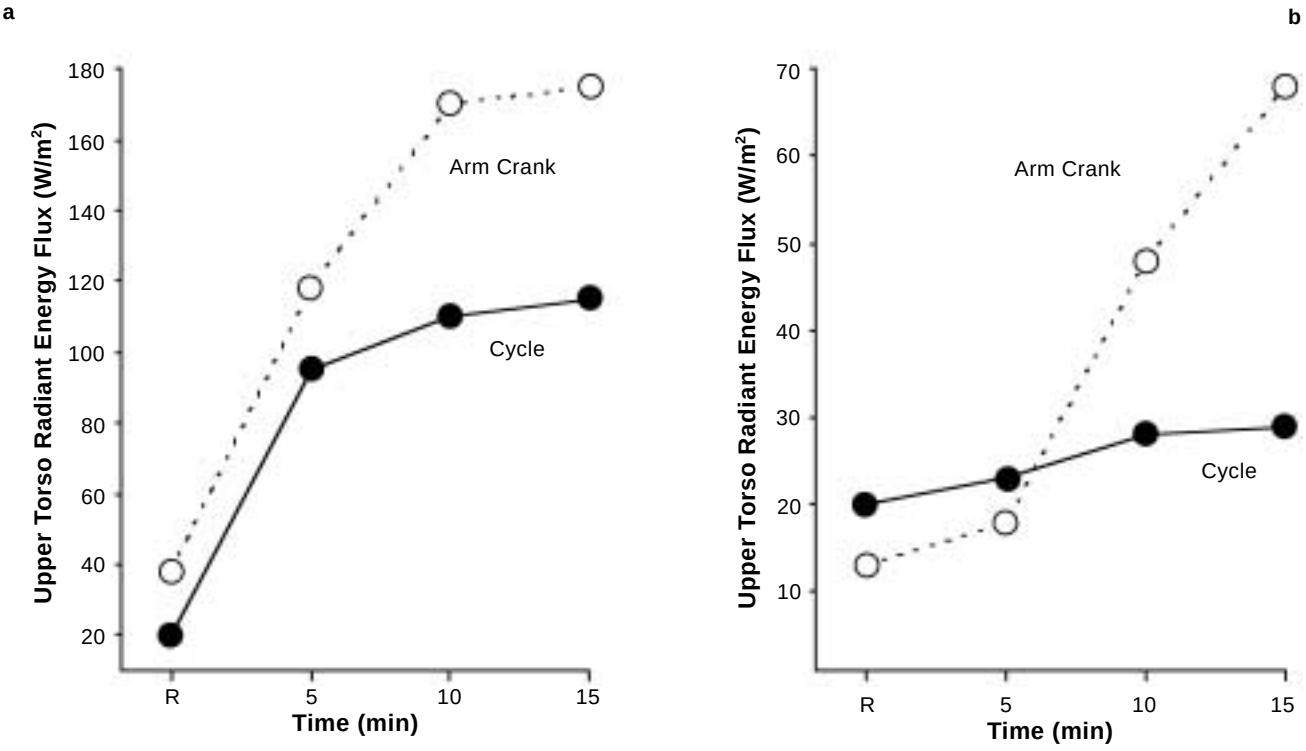

Fig. 3-10. Torso radiative energy flux during upper and lower body exercise at (**a**) 18°C and 78% relative humidity (rh); and (**b**) 35°C and 28% rh. Reprinted with permission from Sawka MN, Gonzalez RR, Drolet LL, Pandolf KB. Heat exchange during upper- and lower-body exercise. *J Appl Physiol.* 1984;57:1051.

(by virtue of being at a lower absolute intensity) than leg exercise.[84] These data indicate that core temperature responses are independent of the skeletal muscle mass employed and dependent on the absolute metabolic intensity during exercise in temperate and hot climates.

The heat exchange mechanisms by which heat balance during exercise is achieved, however, do differ between the muscle groups.[85] Figure 3-10 illustrates the torso net radiative energy flux values (dry heat exchange, R + C) during upper body (arm-crank) and lower body (cycle) exercise in two environments[85]: 18°C ambient temperature with 14°C dew point, which facilitated R + C; and 35°C ambient temperature with 14°C dew point, which facilitated evaporative heat loss (E_{sk}). Torso R + C was greater during upper body exercise regardless of the environment. Torso E_{sk}, arm R + C, and arm E_{sk} were not different between exercise types in each environment. Leg R + C was greater during lower body than upper body exercise in the 18°C environment, whereas leg E_{sk} was greater during lower body than upper body exercise in the 35°C environment. These data indicate that to replace the greater R + C heat loss from the torso during upper body exercise, lower body exercise elicits additional R + C or E_{sk} from the legs.

PHYSIOLOGICAL RESPONSES AND LIMITATIONS

Physiological factors that are critical to work performance and tolerance to heat stress include those related to heat production, heat loss, and the maintenance of muscular contraction. If heat production is not matched by cutaneous heat loss, then body temperatures will continue to rise until heat exhaustion occurs. In addition, the cardiovascular system is challenged to support heat loss and maintain cellular homeostasis via substrate delivery and removal of metabolites.

Metabolism

Knowledge of how heat strain (ie, the physiological change produced by a disturbance) influences metabolic rate and substrate utilization has important implications for calculating nutritional needs as well as heat balance. Acute heat stress increases

resting metabolic rate, but the effect of heat stress on the metabolic rate needed to perform a given submaximal exercise task is not so clear.[88]

Metabolic Rate

Many investigators[89–92] report that a given submaximal exercise task elicits a greater metabolic rate in a hot than in a temperate environment. Some investigators,[93–96] however, report lower metabolic rates in the heat. The subjects' state of heat acclimation does not account for whether a study reported an increased or decreased metabolic rate during exercise in the heat.[88] Most investigators, however, calculated only the aerobic metabolic rate during submaximal exercise, ignoring the contribution of anaerobic metabolism to total metabolic rate. Figure 3-11 shows the total metabolic rate and the percentage of this metabolic rate that was contributed by aerobic and anaerobic metabolic pathways during exercise at three intensities at three ambient temperatures.[91] The anaerobic metabolism was calculated from the postexercise oxygen uptake in excess of resting baseline levels. The aerobic metabolic rate at a given power output decreased with increasing ambient temperature. However, the calculated anaerobic metabolic rate increased more than aerobic metabolic rate decreased, so that the total metabolic rate required to perform exercise at a given power output increases with ambient temperature. Although these calculations of anaerobic metabolic rate may be open to question, the data indicate that studies that ignore possible changes in anaerobic metabolism might underestimate the influence of heat strain on metabolic rate and nutritional needs.

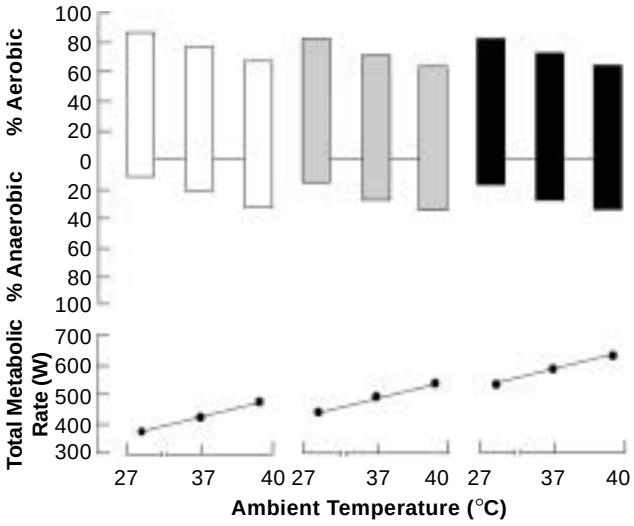

Fig. 3-11. Total metabolic rate and percentage of metabolic rate contributed by aerobic and anaerobic metabolism during exercise in temperate and hot climates. Exercise is performed at three different intensities (light: open; medium: gray; and heavy: solid), which is repeated at the same three temperatures (27°C, 37°C, and 40°C). The partition between anaerobic and aerobic exercise changes as functions of both the intensity of the work and the ambient temperature. Data source: Dimri GP, Malhotra MS, Gupta JS, Kumar TS, Aora BS. Alterations in aerobic–anaerobic proportions of metabolism during work in heat. *Eur J Appl Physiol*. 1980;45:43–50.

Investigations that report a lower metabolic rate during exercise in the heat also report increased plasma or muscle lactate levels[94–96] or an increased respiratory exchange ratio,[93] also suggesting an increased anaerobic metabolism. However, any inference about metabolic events within skeletal muscle based on changes in plasma lactate is inconclusive[97] because plasma lactate concentration reflects the balance between efflux into the blood and removal from the blood. Rowell and associates[98] have shown that during exercise in the heat, splanchnic vasoconstriction reduced hepatic removal of plasma lactate. Therefore, the greater blood lactate accumulation during submaximal exercise in the heat can be attributed in part to a redistribution of blood flow away from the splanchnic tissues.

Skeletal Muscle Metabolism

Several investigators have examined environmental heat stress effects on skeletal muscle metabolism during exercise.[92,96,99–101] When exercising in the heat (vs exercising in the cold), plasma lactate levels and muscle glycogen utilization are increased. When exercising in the heat (vs exercising in temperate conditions), plasma lactate levels are increased and skeletal muscle glycogen utilization is increased[99,100] or not altered.[96,101] Other studies, however, do not support the concept of an increased anaerobic metabolism in the heat. For example, two studies[102,103] that measured arterial and venous lactate concentration across the active musculature during exercise in the heat found that heat stress did not alter lactate levels, muscle blood flow, or glycogen utilization rates. In addition, when exercising in the heat, as opposed to temperate conditions, muscle creatine phosphate levels are lower but muscle creatine levels are higher, with no differences for muscle total adenine nucleotide pool, the ratio of adenosine 5'-triphosphate (ATP) to adenosine 5'-diphosphate (ADP), or inosine monophosphate (IMP).[100]

The question remains as to what physiological mechanism or mechanisms might be responsible for an increased anaerobic metabolism during exercise in the heat. One possibility is the redistribution of blood to the cutaneous veins for heat dissipation; this may result in reduced perfusion of the active skeletal muscles and, thus, local tissue hypoxia.[104] Muscle blood flow has been reported to decrease during resting heat exposure,[105,106] but not during exercise–heat stress.[102,103] A second possibility is that heat stress elevates circulating catecholamines,[107] which increases glycogen utilization in skeletal muscle during exercise.[99,108] A third possibility is that heat stress increases the recruitment of fast-twitch motor units.[109] Fast-twitch skeletal muscles derive a greater percentage of their total energy expenditure from anaerobic pathways, regardless of their level of perfusion, than do slow-twitch fibers.[110,111] A fourth possibility is that high muscle temperatures[99,112] alter glycogen utilization rate through a Q_{10} effect (the ratio of the reaction rates at two temperatures 10 Centigrade degrees apart).[113] Regardless of the mechanism or mechanisms, an increased anaerobic metabolism, increased muscle lactate, and increased glycogen utilization can all contribute to reduced physical exercise performance in the heat.

Influence of Heat Acclimation

If heat stress alters metabolism, then does heat acclimation abate these changes? Heat acclimation probably decreases the metabolic heat load that needs to be dissipated at a given submaximal exercise power output.[88,109] Most reports indicate that oxygen uptake and aerobic metabolic rate during submaximal exercise are reduced by heat acclimation, although a significant effect is not always observed.[88] Large effects (14%–17% reductions) have been reported for stair-stepping,[114–116] but much of the reduction in oxygen uptake required for stair-stepping can be attributed to increased skill and improved efficiency acquired during the acclimation program. By contrast, other studies[109,117–120] report acclimation-induced reductions in the oxygen uptake required for treadmill and cycle-ergometer exercise, which although statistically significant, were much smaller than the large reductions reported for stair-stepping.

Heat acclimation reduces muscle lactate levels[96,100] and glycogen utilization[96,100,121,122] during submaximal exercise. The magnitude of the glycogen-sparing effect during exercise induced by heat acclimation seems to vary widely (15%–50%).[99,121,122] In addition, one investigator[96] observed a statistically significant glycogen-sparing effect during exercise after heat acclimation, but the reduction in glycogen utilization was small (10%) in a hot climate, and more apparent (49%) in a temperate climate.

Cutaneous Heat Loss

The skin provides a large surface area to exchange heat between the body and the environment. Dry heat loss occurs by increasing cutaneous blood volume and flow. Evaporative heat loss occurs by the evaporation of secreted sweat, which cools the blood in superficial vasculature.

Sweating and Evaporative Heat Loss

As the ambient temperature increases, there is greater dependence on evaporative heat loss to defend core temperature during exercise (see Figure 3-4; this concept is also discussed in Chapter 2, Human Adaptation to Hot Environments). Figure 3-12 represents the pattern of sweat secretion, as measured by skin dew point hygrometry, during exercise.[29] Thermoregulatory sweating can begin within a few seconds to minutes after starting muscular exercise,[123] depending on several factors, including skin temperature, previous sweating, and body hydration status. The increase in thermoregulatory sweating closely parallels the increase in body temperature and is accomplished first by recruitment of sweat glands and then by increased sweat secretion per gland.[124–126] Both the density of sweat glands and local sweat rates vary among dif-

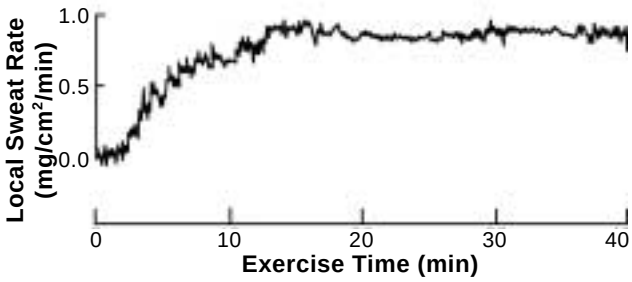

Fig. 3-12. Pattern of sweat gland secretion during exercise–heat stress, at 50% maximal oxygen consumption, 28°C, and 30% relative humidity. Reprinted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise–heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 129.

ferent regions of the skin. Figure 3-13 shows local sweating responses plotted against core temperature. Note that for a given core temperature, the back and chest have the greatest sweating rates. Conversely, the limbs will have relatively high sweating rates only after a substantial elevation (0.5°C is considered substantial) in core temperature.

The rate of sweat evaporation depends on air movement and the water vapor pressure gradient between the skin and the environment, so in still or moist air, sweat tends to collect on the skin. If sweat is secreted but not evaporated rapidly enough to maintain heat balance, core temperature will increase and physical exercise performance will be compromised. In addition, prolonged wetting of the skin surface reduces sweat secretion[127–129] through hidromeiosis (which is probably mediated by the stratum corneum's swelling and occluding the sweat ducts). For these reasons it is important to allow air circulation to the skin, especially on the torso, to maximize evaporative cooling.[130] Likewise, if clothes become soaked, not only are they uncomfortable, but the wetted skin can also inhibit sweat secretion. Clearly, military personnel deployed in hot climates should wear dry clothing next to the skin, and wear clothing systems that allow venting.

Skin Blood Flow and Dry Heat Loss

During heat stress, the cardiovascular system acts to transfer heat from the body core to the skin.[39] Cutaneous vasodilation occurs with heat stress due to reflex and local mechanisms,[25,131,132] and causes warm blood to be redirected from the core to the skin, which increases skin temperature. Skin temperature varies between body regions, but it generally increases with ambient temperature and remains below core temperature. We saw in Figure 3-2 the relationships between regional skin temperatures and core temperature over a range of temperate and hot conditions.[39] In any given environment, skin temperature reflects the balance between blood flow and sweating. Blood flow transfers heat by convection from the deep tissues to the skin. When core and skin temperatures are low enough that sweating does not occur, increasing skin blood flow will elevate skin temperature nearer to blood temperature, and decreasing skin blood flow will lower skin temperature nearer to ambient temperature. Thus, dry heat loss is controlled by varying skin blood flow and, thereby, skin temperature. In conditions in which sweating occurs, the tendency of skin blood flow to warm the skin is approximately balanced by the tendency of sweating to cool the skin. Therefore, after sweating has begun, a high skin blood flow acts primarily to deliver heat for evaporation of sweat.

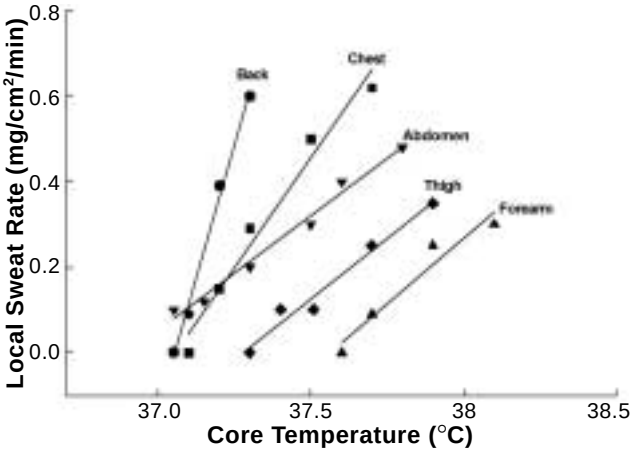

Fig. 3-13. Regional sweating responses plotted against core temperature. Adapted with permission from Nadel ER, Mitchell JW, Saltin B, Stolwijk JAJ. Peripheral modifications to the central drive for sweating. *J Appl Physiol.* 1971;31:830.

As discussed earlier, skin temperature is higher in warmer environments, while core temperature is relatively unaffected over a wide range of ambient temperatures. Thus as ambient temperature increases, the core-to-skin thermal gradient becomes narrower, and skin blood flow increases in response to the high skin temperature, so as to achieve core-to-skin heat transfer sufficient for thermal balance. When performing military tasks, metabolic heat production may often be 3- to 8-fold higher than resting levels (see Table 3-2), and delivery of heat to the skin must increase proportionately to reestablish thermal balance. Core temperature does increase during exercise, thus widening the core-to-skin temperature gradient. However, the widening of the core-to-skin temperature gradient is proportionately less than the increase in heat production, so that only by a skin blood flow increase does core-to-skin heat transfer increase enough to match heat production and allow thermal balance to be reestablished.[25,104] Conversely, if increased evaporative cooling lowers skin temperature, the core-to-skin thermal gradient becomes wider, and skin blood flow requirements to achieve the same heat transfer are proportionately decreased.[131]

Circulation

During maximal vasodilation elicited by heating,

skin blood flow can approach 8 L / min.[132] Although the skin is not maximally vasodilated during exercise in the heat,[133] skin blood flow can still reach levels of several liters per minute.[134] During exercise–heat stress, maintaining a high skin blood flow can impose a substantial burden on the cardiovascular system.[39] High skin blood flow promotes pooling of blood in the skin and superficial veins, thus reducing central venous pressure, cardiac filling, and stroke volume, and requiring a higher heart rate to maintain cardiac output. In addition, if fluid lost through sweating is not replaced, the resulting reduction in blood volume aggravates the hemodynamic effects of peripheral pooling.[123] To help maintain stroke volume despite decreased cardiac filling, cardiac contractility increases as a result of elevated sympathetic activity and direct effects of temperature on the myocardium.[25,132]

During exercise in the heat, the primary cardiovascular challenge is to have sufficient cardiac output to simultaneously support high skin blood flow for heat dissipation and high muscle blood flow for metabolism. Figure 3-14 provides an analysis of cardiac output responses and the redistribution of cardiac output during rest and exercise in temperate and hot environments.[104] This figure depicts cardiac output as unchanged at rest and elevated during exercise in the heat. In this figure, the higher skin blood flow required for thermoregulation in the heat is provided at rest almost entirely by diverting blood flow from other vascular beds (chiefly renal and splanchnic circulations); but during exercise, much of the additional skin blood flow in the heat is provided by increasing cardiac output. Thus maximum cardiac output is reached at a lower exercise intensity in the heat than in a temperate environment. This pattern is consistent in its outlines with other studies that investigated the relation of cardiac output responses during exercise to the intensity and duration of heat stress.[135–137] During mild heat stress, cardiac output is unchanged[135,136] or increased,[137] but during severe heat stress, cardiac output may even be decreased[135] below levels observed in temperate conditions.

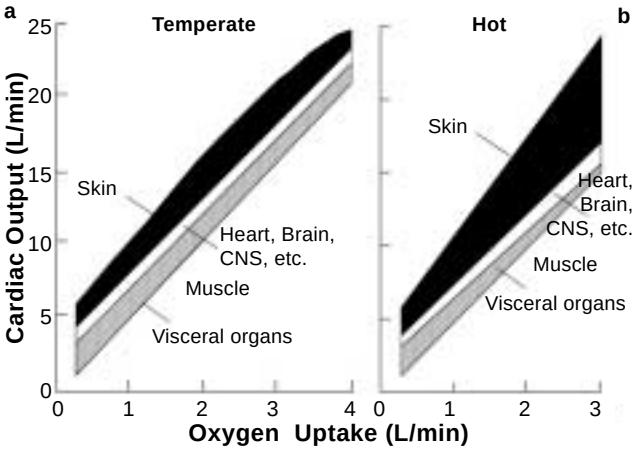

Fig. 3-14. Hypothetical cardiac output response and redistribution during rest (the low end of the curve) and exercise in (**a**) temperate (≤ 25°C) and (**b**) hot (> 30°C) environments. Adapted with permission from Rowell LB. *Human Circulation: Regulation During Physical Stress*. New York, NY: Oxford University Press; 1986: 375.

Brain, spinal cord, and coronary blood flow are believed to be unaffected by exercise–heat stress.[132,138] However, visceral (splanchnic, renal) blood flow is reduced by both exercise and heat stress due to increased sympathetic activity mediated through thermal receptor stimulation.[132,138] The visceral blood flow reductions are graded to the exercise intensity, and the effects of exercise and heat seem to be additive.[25,138] Reduced visceral blood flow allows a corresponding diversion of cardiac output to skin and exercising muscle. Also, secondarily to reduced visceral blood flow, a substantial volume of blood can be mobilized from the compliant splanchnic beds to help maintain cardiac filling during exercise–heat stress.[25] If these compensatory responses are insufficient, skin and muscle blood flow may be compromised. Although Figure 3-14 indicates that muscle blood flow decreases during severe exercise–heat stress,[138] this conclusion is controversial.[25,132]

Body Fluid Losses

If fluid losses during exercise–heat stress are not replaced, then negative fluid balance occurs, decreasing subjects' total body water and making them hypohydrated. Water is the largest component of the human body, comprising 45% to 70% of body weight.[123] The average 75-kg man contains about 45 L of water, or about 60% of body weight. Because adipose tissue is about 10% water and muscle tissue is about 75% water, total body water is inversely related to body fat content.[123] In addition, muscle water and glycogen content parallel each other, probably because of the osmotic pressure exerted by glycogen molecules within the sarcoplasm.[139] As a result, physically trained soldiers have relatively greater total body water than their sedentary counterparts, by virtue of a smaller percentage of body fat and a higher skeletal muscle glycogen concentration.[140]

Figure 3-15 presents resting plasma volume and osmolality values for heat-acclimated persons when hypohydrated at various levels.[64] Sweat-induced

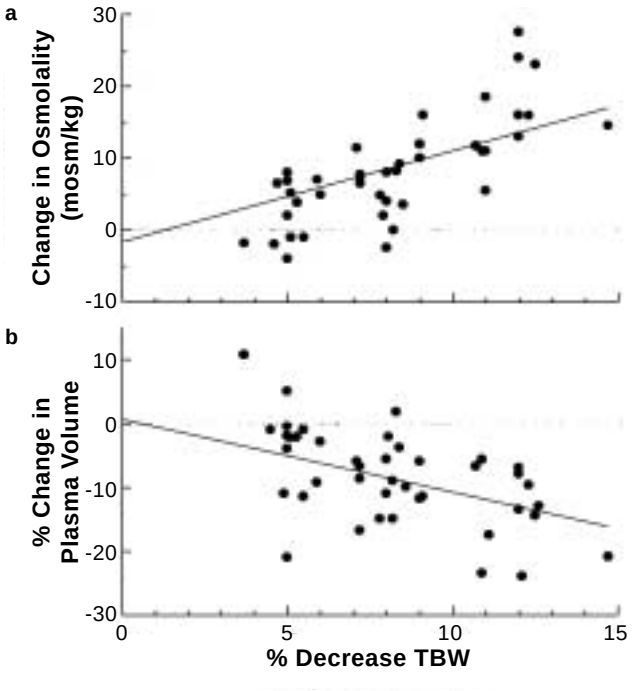

Fig. 3-15. The effect of loss of total body water (TBW) on changes in (**a**) plasma osmolality and (**b**) plasma volume (PV) in heat-acclimated persons. Reprinted with permission from Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise–heat exposure. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport*. Boca Raton, Fla: CRC Press; 1996: 142.

dehydration will decrease plasma volume and increase plasma osmotic pressure in proportion to the level of fluid loss. Plasma volume decreases because it provides the precursor fluid for sweat, and osmolality increases because sweat is ordinarily hypotonic relative to plasma. Increased sodium and chloride concentrations are primarily responsible for the elevated plasma osmolality.[141–143] It is the plasma hyperosmolality that mobilizes fluid from the intracellular to the extracellular space to enable plasma volume defense in hypohydrated subjects. An important consequence of this mechanism is that heat-acclimated persons, who secrete more dilute sweat than unacclimated persons, have a smaller plasma volume reduction for a given body water deficit.[144] The smaller loss of plasma volume occurs because the more dilute sweat of heat-acclimated persons leaves more solute remaining within the extracellular space to increase extracellular osmotic pressure and redistribute fluid from the intracellular space.

Some persons use diuretics for medical purposes or to reduce their body weight to "make weight" for an athletic competition. Commonly used diuretics include thiazides (eg, Diuril), carbonic anhydrase inhibitors (eg, Diamox), and furosemide (eg, Lasix). Diuretics increase the formation of urine and most of them cause the loss of sodium as well as water, so that diuretic-induced dehydration generally results in an isosmotic hypovolemia, with a

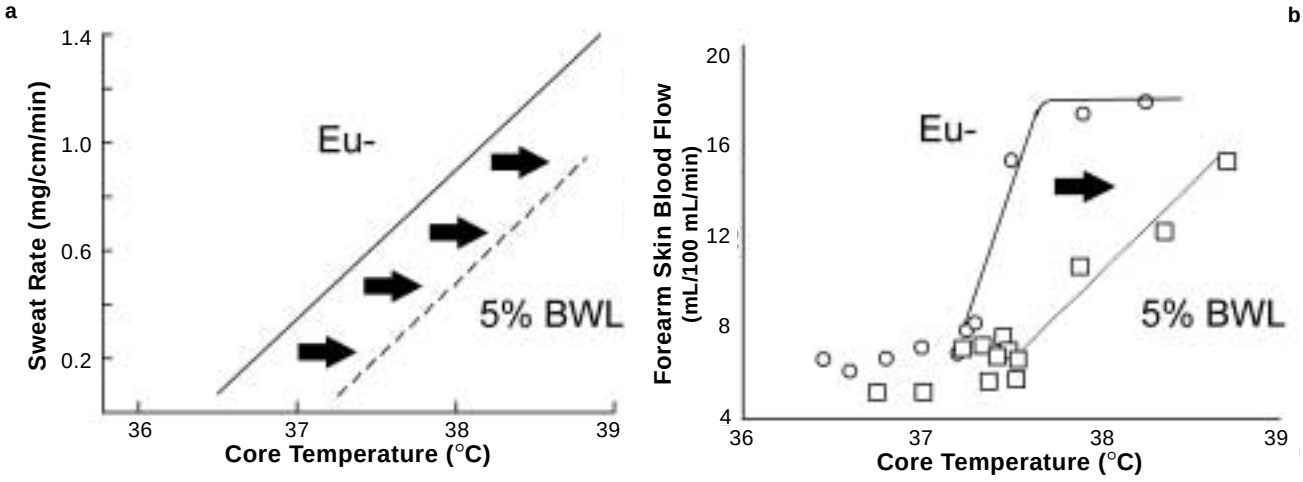

Fig. 3-16. (**a**) Local sweat rate and (**b**) forearm skin blood flow (FBF) response data for euhydrated (Eu) and dehydrated (5% body water loss [BWL]) individuals during exercise–heat stress. Open circles: euhydrated; open squares: 5% BWL. Data sources: (a) Sawka MN, Gonzalez RR, Young AJ, Dennis RC, Valeri CR, Pandolf KB. Control of thermoregulatory sweating during exercise in the heat. *Am J Physiol.* 1989;257:R311–R316. (b) Kenney WL, Tankersley CG, Newswanger DL, Hyde DE, Puhl SM, Turnera NL. Age and hypohydration independently influence the peripheral vascular response to heat stress. *J Appl Physiol.* 1990;8:1902–1908. Adapted with permission from Sawka MN. Physiological consequences of hydration: Exercise performance and thermoregulation. *Med Sci Sports Exerc.* 1992;24:664.

much greater ratio of plasma loss to total body water loss than does either exercise- or heat-induced dehydration. Correspondingly less intracellular fluid is lost after diuretic administration because there is not an increase in extracellular osmolality to promote redistribution of water from the intracellular to the extracellular space. Diuretic-induced dehydration produces the same adverse effects, including core temperature elevations, as those mediated by underdrinking.[144] For the same total loss of body water one might theoretically expect diuretic-induced dehydration to cause greater cardiovascular strain than thermal dehydration because of the greater loss of extracellular fluid volume in diuretic-induced dehydration. However, such a difference in cardiovascular strain has not been established.

As discussed earlier, dehydration causes an increased core temperature during rest and exercise–heat stress, by impairing both dry and evaporative heat loss (or, if the air is warmer than the skin, by aggravating dry heat gain).[144–147] Figure 3-16 presents the local sweating responses[148] and skin blood flow responses[149] to dehydration (5% body weight loss) during exercise in the heat. These curves indicate that dehydration reduced both heat-loss effector responses (ie, sweat rate and skin blood flow) for a given core temperature level.[144] Dehydration is usually associated with either reduced or unchanged whole-body sweating rates at a given metabolic rate in the heat.[150] However, even when dehydration is associated with no change in sweating rate, core temperature is usually elevated, so that sweating rate for a given core temperature is lower when hypohydrated.[150] The physiological mechanisms mediating the reduced dry and evaporative heat loss from dehydration include both the separate and combined effects of plasma hyperosmolality[144,151,152] and reduced blood volume.[153–155]

PHYSICAL EXERCISE PERFORMANCE

Troops deployed to hot climates are usually there to perform a mission that requires physical exercise. The amount of physical exercise they can perform is based on the climatic conditions, biomedical state (ie, acclimation state, physical fitness, hy-dration level, and health), and mission requirements (eg, required metabolic rate, duration, and clothing or equipment worn). Together, these factors determine the physiological strain (elevated core temperature and cardiovascular strain), which, in turn, determines exercise performance decrements. Heat stress will induce decrements in physical exercise performance; however, the exact magnitude of the decrement is difficult to predict. The effects of heat stress on exercise performance can be divided into (*a*) the effects on the maximum exercise intensity that an individual can achieve and (*b*) the effects on performance of submaximal exercise.

Maximal Intensity Exercise

Maximal exercise intensity is achieved by performing exercise of increasing intensity until physiological criteria (ie, the determination of maximal aerobic power, or maximal oxygen uptake [$\dot{V}O_2max$ or volitional exhaustion (ie, physical exercise capacity) are reached. An activity that requires a low fraction of $\dot{V}O_2max$ is sustained more easily than one that requires a high fraction of $\dot{V}O_2max$. In addition, the sorts of training that increase $\dot{V}O_2max$ also increase the ease of performing an activity at any given fraction of $\dot{V}O_2max$. For both reasons, high $\dot{V}O_2max$ is important to successfully perform tasks that require sustained high metabolic rates, so that a lower $\dot{V}O_2max$ often translates into reduced physical exercise performance. $\dot{V}O_2max$ is defined as the maximal rate at which oxygen can be utilized by body tissues during physical exercise. Most investigators find that $\dot{V}O_2max$ is lower in hot than in temperate climates.[156–159] For example, $\dot{V}O_2max$ was 0.25 L/min (7%) lower at 49°C than at 21°C in one study,[160] and the state of heat acclimation in this study did not alter the size of the $\dot{V}O_2max$ decrement (Figure 3-17). However, some investigators report no effect of ambient temperature on $\dot{V}O_2max$.[95,161]

What physiological mechanisms might be responsible for such a reduction in $\dot{V}O_2max$? Thermal stress, by dilating cutaneous arterioles, might divert some of the cardiac output from skeletal muscle to skin, thus leaving less blood flow to support the metabolism of exercising skeletal muscle. In addition, dilation of the cutaneous vascular bed may increase cutaneous blood volume at the expense of central blood volume, thus reducing venous return and cardiac output. In one study,[135] for example, heat exposure reduced cardiac output during intense (~ 73% $\dot{V}O_2max$) exercise by 1.2 L/min below control levels. Such a reduction in cardiac output during heat exposure could account for a decrement of 0.25 L/min in $\dot{V}O_2max$, assuming each liter of blood delivers approximately 0.2 L of oxygen (1.34 mL of oxygen per gram of hemoglobin • 15 g hemoglobin per 100 mL of blood).

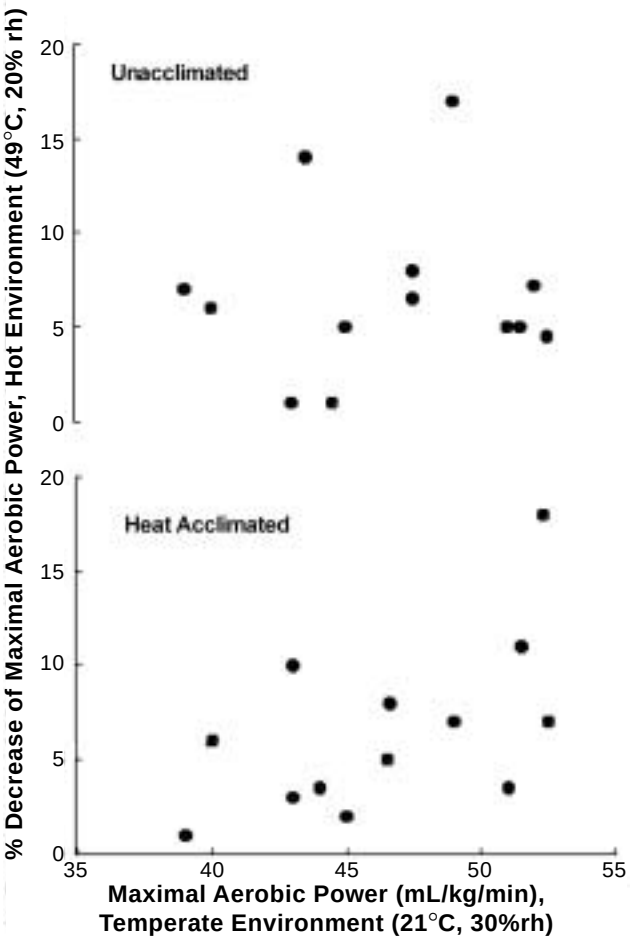

Fig. 3-17. Effects of heat exposure and heat acclimation on maximal aerobic power. The data show that neither initial fitness nor the heat acclimation state have an effect. Adapted with permission from Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol.* 1985;53:296.

Submaximal Intensity Exercise

Numerous studies have examined the influence of dehydration on $\dot{V}O_2$max and physical exercise capacity (exercise to volitional exhaustion; ie, exhaustion as determined by the subject rather than by physiological criteria).[64] In temperate environments, a body water deficit of less than 3% body weight loss does not alter $\dot{V}O_2$max,[64] but $\dot{V}O_2$max is reported to be decreased[66,162,163] by dehydration that equals or exceeds 3% body weight loss (Figure 3-18). Therefore, there may be a critical water deficit (3% body weight loss) that exists before dehydration reduces $\dot{V}O_2$max in temperate environments. In the heat, small to moderate water deficits (ie, 2%–4% body weight loss) can result in a large reduction of $\dot{V}O_2$max.[164] Thus, it seems that climatic heat stress has a potentiating effect on the reduction of $\dot{V}O_2$max caused by dehydration.

Physiological mechanisms for reduced submaximal intensity exercise performance in the heat include increased thermal and cardiovascular strain, more rapid glycogen depletion, increased metabolite accumulation, and diminished motivation for exercise.[25,165,166] Although the exact mechanism or mechanisms are unknown, they probably depend on the specific heat stress, exercise task, and biomedical state of the individual.

Figure 3-19 demonstrates the effects of air temperature and dehydration on the submaximal exercise output of soldiers.[53] This analysis is based on heat-acclimated soldiers marching at a metabolic rate of approximately 650 W (which represents high-intensity exercise for military or occupational tasks, or both), and an air temperature of 43°C with low humidity.[41] Environmental heat stress reduced submaximal exercise output at all hydration levels. Adolph and associates[53] suggested that for every five Centigrade degree increase in skin temperature there was a 10% decrement in endurance time during submaximal exercise. In addition, the decrements in submaximal exercise output from heat stress and from dehydration were additive.[53] For example, exposure to 43°C reduced submaximal exercise output by approximately 20% (compared with temperate conditions), and a 2.5% (body weight loss) dehydration reduced submaximal exercise output (compared with euhydration) by the same amount. If heat stress and dehydration were experienced together, soldiers would experience a 50% decrease in submaximal exercise output.

Studies of the effects of dehydration on soldiers' ability to tolerate heat strain during submaximal intensity exercise demonstrate that soldiers who drink water can continue to exercise in the heat for many hours, whereas those who do not drink adequate amounts of water must discontinue because of exhaustion. In the summers of 1942 and 1943, Adolph and associates[53] conducted experiments in the California deserts, in which heat-acclimated soldiers attempted endurance marches (4–6.5 km/h) in an ambient temperature of approximately 38°C, and either drank water ad libitum or refrained from drinking. They reported that during 3- to 8-hour desert walks, 1 of 59 (2%) and 11 of 70 soldiers (16%) suffered exhaustion from heat strain when they did

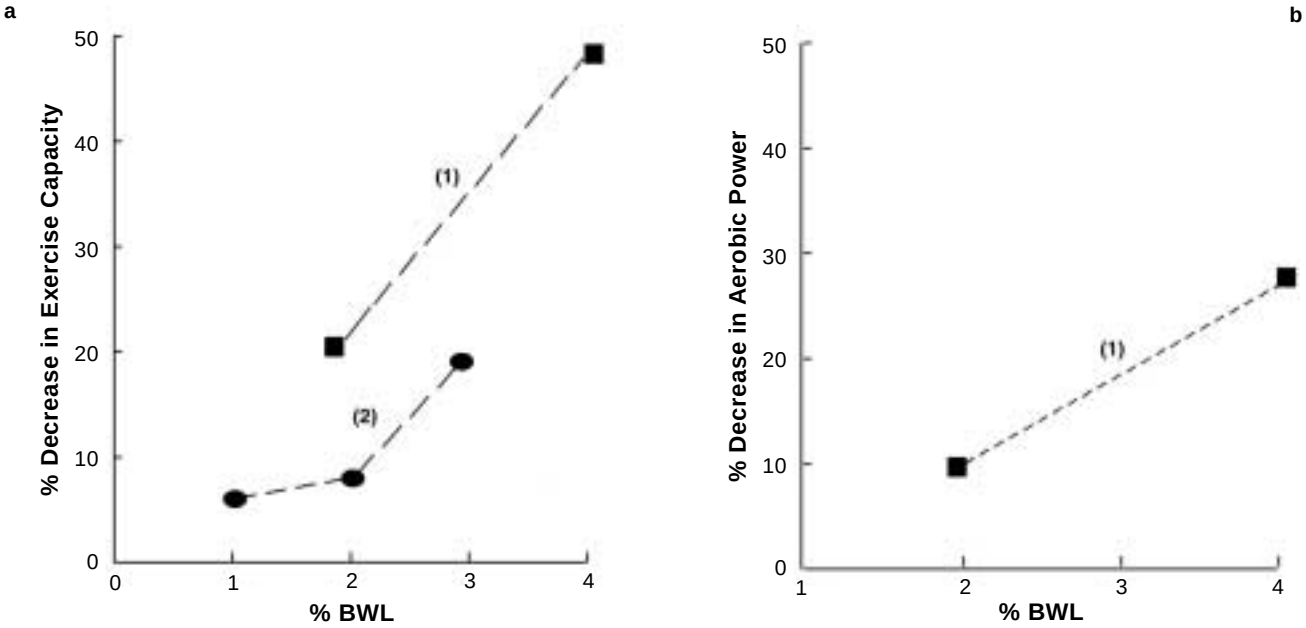

Fig. 3-18. Relationships between dehydration level and decrements in (**a**) physical exercise capacity and (**b**) maximal aerobic power during heat exposure. BWL: body water loss. Data sources: (1) Craig FN, Cummings EG. Dehydration and muscular work. *J Appl Physiol*. 1966;21:670–674. (2) Pinchan G, Gauttam RK, Tomar OS, Bajaj AC. Effects of primary hypohydration on physical work capacity. *Int J Biometeorol.* 1988;32:176–180.

or did not drink water, respectively. In other experiments, they reported that 1 of 59 soldiers (2%) and 15 of 70 soldiers (21%) suffered exhaustion from heat strain during an attempted 8-hour desert march when they did or did not drink water, respectively.

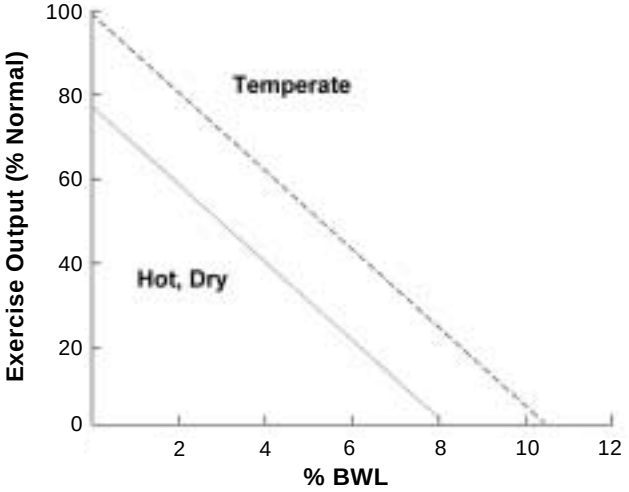

Fig. 3-19. Effects of air temperature and dehydration level on submaximal exercise output of soldiers. BWL: body water loss. Adapted from Adolph EF, Associates. *Physiology of Man in the Desert*. New York, NY: Intersciences, Inc; 1947: 207.

Ladell[167] had subjects attempt a 140-minute walk in a hot (T_a = 38°C) climate while ingesting different combinations of salt and water. He reported that exhaustion from heat strain occurred in 9 of 12 (75%) experiments when subjects received neither water nor salt, and 3 of 41 (7%) experiments when they received only water. Sawka and colleagues[61] had eight heat-acclimated soldiers attempt treadmill walks (~ 25% $\dot{V}O_2$max for 140 min) in a hot–dry (T_a = 49°C, rh = 20%) climate when euhydrated and when hypohydrated by 3%, 5%, and 7% of their body weight. All eight soldiers completed the euhydration and 3% dehydration experiments, and seven subjects completed the 5% dehydration experiments. For the 7% dehydration experiments, six soldiers discontinued after completing an average of 64 minutes. Clearly, dehydration reduces submaximal exercise performance and increases the occurrence of exhaustion from heat strain.

Either heat stress alone or dehydration alone can impair athletic endurance exercise performance. One study[166] found that cycle exercise endurance time was reduced by 38% in hot versus temperate conditions. Another study[168] examined the effects of dehydration on the performance of athletes competing in 1,500-m, 5,000-m, and 10,000-m races when euhydrated and when hypohydrated by only 2% of body weight. Dehydration impaired running

performance at all race distances, but to a greater extent in the longer races (~ 5% for the 5,000 m and 10,000 m) than in the shorter race (3% for 1,500 m). Dehydration also adversely affects rowing performance.[169]

Studies[170,171] have examined the adverse effects of dehydration on moderate to intense cycle ergometer performance. In both, high-intensity performance tests were conducted immediately after approximately 60 minutes of cycling, during which volunteers either drank nothing or drank sufficient fluid to replace sweat losses. In one of the studies,[170] time to fatigue when cycling at 90% $\dot{V}O_2$max was 51% longer (9.8 min vs 6.5 min) when subjects drank sufficient fluids to prevent dehydration. The other study[171] found that cyclists completed a performance ride 6.5% faster if they drank fluids during exercise. The results of these studies clearly demonstrate the detrimental effects of dehydration on submaximal exercise performance.

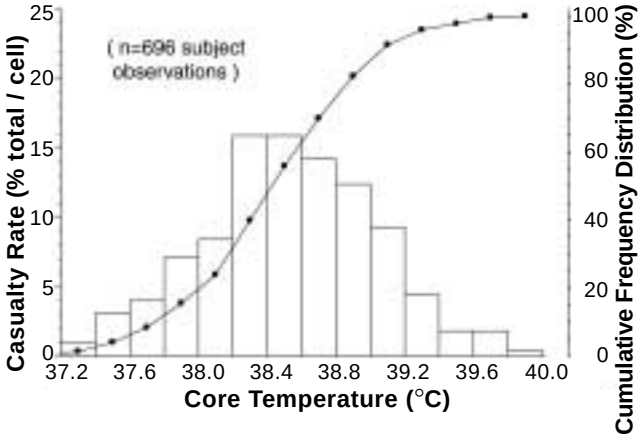

Fig. 3-20. Relationship between core temperature and cumulative occurrence of exhaustion from heat strain, and the fraction of cases of exhaustion from heat strain occurring in each 0.2 Centigrade degree interval of core temperature. Data source: Kraning KK. Analysis of data from 696 subject observations during experiments conducted at US Army Research Institute of Environmental Medicine, Natick, Mass; 1997. Unpublished.

Heat Tolerance

Uncompensable heat stress is a condition in which the required evaporative cooling (E_{req}) is greater than the maximal evaporative cooling (E_{max}) permitted by the environment. Therefore, during uncompensable heat stress, steady state core temperature cannot be achieved and body temperature continues to rise until exhaustion occurs. Ambient temperature, dew point, wind velocity, metabolic rate, and clothing are important factors that influence whether exercise–heat stress will be compensable or uncompensable. Uncompensable heat stress is associated with exhaustion from heat strain occurring at relatively low core temperatures.[5,6,172] Because evaporative cooling is impaired during uncompensable heat stress, skin temperature is high, causing the blood vessels in the skin to dilate. It is believed that the resulting displacement of blood to skin causes cardiovascular strain and instability, which accounts for the occurrence of exhaustion at relatively low core temperatures during uncompensable heat stress. Much higher core temperatures can be tolerated during compensable heat stress, and exhaustion is then usually associated with dehydration or substrate depletion.

Little is known about relationships between physiological indices and exhaustion from heat strain.[5,6,173,174] Core temperature provides the most reliable physiological index to predict the incidence of exhaustion from heat strain.[5,6] For heat-acclimated subjects exercising in uncompensable heat stress, Figure 3-20 presents the relationship between core temperature and the cumulative occurrence of exhaustion from heat strain, and also the fraction of cases of exhaustion from heat strain occurring in each interval of 0.2 Centigrade degrees of core temperature. Figure 3-21 presents the relationships between core temperature and the cumulative occurrence of exhaustion from heat strain for heat-acclimated persons exercising in uncompensable[5] and compensable[175] heat stress. During uncompensable heat stress, exhaustion was rarely associated with a core temperature below 38°C, but exhaustion always occurred before a temperature of 40°C was achieved.[5,6]

Several recent studies[5,6,77,174] examined the effects of different biomedical factors (eg, aerobic fitness, hydration, heat acclimation state) on physiological tolerance to uncompensable heat stress (Table 3-3). Sawka and colleagues[5] studied the effects of dehydration and aerobic fitness on physiological tolerance to uncompensable heat stress. Heat-acclimated soldiers walked to physical exhaustion when either euhydrated or hypohydrated (8% of total body water). A combination of environmental conditions (T_a = 49°C, rh = 20%) and exercise intensity (47% $\dot{V}O_2$max) was used that would not allow thermal equilibrium to be reached, so that exhaustion from heat strain would eventually occur. Dehydration reduced tolerance time (from 121 min to 55 min), but more important, dehydration reduced the core temperature that a person could tolerate. Exhaustion occurred at a core temperature approximately 0.4°C lower when hypohydrated than when euhydrated. They found no influence of aerobic fitness on physi-

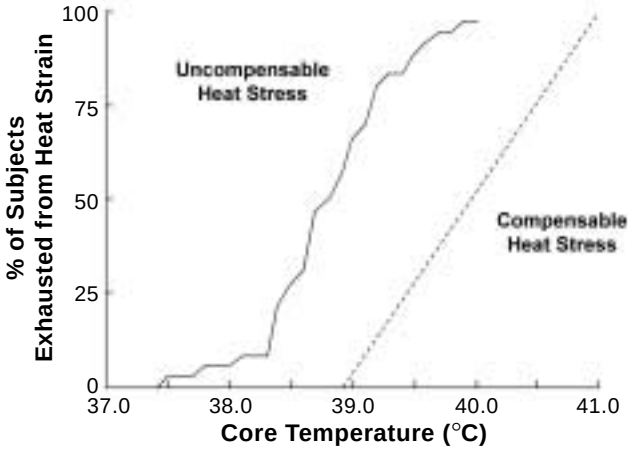

Fig. 3-21. Relationships between core temperature and occurrence of exhaustion from heat strain during uncompensable and compensable heat stress. The different shapes of the curves reflect the different experimental conditions. Data sources for uncompensable heat stress: (1) Sawka MN, Young AJ, Latzka WA, Neufer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. *J Appl Physiol*. 1992;73:368–375. (2) Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol*. 1994;77:216–222. (3) Latzka WA, Sawka MN, Matott RP, Staab JE, Montain SJ, Pandolf KB. *Hyperhydration: Physiologic and Thermoregulatory Effects During Compensable and Uncompensable Exercise–Heat Stress.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1996. Technical Report TR96-6. Data source for compensable heat stress: (1) Pandolf KB, Stroschein LA, Drolet LL, Gonzalez RR, Sawka MN. Prediction modeling of physiological responses and human performance in the heat. *Comput Biol Med*. 1986;16:319–329.

ological tolerance to uncompensable heat stress. These findings suggest that dehydration not only impairs exercise performance but also reduces tolerance to heat strain.

Montain and colleagues[6] studied the influence of exercise intensity, protective clothing (including NBC protective gear, armored vests, etc), and environment on physiological tolerance to uncompensable heat stress. They had heat-acclimated soldiers walk to physical exhaustion at two metabolic rates (425 W and 600 W) when wearing full or partial protective clothing ensembles in a hot–humid and a desert environment. They observed that wearing the full protective clothing ensemble reduces physiological tolerance, as core temperature at exhaustion was lower than in the partial ensemble. They also reported that the exercise intensity and the environment in the laboratory did not alter the core temperature at exhaustion during uncompensable heat stress. Wearing protective clothing was believed to reduce physiological tolerance to heat strain because of higher skin temperature causing greater displacement of blood from central circulation to the skin.

Nielsen and colleagues[174] studied the effects of heat acclimation in an uncompensable heat stress condition. They had highly trained subjects perform exercise (60% $\dot{V}O_2max$) to exhaustion for 9 to 12 days at 40°C. They reported that final core temperature was consistently 39.7°C at exhaustion and was not changed by heat acclimation; however, endurance time was increased (because the rate of core temperature increase was slowed). They also observed increased sweating rate, increased cardiac output, and reduced heart rate at exhaustion after heat acclimation. The state of heat acclimation did not affect these trained subjects' tolerance to high core temperature during uncompensable heat stress.

High core temperatures are better tolerated during compensable heat stress, presumably because the lower skin temperatures result in a lesser degree of cardiovascular strain than at the same core temperature during uncompensable heat stress. Joy and Goldman,[173] for example, reported that 35 of 63 (56%) elite soldiers were still performing military tasks when core temperature reached 39.5°C, their predetermined endpoint criterion. Some individuals can tolerate core temperatures greater than 40°C and continue to exercise during compensable heat stress.[176–180] For example, Pugh and colleagues[177] measured the core temperatures of 47 runners immediately after they completed a marathon race. Seven, including three of the first five finishers, had core temperatures greater than 40°C (the highest value was 41°C). None of the runners who did not finish the race achieved core temperatures as high as 40°C. In another study,[180] eight trained and acclimatized subjects marched 31.5 km in 8 hours on a summer day, carrying 35-kg loads. On completion, five had rectal temperatures between 41.5°C and 42.4°C. Such high core temperatures should not be considered to be safe, however. In a series of young men (aged 17–24 years) with exertional heatstroke, presenting rectal temperatures were recorded on 30 patients; and of these, 14 had presenting rectal temperatures lower than or equal to 41°C.[181] Tolerance to high core temperature is not well understood, and it is not possible to predict who can tolerate a high core temperature, and in what circumstances, without ill effects.

TABLE 3-3

SUMMARY OF STUDIES EXAMINING THE INFLUENCE OF BIOMEDICAL FACTORS ON PHYSIOLOGICAL TOLERANCE TO UNCOMPENSABLE EXERCISE–HEAT STRESS

Factor	Effect		Study
	Yes	No	
Aerobic Fitness		X	Sawka MN, Young AJ, Latzka WA, Neufer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. *J Appl Physiol*. 1992;73:368–375.
Climate		X	Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol*. 1994;77:216–222.
Clothing Encapsulation	↓ 0.2°C		Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol*. 1994;77:216–222.
Exercise Intensity		X	Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol*. 1994;77:216–222.
Gender	—	—	—
Intermittent Exercise	—	—	—
Heat Acclimation		X	(1) Nielsen B, Hales JRS, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol.* 1993;460:467–485. (2) Nielsen B, Strange S, Christensen NJ, Warberg J, Saltin B. Acute and adaptive responses in humans to exercise in a warm humid environment. *PflÚgers Arch.* 1997;434:49–56.
Hyperhydration		X	Latzka WA, Sawka MN, Montain SJ, et al. Hyperhydration: Tolerance and cardiovascular effects during uncompensable exercise–heat stress. *J Appl Physiol.* 1998;84:1858–1864.
Hypohydration	↓ 0.5°C		Sawka MN, Young AJ, Latzka WA, Neufer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. *J Appl Physiol*. 1992;73:368–375.

Encapsulation: wearing closed mission-oriented protective posture (MOPP) 4 gear
No: Effect not found; Yes: Effect found

STRATEGIES TO SUSTAIN PHYSICAL EXERCISE CAPABILITIES

Strategies to manage heat stress and sustain physical exercise capabilities can include improving and maintaining the soldiers' heat dissipation capabilities and managing the amount of heat stress exposure. Heat dissipation capabilities can be improved by heat acclimation and physical training, whereas rehydration allows maintenance of these capabilities. Heat stress can be managed by reducing climatic exposure (seeking shade and wind), wearing less clothing or equipment, or both, and reducing the physical exercise output. Microclimate cooling can artificially improve heat loss capabilities. Most of these heat stress management approaches either reduce the amount of work accomplished or increase the manpower requirements to complete a given task.

Heat Acclimation and Physical Fitness

Heat acclimation and aerobic training both im-

EXHIBIT 3-1

ACTIONS OF HEAT ACCLIMATIZATION

Thermal Comfort: Improved
- Core Temperature: Reduced
- Sweating: Improved
  - Earlier onset
  - Higher rate
  - Redistribution (tropic)
  - Hidromeiosis resistance (tropic)
- Skin Blood Flow: Increased
  - Earlier onset
  - Higher flow

Exercise Performance: Improved
- Metabolic Rate: Lowered
- Cardiovascular Stability: Improved
  - Heart rate: lowered
  - Stroke volume: increased
  - Blood pressure: better defended
- Fluid Balance: Improved
  - Thirst: improved
  - Electrolyte loss: reduced
  - Total body water: increased
  - Plasma volume: increased and better defended

Reprinted with permission from Montain SJ, Maughan RJ, Sawka MN. Heat acclimatization strategies for the 1996 Summer Olympics. *Athletic Ther Today.* 1996;1:43.

pose heat stress on the body that induces an elevated core temperature and increased sweating rate. Repeated exposure to this heat stress will result in an improved ability to thermoregulate.

Heat Acclimation

Heat acclimation results in biological adaptations that reduce the negative effects of heat stress. The magnitude of the biological adaptations depends largely on the intensity, duration, frequency, and number of heat exposures. Thus daily sessions of exercise in the heat, so as to elevate both core and skin temperatures and provoke profuse sweating, are the most effective method for developing heat acclimation. However, even resting in the heat results in some acclimation, although to a lesser degree.[182–185] During the initial heat exposure, there are high levels of physiological strain, as manifested by elevated core temperature and heart rate; but the strain decreases progressively with each day of acclimation (see Figure 2-14 in Chapter 2, Human Adaptation to Hot Environments). Exhibit 3-1 provides a brief description of the actions of heat acclimation.[185] The benefits of heat acclimation are achieved by improved sweating and skin blood flow responses, better maintenance of fluid balance and cardiovascular stability, and a lowered metabolic rate.[25,184,185]

Heat acclimation can improve performance quite dramatically so that acclimated subjects can easily complete exercise in the heat, which earlier was difficult or impossible to achieve. Figure 3-22 depicts the improvement in exercise–heat tolerance time for 24 men who attempted 100 minutes of treadmill exercise at 49°C, 20% rh, for 7 days. This figure shows that no one completed the 100-minute walk on day 1; however, 40% were successful by day 3, 80% by day 5, and all but one of these men were successful by the seventh acclimation day.[25]

During acclimation through daily exercise in a hot climate, most of the improvement in heart rate, skin and core temperatures, and sweat rate is achieved during the first week of exposure, although there is no sharp end to the improvement.[25,184] Heart rate shows the most rapid reduction, most of which occurs in 4 to 5 days.[25,184] The thermoregulatory acclimation response,[25,184] improved sweating response, and greater ease of walking develop more slowly.[25,184] Heat acclimation is transient and gradually disappears if not maintained by repeated heat exposure, but there is considerable variability in the literature concerning its rate of decay. High levels of physical fitness[186]

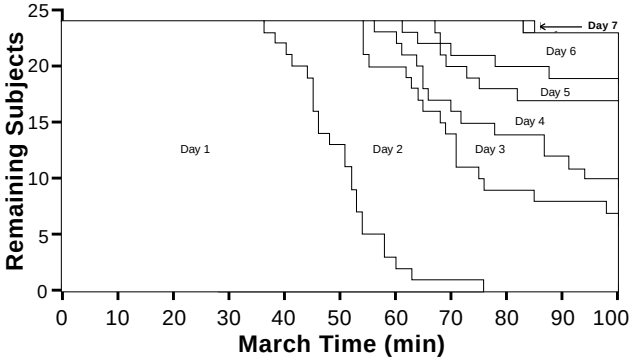

Fig. 3-22. Day-to-day improvements in exercise–heat tolerance of 24 men participating in a dry-heat acclimation program, expressed as the number able to complete a 100-minute exercise–heat exposure each day; dry bulb temperature 49°C, wet bulb temperature 27°C (relative humidity 20%). Adapted with permission from Pandolf KB, Young AJ. Environmental extremes and performance. In: Shephard RJ, Åstrand PO, eds. *Endurance in Sport.* Oxford, United Kingdom: Blackwell; 1992: 271.

and perhaps warm weather[117] also favor retention of acclimation, but there is conflicting evidence on the effect of the weather.[187] Finally, the literature suggests that dry-heat acclimation is retained longer than humid-heat acclimation.[120,182,183,186,188–192]

Physical Fitness

An improvement in $\dot{V}O_2$max achieved through endurance training in a temperate climate reduces physiological strain and increases physical exercise capabilities in the heat,[25,115,168] and endurance-trained individuals exercising in the heat exhibit many of the characteristics of heat-acclimated individuals. In addition, high $\dot{V}O_2$max facilitates acquisition of heat acclimation,[186] and may reduce susceptibility to heat injury or heat illness.[193]

From data collected in both hot–humid and hot–dry environments, it has been estimated that a person's $\dot{V}O_2$max accounts for approximately 44% of the variability in core temperature after 3 hours of exercise in the heat, or the number of days of heat acclimation required to achieve a steady state in final core temperature, as illustrated in Figure 3-23.[194] However, endurance training alone does not entirely replace the benefits of heat acclimation produced by a program of exercise in the heat.[43,195,196]

High $\dot{V}O_2$max is not invariably associated with improved heat tolerance. It is likely that for endurance training to improve thermoregulatory responses during exercise in the heat, the training sessions must elicit substantial elevations of core temperature and sweating rate.[197,198] Investigators[198] who compared thermoregulatory responses of six skiers ($\dot{V}O_2$max = 66.5 mL/kg/min) with those of four swimmers ($\dot{V}O_2$max = 65.8 mL/kg/min) found that skiers were more heat-tolerant and better acclimatized than swimmers, and attributed the difference to a smaller increase in the swimmers' core temperatures produced by training in cold water. In support of this interpretation, other investigators[197] found that 4 weeks of training by cycle exercise in 20°C water increased $\dot{V}O_2$max by about 15% but did not improve thermoregulation during exercise–heat stress.

To achieve optimal thermoregulatory results from endurance training in temperate climates, either strenuous interval training or continuous training at an intensity greater than 50% $\dot{V}O_2$max should be employed.[25,185,194] Lesser training intensities produce questionable effects on performance during exercise–heat stress.[199] The endurance training must last at least 1 week,[200,201] and some authors[194] show that the best improvements require 8 to 12 weeks of training.

Rehydration

Major battles have been decided by water availability to the opposing forces, but Adolph and associates[53] were the first to study in a systematic way the importance of fluid replacement on the ability to perform military tasks in the heat. The importance of hydration during military operations was demonstrated more recently: dehydration-induced heatstroke was believed responsible for 20,000 deaths among Egyptian troops during the 1967 Arab–Israeli Six-Day War.[56] Physical exercise and

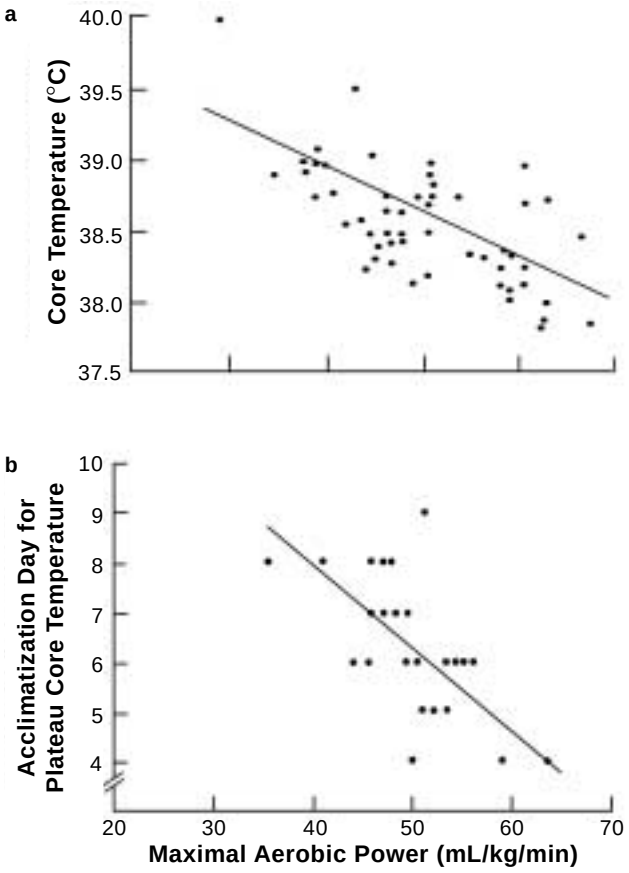

Fig. 3-23. Relationship between (**a**) maximal aerobic power and core (rectal) temperature in a hot–humid environment (n = 26) and (**b**) maximal aerobic power and plateau core (rectal) temperature during dry-heat exposures was first achieved (n = 24, r = –0.68). The equation describing the relation between the variables in (a) is 40.15 – 0.03 $\dot{V}O_2$max; the correlation coefficient is –0.65. The equation describing the relation between the variables in (b) is 14.49 – 0.165 $\dot{V}O_2$max; the correlation coefficient is –0.68. Adapted with permission from Pandolf KB. Effects of physical training and cardiorespiratory physical fitness on exercise-heat tolerance: Recent observations. *Med Sci Sports.* 1979;11:60–65.

heat stress will cause both fluid and electrolyte imbalances that need to be corrected.[146,202–204] The goal of drinking is to replace fluid (and sometimes electrolytes also) lost by sweating. The requirements for fluid replacement depend on losses through sweating, which, in turn, are determined by environmental conditions, physical activity level, and the clothing and equipment worn, as well as by an individual's own characteristics.

Military operations in hot climates may require high sweating rates over many days.[202,203] For example, soldiers might have sweating rates of 0.3 to 1.2 L/h while performing routine military activities,[53,205–207] and those wearing protective clothing might have sweating rates of 1 to 2 L/h while performing light-intensity exercise.[6,208,209] These high sweating rates will increase daily fluid needs. Figure 3-24 presents the influence of climate and physical activity level on daily fluid requirements.[210,211] Daily fluid requirements range (for sedentary to very active soldiers) from 2 to 4 L/d in temperate climates and from 4 to 12 L/d in hot climates.

Electrolytes—primarily sodium chloride and to a lesser extent potassium, calcium, and magnesium—are contained in sweat. Sweat sodium concentration averages approximately 35 mEq/L (range < 10–70 mEq/L) and varies depending on diet, sweating rate, hydration, and heat acclimation level.[212,213] Sweat glands reabsorb sodium by active transport, and because the ability to reabsorb sweat sodium does not increase in proportion to the sweating rate, the concentration of sodium increases at high sweating rates.[25] Heat acclimation improves the ability to reabsorb sodium, so acclimated persons have lower sweat sodium concentrations (> 50% reduction) for any sweating rate.[213] Average concentrations of other electrolytes in sweat are $[K^+]$, 5 mEq/L; $[Ca^{2+}]$, 1 mEq/L; and $[Mg^{2+}]$, 0.8 mEq/L,[212] and neither gender nor age appears to have a marked effect on these concentrations.[214,215] Sweat also contain vitamins, but in very low concentrations.[216] Except for sodium, losses of solute in sweat are so low that supplementation to replace the losses is unnecessary. However, if sweating rates are high, sodium losses are also high, and providing some extra salt with meals or in beverages may be beneficial to unacclimatized troops. If troops are eating normally, supplemental salt is not necessary,[217] except for their first several days of heat exposure,[202,204] as normal dietary sodium intake will cover the sweat losses[202–204] once troops become acclimatized. Salt pills have been overused in the past and may be harmful in the presence of a water deficit. Therefore, troops should not take salt pills unless advised to do so by a physician.

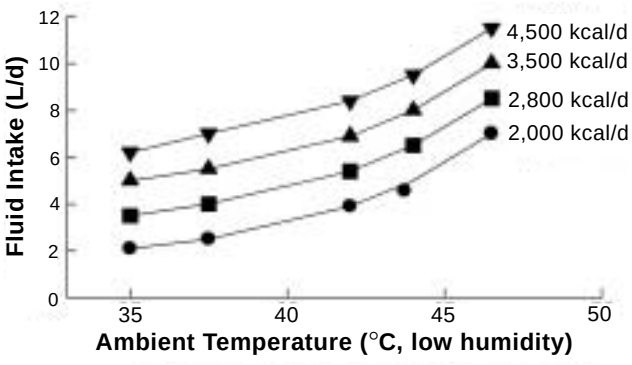

Fig. 3-24. Daily fluid intake plotted as a function of ambient temperature and metabolic rate. On the curves, top to bottom: 4,500 kcal/d represents heavy work; 3,500 kcal/d, moderate work; 2,800 kcal/d, light work; and 2,000 kcal/d, resting. (1) Data source: Nelson N, Eichna LW, Bean WB. *Determination of Water and Salt Requirements for Desert Operations.* Fort Knox, Ky: Armored Force Medical Research Laboratory; 1943. Report 2-6. (2) Adapted with permission from Greenleaf JE. Environmental issues that influence intake of replacement beverages. In: Marriott BM, ed. *Fluid Replacement and Heat Stress*. Washington, DC: National Academy Press; 1994: 205.

The problem of fluid replacement is to match fluid intakes to evaporative and renal losses in order to avoid dehydration. Thirst is probably not perceived until a water deficit of 2% of body weight is incurred,[53,218,219] and thirst provides a poor index of body water needs.[53,218,220] Therefore, ad libitum water intake during exercise in the heat results in incomplete replacement of body water losses,[53,218] (Figure 3-25) and physically active troops will typically dehydrate by 2% to as much as 8% of body weight (dehydration is measured by change in body weight) in situations of heat stress and prolonged high sweat loss in the field. Note that at a sweat rate of 1.0 L/h, the soldiers are underconsuming fluids by about 0.5 L.[53] As a result, some dehydration is likely to occur during exercise in the heat, unless troops make a conscious effort to drink, even when not thirsty. Flavoring and cooling fluids will increase palatability and help minimize voluntary dehydration.[218,221–223] On the other hand, drinking carbonated beverages may give a false feeling of stomach fullness and reduce fluid consumption.[224] People exercising in the heat usually fully rehydrate only at mealtime, when fluid intake is stimulated by consuming food,[53,203] which increases thirst and fluid intake above what would occur if only fluids were consumed. Meals should be scheduled at specific times and provide enough time for troops to

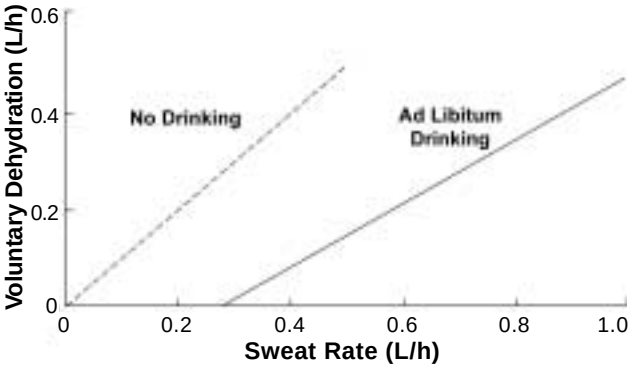

Fig. 3-25. Relationship between sweating rate and rate of dehydration (water deficit, based on change in body weight) without drinking (broken line) and during ad libitum drinking (solid line). Voluntary dehydration is dehydration that occurs during ad libitum drinking. Data are for heat-acclimated soldiers walking in the desert. Adapted from Adolph EF, Associates: *Physiology of Man in the Desert*. New York, NY: Intersciences, Inc; 1947: 259.

drink to satiation. If they do not eat regular meals, their existing water deficits will persist, and performance will suffer. Therefore, the importance of meals for promoting full rehydration cannot be overstressed.

Troops should be fully hydrated when they begin exercise or heat exposure. A good habit is to drink 400 to 600 mL approximately 1 hour before beginning activity to offset any preexisting water deficit.[146,204] If this volume does not induce urination prior to exercise, it is likely that the person is already dehydrated. Preexisting dehydration can be a problem during early morning activities because water loss occurs during sleep. Adequate fluids should be available during physical activity, and troops should be encouraged to drink whenever they get the opportunity. The rate at which fluids can be replaced by mouth is limited by the rate of gastric emptying, and the rate of fluid absorption by the small intestine. Gastric emptying rate is believed to be the most limiting factor, and, for most persons, maximal emptying rates approximate 1 to 1.2 L/h.[146,225,226] However, gastric emptying and fluid absorption rates vary among individuals, so each person must determine his or her own best drinking pattern. If fluid losses during exercise and heat stress exceed the gastric emptying rate, the remaining deficits should be replaced later. Because gastric emptying rate is greatest when the stomach is full,[225] it can be facilitated by drinking enough to keep the stomach full.[226] To maximize fluid replacement during exercise, it is recommended that troops drink a large volume (500–1,000 mL) initially to fill the stomach, followed by regular drinking of smaller volumes (200–400 mL) to keep the stomach as full as is comfortable. (The actual volumes will vary depending on expected sweating rates, body size, tolerance, and other individual factors.)

Dehydration reduces the gastric emptying rate of ingested fluids during exercise in the heat.[227,228] In one study, for example, gastric emptying rate was reduced approximately 20% to 25% when subjects were hypohydrated by 5% of body weight,[227] and the reduction was related to increased core temperature. Therefore, it is important to begin fluid intake during the early stages of exercise–heat stress, not only to minimize dehydration but also to maximize absorption of the ingested fluids.

Exercise/Rest Cycles

In some conditions of high exercise–heat stress (especially uncompensable heat stress), the ability to tolerate the exercise–heat stress is determined largely by biophysical limits on heat dissipation, which are determined by the environment and the clothing and equipment that are worn. In such conditions (eg, wearing protective clothing), physiological adaptations obtained from heat acclimation or aerobic training can do little to improve exercise performance.[229–231] Even if little can be done to reduce the effects of clothing and climate, the metabolic heat load can often be managed. The options are to exercise at a given metabolic rate until exhaustion from heat strain occurs, or to decrease the metabolic rate and therefore dissipate the heat load.[232,233] Metabolic rate can be decreased by reducing the exercise intensity or using rest periods (exercise/rest cycles) or both to lower the time-averaged metabolic rate. Decreasing the metabolic rate will extend tolerance time, although it may result in less total work being accomplished in a given period of time.

Exercise/rest cycles have been used in both military[13] and civilian[12,234] occupational settings to extend exercise tolerance time in high heat stress conditions. (During military operations, however, this approach is not always acceptable because it increases the time or personnel requirements to complete a task, and so may jeopardize the mission.) Figure 3-26 presents the relationship between exercise tolerance time and the average metabolic rate for troops working in the heat (30°C, 50% rh) while wearing chemical protective clothing either open (solid curve) or closed (dotted curve).[235] When 1:1 exercise/rest cycles are employed to reduce the average metabolic rate, the tolerance times are increased in a hyperbolic manner. Therefore, tolerance

times are closely related to the average metabolic rate when wearing protective clothing in climatic heat stress conditions.

A critical problem for managing heat stress during military operations is determining the effectiveness of exercise/rest cycles and calculating the optimal ratios.[41] The US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts, has developed a family of mathematical models[207,235–239] that enable predictions that are customized to the climatic conditions, clothing and equipment worn, and the soldier's state of hydration and acclimatization. Tables 3-4 and 3-5 present examples of exercise/rest cycles for a matrix of conditions in desert[13] and warm, humid[240] climates. Calculations are for four exercise intensity levels (very light = 150 W, light = 250 W, moderate = 425 W, high = 600 W), six levels of WBGT, and two clothing configurations. The calculation represents the recommended number of minutes per hour for exercise, with the remaining time spent resting. For example, "34" means that the exercise/rest ratio is 34 minutes of exercise to 26 minutes of rest. These tables provide a margin of safety because the model used to generate these tables is somewhat conservative.

Microclimate Cooling

Because protective clothing reduces both evaporative and dry heat exchange, it impairs a person's ability to dissipate body heat and can result in extreme elevations in body temperatures during exercise in moderate climatic conditions.[4,6,173,209,241–243] Because the clothing has high insulation and low permeability to water vapor, heat and moisture from the body are retained within the clothing, where they create a warm, humid microenvironment that seriously impairs heat loss from the body. To alleviate heat stress under these conditions, microclimate cooling systems have been developed[208,244–246] that cool the microenvironment immediately surrounding the person, rather than the macroenvironment (ie, the working area).[247]

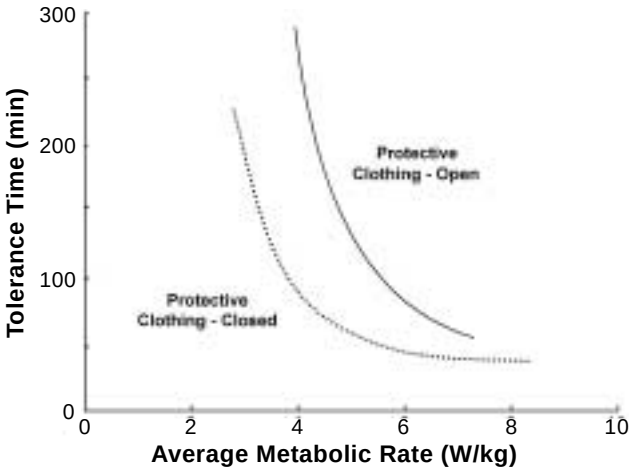

Fig. 3-26. Relationship between exercise tolerance time and the average metabolic rate while subjects wear protective clothing in the heat (35°C, 50% rh). Adapted with permission from Mclellan TM, Jacobs I, Bain JB. Continuous vs intermittent work with Canadian Forces NBC clothing. *Aviat Space Environ Med.* 1993;64:596.

Microclimate cooling systems use circulating cooled air, liquid in tubes next to the skin, or ice-packet vests to remove body heat.[208,245] In addition, microclimate cooling facilitates heat loss by maintaining the temperature gradient between the body core and the cooled skin. The amount of heat transferred from the body to any microclimate system depends on several factors, including the size and location of skin regions being cooled, coolant temperature and flow rate, and insulation from the ambient heat.[87,248–250]

Air-cooled garments are lighter to wear and rely on sweat evaporation to cool a person. Although air is not as effective as water in removing heat by convection due to the difference in specific heat, air-cooled systems promote evaporation of sweat and are effective in reducing heat strain; and in some environments they are believed to be as effective as water-cooled devices.[245] In addition, air-cooled vests keep the skin drier than do liquid-cooled systems, thereby improving thermal comfort. If the environment is not contaminated by biological or chemical agents, untreated ambient air can be circulated through an air-cooled system. However, warm, humid air will reduce cooling due to evaporation of sweat. On the other hand, air that is too hot and dry may cause local skin irritation.[246]

Microclimate cooling systems that utilize ice as the cooling medium are not as effective as either liquid- or air-cooled systems.[208,245] Furthermore, the cooling lasts only until the ice has completely melted, so the ice must periodically be replenished. The logistical problems associated with ice-cooled systems make them impractical for use as cooling devices in all but very short-duration situations.

Microclimate cooling systems are effective in alleviating heat stress and extending exercise capabilities in soldiers who are wearing protective clothing,[246,251–256] as illustrated in Figures 3-27 and 3-28. These curves, which are based on a modeling analysis, illustrate the effectiveness of microclimate cooling to extend physical exercise to 5 hours in hot,

TABLE 3-4

RECOMMENDED EXERCISE/REST CYCLES (MINUTES OF EXERCISE PER HOUR) IN HOT, DRY CLIMATES

			DBDU				DBDU and Flak Vest			
WBGT	**T_a (°F)**	**rh (%)**	**VL**	**L**	**M**	**H**	**VL**	**L**	**M**	**H**
82°F (28°C)	87	20	NL	NL	33	21	NL	NL	36	23
86°F (30°C)	91	20	NL	NL	30	20	NL	NL	34	22
88°F (31°C)	94	20	NL	NL	28	18	NL	NL	31	20
90°F (32°C)	96	20	NL	NL	26	17	NL	NL	30	19
98°F (37°C)	120	20	NL	NF	NF	NF	NL	9	NF	NF
115°F (46°C)	120	60	NF	NF	NF	NF	NL	NF	NF	NF

DBDU: desert battle dress uniform; H: heavy work intensity; L: light work intensity; M: moderate work intensity; NF: not feasible (exercise/rest cycle not feasible); NL: no limit (continuous exercise possible); rh: relative humidity; T_a: ambient temperature (°F); VL: very light work intensity; WBGT: wet bulb globe temperature

NOTE: This table provides, for four levels of work intensity, the number of minutes of exercise per hour in exercise/rest schedules tailored to the conditions specified. Spend the remainder of the hour at rest. This model was prepared using the USARIEM Heat Strain Model. Assumptions used in this model include: (1) troops fully hydrated, rested, and acclimated; (2) windspeed = 2 m/s; (3) clear skies (full solar load); and (4) heat casualties < 5%. This guidance should not be used as a substitute for common sense or experience. Individual requirements vary greatly. Appearance of heat casualties is evidence that the selected exercise/rest cycle is inappropriate for the conditions.

Adapted from Sawka MN, Modrow HE, Kolka MA, et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders.* Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. Technical Note 95-1.

humid (35°C T_a, 70% rh) and desert (49°C T_a, 20% rh) climates, respectively.[245] The cooling extraction rates on the abscissa represent heat actually removed from the body. These values may be only 30% to 80% of the heat taken up by the cooling systems because of heat gained by the systems from the environment. In each environment, curves are calculated for soldiers wearing chemical protective clothing while exercising at five different metabolic rates, from 250 to 600 W, which represent the range

TABLE 3-5

RECOMMENDED EXERCISE/REST CYCLES (MINUTES OF EXERCISE PER HOUR) IN HOT, WET CLIMATES

			DBDU				DBDU and Flak Vest			
WBGT	**T_a (°F)**	**rh (%)**	**VL**	**L**	**M**	**H**	**VL**	**L**	**M**	**H**
82°F (28°C)	82.9	75	NL	NL	NL	25	NL	NL	NL	29
86°F (30°C)	87.1	75	NL	NL	33	21	NL	NL	NL	27
88°F (31°C)	89.2	75	NL	NL	29	18	NL	NL	37	23
90°F (32°C)	91.3	75	NL	NL	25	16	NL	NL	32	20
98°F (36°C)	99.7	75	NL	NF	NF	NF	NL	16	6	NF
110°F (43°C)	109.0	50	NL	NF	NF	NF	NL	23	10	5

DBDU: desert battle dress uniform; H: heavy work intensity; L: light work intensity; M: moderate work intensity; NF: not feasible (exercise/rest cycle not feasible); NL: no limit (continuous exercise possible); rh: relative humidity; T_a: ambient temperature (°F); VL: very light work intensity; WBGT: wet bulb globe temperature

Adapted from Burr RE, Modrow HE, King N, et al. *Sustaining Health and Performance in Haiti: Guidance for Small Unit Leaders.* Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. USARIEM Technical Note 94-4.

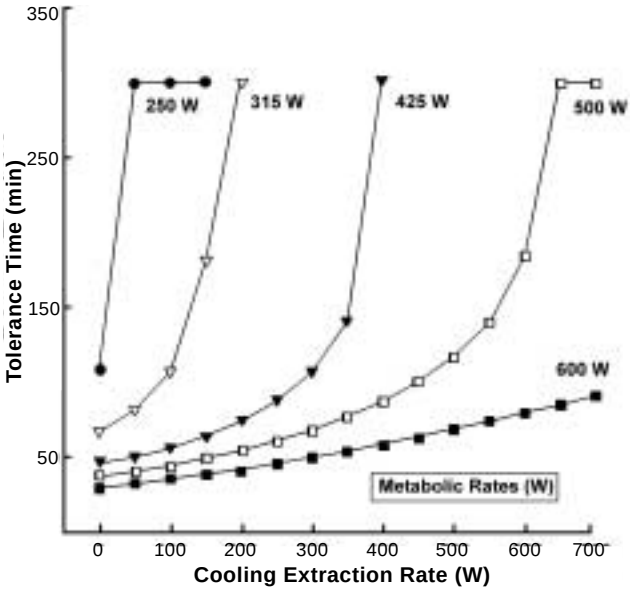

Fig. 3-27. The relationship between microclimate cooling and endurance times at selected metabolic rates (W) when subjects are wearing chemical protective clothing in a hot, humid climate (35°C, 70% rh). Reprinted from Pandolf KB, Gonzalez JA, Sawka MN. *An Updated Review: Microclimate Cooling of Protective Overgarments in the Heat.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1995: 69. USARIEM Technical Report T95-7.

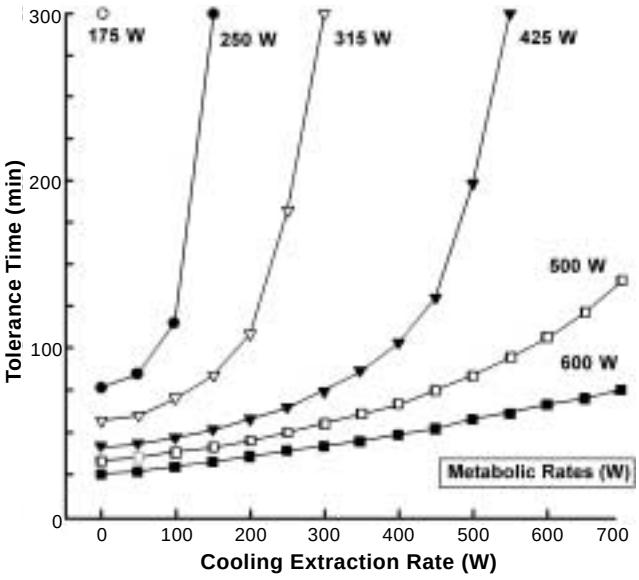

Fig. 3-28. The relationship between microclimate cooling and endurance times at selected metabolic rates (W) when subjects are wearing chemical protective clothing in a desert climate (49°C, 20% rh). The value 175 W is not a control; it demonstrates that at this lower rate, 300 min of tolerance time is possible. Reprinted from Pandolf KB, Gonzalez JA, Sawka MN. *An Updated Review: Microclimate Cooling of Protective Overgarments in the Heat.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1995: 69. USARIEM Technical Report T95-7.

of tasks performed by soldiers for extended periods during military operations.[41] These curves indicate that if heat extraction rates of 300 to 400 W are achieved, then microclimate cooling can substantially improve tolerance time (although microclimate cooling alone is not sufficient to alleviate the problem). Note that at higher metabolic rates, microclimate cooling will need to be combined with exercise/rest cycles (which lower the metabolic rate) to markedly extend performance.[251]

BIOMEDICAL ISSUES

The biomedical status of a soldier is an important modifier of environmental and mission-related factors to influence the physical work capacity and tolerance to heat stress. Biomedical factors include the soldier's gender, race, age, biological rhythms, health, and medications. An increased reliance on reserve forces, who are older and may use medications for health reasons, pose deployment considerations that were not present a decade ago.

Gender and Race

Thermoregulatory responses to heat exposure at rest appear to be similar for both genders.[257] Although it was once believed that women are less heat tolerant than men, it now seems that the studies on which that belief was based compared relatively unfit women to more fit men.[258,259] Recent research demonstrated that if populations of men and women are matched for aerobic fitness or compared at the same relative exercise intensity, they have similar tolerance and body temperature responses to exercise in both dry and humid heat,[260–263] and respond similarly to heat acclimation[260,261] and dehydration.[67] Core temperature changes with the phase of the menstrual cycle in a cyclic fashion,[258,259] reaching its lowest point just before ovulation, and then rising 0.4 to 0.6 Centigrade degrees over the next few days to a plateau that persists throughout the luteal phase. This pattern results from a similar variation in the thermoregulatory set point, and thus is accompanied by corresponding changes in the thresholds for all the thermoregula-

tory responses.[264] There is some indication, as discussed in the chapter by Kolka and Stephenson, Women's Health in Deployment, in the third volume of *Medical Aspects of Harsh Environments*, of changes in heat tolerance with the phase of the menstrual cycle.

There has long been a perception that descendants of ethnic groups who are indigenous to hot climates are more heat tolerant than are descendants of ethnic groups who are indigenous to cool climates. There is some support in the literature for this perception, mostly based on comparison of white Americans, Britons, or South Africans with persons of tropical African descent. The reported differences apply mostly to hot, humid conditions,[265–269] with the advantage in humid heat being enjoyed by tropical Africans and their descendants. (This topic is explored more fully elsewhere.[184]) These results are consistent with the notion that populations gradually acquire certain biological adaptions in the course of living many generations in a particular environment. In interpreting these studies, however, it should be remembered that the subject populations were not matched for factors such as aerobic fitness and body composition. In fact, Robinson[270] said that such differences in responses (including those that he had reported 11 years earlier[266]) were

> probably due largely to differences in nutritional status, acclimatization, and training and not dependent on racial characteristics or length of residence in the tropics.[270(p73)]

Age

For the general population, physiological heat strain in middle-aged men and women during exercise is greater than in younger adults, particularly in the heat,[198,271–276] as manifested by higher core and mean skin temperatures, heart rate, and skin blood flow, as well as reductions in sweating. These differences between young and middle-aged adults appear to be greater during continuous exercise in the heat than during intermittent exercise or heat exposure at rest.[275,276] In addition, the differences in heat strain between young and middle-aged individuals were accentuated during prolonged exposures[273,277] and at higher levels of environmental heat stress, particularly those exceeding the prescriptive zone.[274,276]

It is not clear from the foregoing reports how much of this lesser tolerance was the result of age per se, and how much could be explained by other factors such as poorer health and decreased physical activity or low aerobic fitness. However, if middle-aged and younger men and women are matched for aerobic fitness or habitual physical activity levels, and for certain anthropometric factors (such as body surface area, surface area-to-weight ratio, and percentage of body fat), the heat-strain differences between age groups tends to disappear.[278–282] Two of these reports[280,281] emphasized the importance of maintaining physical fitness and body weight in preserving exercise–heat tolerance as one ages.

Middle-aged men and women can acclimate to exercise in the heat; but whether they acclimate as rapidly as younger persons, or achieve the same degree of acclimation, is not yet established.[283] Some studies[280,281] report that during heat acclimation, the thermoregulatory responses of physically fit middle-aged men improved as rapidly and to the same degree as those of younger men. Other studies,[275,278] however, report that although middle-aged men and women acclimate to exercise–heat stress, the degree of heat acclimation achieved was less than that for younger persons.

Circadian Patterns and Sleep Loss

Core temperature at rest varies with time of day in a sinusoidal fashion, with the minimum at night, and the maximum, which is 0.5°C to 1°C higher, occurring in the late afternoon or evening. This pattern is an example of a *circadian* rhythm (ie, a rhythmic pattern in a physiological function with a period of about 1 day). The circadian rhythm in core temperature results from a similar circadian rhythm in the thermoregulatory set point, and is accompanied by corresponding changes in the thresholds for all the thermoregulatory responses that have been studied.[258,284] Sleep deprivation disrupts the circadian rhythm, delaying the time of minimum core temperature at rest, and altering thermoregulatory responses during exercise.[285,286] In addition, sleep deprivation is reported[287,288] to impair heat tolerance and negate the effects of heat acclimation, but it is not known whether the effects on heat tolerance are mediated through the thermoregulatory changes or through other mechanisms.

Skin Disorders

Certain skin disorders impair the ability to dissipate body heat, and thus increase thermal strain.[25] Heat rash and sunburn may have substantial thermoregulatory effects, and all too often their adverse effects on exercise–heat tolerance fail to get the attention they deserve.

Artificially induced miliaria rubra (heat rash) over as little as 20% of the body surface causes an observable reduction in exercise–heat tolerance[289];

and involvement of 40% or more of the body surface markedly reduces tolerance time during exercise in the heat (49°C, 20% rh) and causes greater heat storage compared with responses in a nonrash state.[289,290] These effects may persist for up to 3 weeks after the rash has resolved clinically.[290] The degree of heat intolerance that occurs depends both on the total area of skin affected and on the specific region of the body, and that region's normal sweating responses. Thus, smaller rashed areas of the trunk may, because of the greater sweating capacity of normal trunk skin, affect responses to dry-heat stress as much as larger rashed areas of the limbs. Observable sweating was absent in the rashed areas, perhaps because of physical occlusion of the affected sweat glands by keratotic plugs.[290]

Mild artificial sunburn impairs sweat gland activity during exercise in the heat.[291] Both the local sweating sensitivity and steady state sweating rate from the burned areas are reduced 24 hours after the sunburn compared with values before the sunburn, but they return to normal within 1 week. Mild sunburn thus appears to have a locally mediated effect on both the responsiveness of the sweat gland and its capacity to deliver sweat to the skin surface. These effects perhaps result from damage to the cuboidal epithelial cells composing the sweat duct, which blocks the egress of sweat from the affected duct. More severe levels of sunburn may have more profound thermoregulatory consequences during exercise–heat stress.

Medications

For protection against organophosphate (anticholinesterase) nerve agents, military personnel are issued nerve agent antidote kits containing atropine sulfate autoinjectors, and nerve agent pretreatment kits containing pyridostigmine bromide pills. Atropine binds with muscarinic receptors for acetylcholine, where atropine acts as a competitive antagonist to acetylcholine. Atropine thus reduces sweating and impairs tolerance to exercise–heat stress.[292–298] Pyridostigmine bromide, a reversible anticholinesterase, increases sweating and inhibits cutaneous vasodilation during exercise–heat stress.[299,300] At the standard pretreatment dosage (30 mg, 3 times daily), pyridostigmine administration has little effect on tolerance to exercise–heat stress.[301] Several other drugs that have some anticholinergic action, plus a number of others,[57,302,303] impair tolerance to exercise–heat stress and have been associated with heatstroke.

Many senior enlisted personnel, officers, and reservists take medications (including diuretics, ß-adrenergic blockers, and angiotensin-converting enzyme inhibitors) for treatment of hypertension and arrhythmias. These drugs have cardiovascular effects via modifying heart rate, blood pressure, and vascular resistance. During exercise–heat stress, ß-adrenergic blockers reduce active cutaneous vasodilation[304–306] and sweating[306,307] responses, and these actions combine to increase core temperature.[304,306,307] Nonselective ß-adrenergic blockers, such as propranolol, have greater adverse thermoregulatory effects than selective blockers, such as metoprolol[306] and atenolol.[305,308] Angiotensin-converting enzyme inhibitors, such as enalapril, do not impair thermoregulatory responses to acute exercise–heat stress[309]; however, this drug might reduce thirst.[310] During short-term administration, diuretics reduce extracellular fluid volume, plasma volume, and tolerance to exercise–heat stress, as discussed earlier. With longer-term administration, plasma and extracellular fluid volumes return toward normal, but some authors report persistent reductions in plasma or blood volume,[311–313] suggesting the possibility of long-term impairment in tolerance to exercise–heat stress. Little is known about the effects of chronic administration of antihypertensive drugs on the ability to acclimatize to heat.

SUMMARY

Troops participating in all military deployments may encounter heat stress. The magnitude of heat stress encountered is dependent on complex interactions among climatic conditions, exercise intensity, health and nutrition status, and clothing and equipment worn. Body temperature is regulated by two parallel processes: behavioral regulation and physiological regulation. In combat, behavioral thermoregulatory drives are overridden by motivation to successfully complete the mission. Physiological thermoregulation relies on increased skin blood flow and sweating to dissipate body heat to the climate. The hotter the climate the greater the dependence on sweating and evaporative heat loss. Therefore, in hot climates, sweating rates will be high and drinking must be emphasized to avoid dehydration. Heat stress decreases the capacity for both maximal and submaximal intensity exercise, and dehydration magnifies these decrements in performance capacity. Strategies to manage heat stress and sustain exercise capacity include improving the troops' ability to dissipate body heat (by heat acclimation and physical

training programs), managing the amount of heat stress (reducing climatic exposure, wearing less clothing, reducing physical exercise, providing microclimate cooling), and maintaining hydration and health (avoiding certain medications, sunburn, and sleep loss). Mathematical models such as the USARIEM Heat Strain Model[41] can provide specific guidance to manage heat stress during military operations.

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Chapter 4

PSYCHOLOGICAL ASPECTS OF MILITARY PERFORMANCE IN HOT ENVIRONMENTS

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INTRODUCTION

The success of military operations as well as survival in combat greatly depends on the ability of military personnel to deal effectively with environmental conditions. Throughout military history, more deaths and injuries in war have been attributed to extreme weather conditions than to battle casualties. By far the most important weather extreme in military operations is heat, because so many strategically important world areas are in hot regions, and because heat exposure can seriously impair human performance in so many ways.

The military needs to understand how cognitive, behavioral, and subjective responses vary with severity of heat stress not only because heat stress can significantly impair military performance but also because psychological changes often precede the onset of critical physiological changes. As a matter of fact, some have argued that a decrement in psychological performance may be considered a precursor to critical physiological change.[1] Establishing well-defined relationships between climatic conditions and psychological performance has been difficult. During World War II, thermal stress researchers attempted to identify psychological "breaking points" in performance. Unfortunately, the environmental conditions employed to simulate the natural world (combinations of temperature, humidity, wind speed, and exposure time) were not systematically organized, which made it difficult to make broad generalizations about the effects of heat stress on psychological performance.

The physical principles of heat dynamics and the physiology of human thermoregulation during heat exposure are discussed in other chapters of this book. We will address other important issues involved in adjustment to heat exposure that vitally depend on behavioral rather than physical or physiological factors, or both. Although the chapter is primarily organized around the experiences and operational mission requirements of personnel in the US Army and US Marine Corps, the facts and principles apply to all services.

PSYCHOLOGICAL PERFORMANCE IN HOT ENVIRONMENTS

When it is hot, we often hear ourselves or others complain that we cannot do our jobs because we cannot concentrate, that we are tired, lethargic, and just downright uncomfortable. It is a common belief that we need air conditioning, or cool, dry air, to maintain proficient mental performance. We also feel that we need cool, dry air to keep from feeling sticky, or being sweaty, so that we can perform tasks requiring fine manual dexterity; otherwise we lose our grip on tools. In this section, we will discuss human psychological performance in hot environments. We organize psychological performance into behaviors that are mainly intellectual or rational in terms of the task at hand, which we will call *mental*; those that are mainly manual tasks, which we will call *psychomotor*; and those that concern our feelings, moods, and attitudes, which we will call *subjective*.

The performance measures used to assess psychological performance are many and varied. They include those that assess tasks requiring simple and swift reactions to basic changes in the environment. They include sensory tasks, primarily the domains of vision and hearing. They include interpretations of basic changes in the environment, otherwise known as perception, which include the ability to discriminate friendly from enemy targets. They include the ability to perform complex mental tasks that generally require verbal reasoning, mathematical reasoning, or both, and sometimes require the performance of two or more tasks at the same time, called dual task or concurrent task performance.

Figure 4-1 presents the basic psychological model and the main research variables or factors that research psychologists must consider when investigating the relationship between the hot environment and psychological performance. The conceptual model being presented is one in which behavior (B) is a function of the thermal environment (E), the person (P), the task (T), and the situation (S), or $B = f(E,P,T,S)$. Notice that accompanying the environmental variables (air temperature, radiant temperature, humidity, wind, clothing, and duration of exposure) are a host of other relevant, independent variables that may influence how a person responds to a hot environment, as reflected in the various categories of dependent variables (sensation, perception, cognition, psychomotor, and subjective response). For example, it has been found that some people respond differently as a function of their experience or training, intellect, skill at the relevant task, personality, current mood, motivation, and

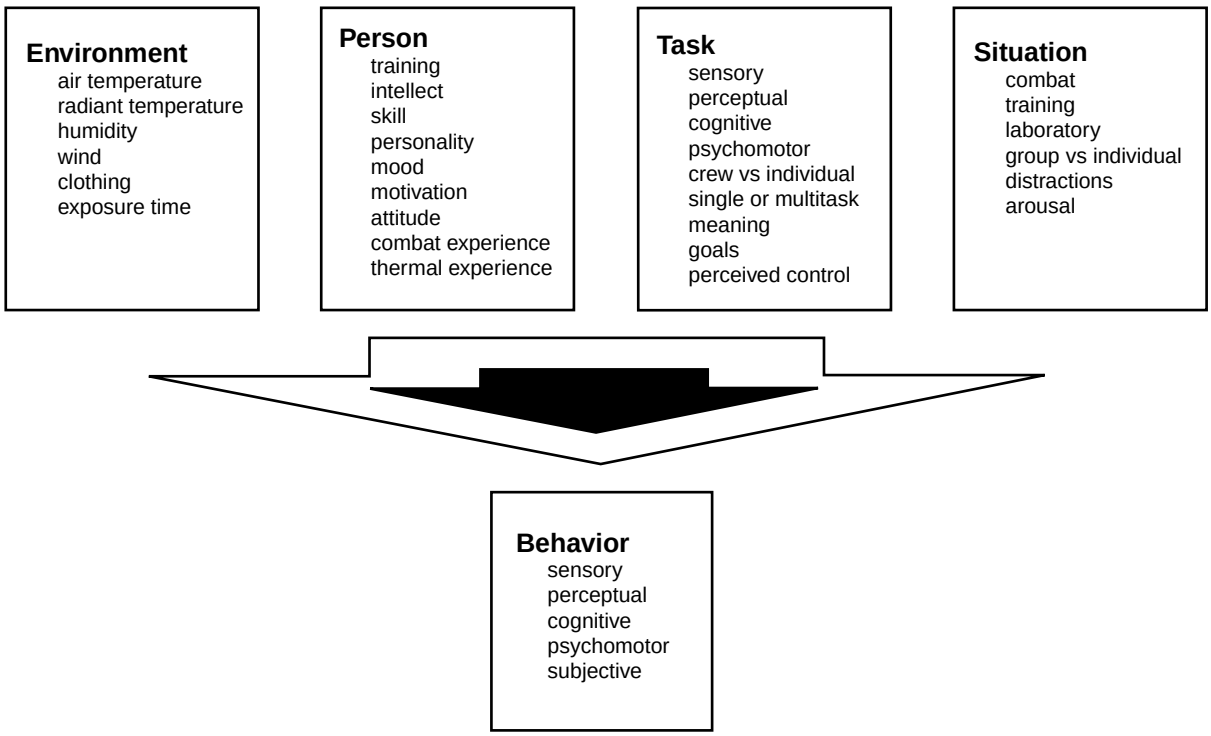

Fig. 4-1. The basic psychological model, in which behavior (the dependent variable) is a function of the environment, the person, the task, and the situation (the independent variables), or B = f(E,P,T,S).

attitude. Military tasks also vary along dimensions such as crew versus individual. And the type of situation in which the task is assessed has been deemed important; for example, the task may vary as a function of whether it is performed in combat, during training, or in a laboratory; whether it is performed in a group setting, whether there are distracting stimuli; and how long the task must be performed before it is considered completed.

Mental Performance

In general, there seems to be agreement that the upper limits of heat exposure for unimpaired mental performance is about 85°F wet bulb globe temperature (WBGT) if the individual is required to perform the task for 2 hours or longer (Figure 4-2). At less than 1 hour on the task, individuals can perform proficiently at higher ambient temperatures approaching 109°F WBGT.[2] WBGT is an index of the thermal environment used to express the combined effects of heat, humidity, and radiation.[3] Continuous, repetitive tasks that are boring tend to be affected most. Interesting tasks requiring frequent and varied responses to the environment are affected less.

Reaction Time

Studies of reaction time traditionally have distinguished between two types of tasks: *simple* reaction time, involving direct response to the onset of a stimulus; and *choice* reaction time, involving a selection of alternative reactions. Simple reaction time is believed to represent mainly neural processing time and is usually shorter than choice reaction time, which involves additional cortical processing. Studies of the effects of ambient heat exposure on both types of reaction time have yielded mixed findings, including increases[4,5] and decreases,[6] as well as no change[6] in reaction time. Acclimation (physiological adjustment to a controlled environment) to prolonged heat exposure has been shown to result in a reduction of initial impairment of simple visual reaction time by heat.[7] Although it has been reported that men have consistently faster reaction times than women,[8] heat had only a negligible overall effect. A study of sustained attention in the heat showed reduced accuracy in a choice reaction time task.[9]

Studies of the effects of direct heating of the body on reaction time have given rather contradictory findings. Heating of the entire body[10] was shown

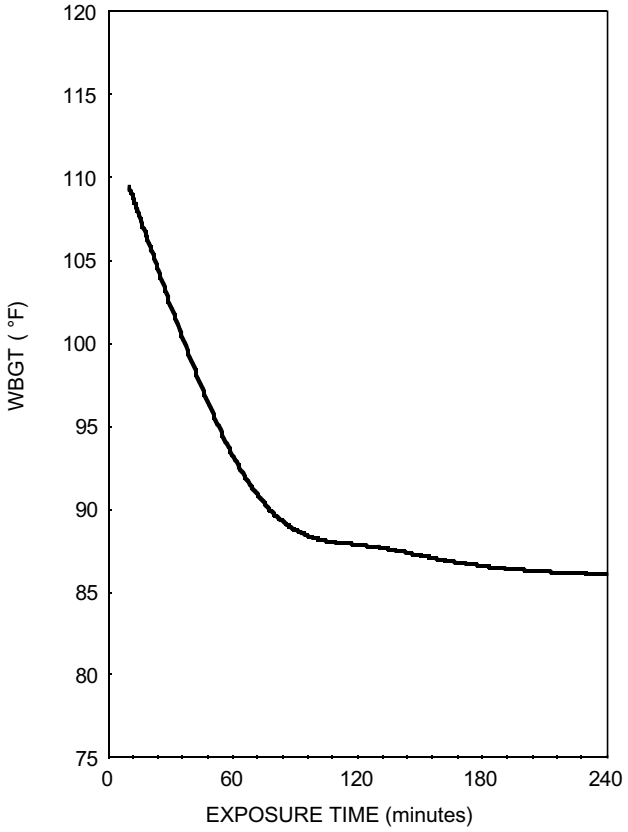

Fig. 4-2. Upper limits of thermal stress (wet bulb globe temperature, WBGT) for unimpaired mental performance as a function of exposure time. Reprinted from National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard–Occupational Exposure to Hot Environments.* Washington, DC: NIOSH; 1972.

to shorten reaction times but to reduce performance accuracy. Warming the head[11] with a heated helmet resulted in increased reaction times but reduced error rates. Based on tympanic measurements, the authors ascribed this effect to an increase in cortical temperature.[11] Because tympanic temperature is affected by the temperature of nearby skin, however, tympanic temperature measurements do not necessarily indicate that the observed responses were the result of changes in cortical temperature.

The role of heat-induced reaction time changes on overall task performance is suggestive rather than definitive. That is, a review of thermal stress effects on human performance[12] shows that interpretation of the available data is complicated by differences in methodology and definition of the basic stimuli. Thus, we cannot draw definitive conclusions about the effects of heat exposure on reaction time because the testing conditions used in the available studies vary considerably, and the resulting findings tend to be contradictory.

Sensation

Studies of sensory processes typically have been conducted on the separate sensory modalities, principally vision, audition, olfaction, taste, skin senses (thermal, tactual, vibratory, pressure), motion detection, vestibular orientation, and pain. Considerably less research has been done involving interactive combinations of the separate modalities.

Most research on effects of heat exposure on sensory action has concerned visual functions, probably because of the major role of vision in human performance. Examination of effects of both hot–dry and hot–humid conditions on visual contrast sensitivity, a measure of brightness and contrast discrimination, showed reduced sensitivity for the higher spatial frequencies.[13] Investigation of hot–dry and hot–humid conditions on visual acuity found diminished acuity at both bright and dim luminance levels.[14]

Some studies of heat effects on vision have involved the additional heat stress generated by wearing encapsulating chemical protective clothing systems at high ambient heat and humidity levels. One study[15] tested heat effects on visual acuity, phoria (a measure of ocular muscle balance), stereopsis (a measure of depth discrimination), and contrast sensitivity. All measures were significantly and progressively impaired over a 2-hour exposure period. Another study[16] investigated similar heat effects on visual detection capability for target locations distributed throughout the entire visual field. Conditions of ambient heat while subjects were wearing chemical protective clothing produced significant systematic increases in response time to all signals; the worst performance occurred when subjects wore chemical protective clothing in high temperatures. One study[17] investigating tactile sensitivity at low, moderate, and high ambient temperatures indicated that sensitivity was greatest at a moderate heat level.

Vigilance

Vigilance is a major element in many important military activities and tasks, such as sentry duty, watch keeping, vehicle operation, instrument monitoring, and surveillance. Analysis of vigilance behavior is a complex problem because it consists of several component behaviors such as attention, alertness, cognitive sensitivity, and judgment and

decision making. Because of the role of vigilance in military activities, it is important for all military personnel, but especially commanders, to understand the effects of stressors such as ambient heat exposure. Most studies that have explored the effects of ambient heat on vigilance performance have used visual monitoring tasks.

In an early study, Mackworth[18] found that visual search performance is best at 85°F, 63% relative humidity (rh), but that visual search tasks are impaired above and below that temperature, even in heat-acclimatized subjects. These findings were supported in later studies by Mortagy and Ramsey[19] and by Pepler,[20] wherein visual vigilance decrements occurred at 102°F, 24% rh; and also below 90°F, 65% rh. Poulton and Edwards[21] and Poulton, Edwards, and Colquhoun[22] also reported that both visual and auditory vigilance tasks are impaired at 100°F, 74% rh. Thus, the optimum conditions for visual vigilance tasks that have been studied appear to be around 85°F, as Mackworth[18] originally reported.

Complex Mental Performance

There is considerable support for the idea that heat impairs complex mental performance. Complex mental processes can be grouped for practical purposes into three categories: storage and retrieval of information (memory); understanding, reasoning, and evaluation (cognition); and interpreting incoming sensory information in terms of previous experience (perception). It has been proposed[23] that impairment of mental performance by heat is a function of the resulting internal body temperature during heat exposure. This notion may be useful in developing a system for explaining decrements in mental tasks under heat exposure. Hancock[24] attempted to validate this view using published findings based on performance of mental and cognitive skills, tracking, and dual task performance under exposure to effective temperatures above 85°F. Effective temperature (ET) is an index of the thermal environment based on the subjective matching by human raters of various experienced combinations of dry bulb temperatures, relative humidities, and air velocities to determine those combinations that produce equivalent feelings of personal comfort.[25,26] Hancock also suggested that practice on the specific tasks involved may be useful in offsetting such decrements.

Although systematic demonstration of heat effects on complex mental performance has been difficult, some militarily meaningful studies have been conducted. Fine and Kobrick[27] studied the effects of heat on complex cognitive tasks involved in soldiers' performance at an artillery fire direction center. For 7 hours at 90°F, 88% rh, five 6-man groups performed message reception and decoding, arithmetic conversions, and reception and recording of meteorological data. All tasks were significantly and similarly impaired by heat, although the participants differed considerably in the degree and type of their stress response. Errors of omission greatly exceeded errors of commission. These same investigators[28] later examined the effects of heat on sustained cognitive performance of soldiers in the heat while clad in chemical protective clothing. After 4 to 5 hours, cognitive performance began to deteriorate markedly, and after 7 hours the percentage of group error on fast-paced tasks ranged from 17% to 23% over control conditions, largely due to errors of omission. Productivity on a slower, self-paced task diminished by 40% from control conditions. Accuracy of map plotting, however, was not markedly affected.

Curley and Hawkins[29] assessed cognitive performance during a 10-day heat acclimatization regimen (in a natural environment), using repeated acquisition and time estimation tasks. The participants walked on a treadmill at 90°F dry bulb, 70°F wet bulb temperature for 155 minutes daily. By the 10th day of heat exposure, all participants were heat-acclimatized, but mean performance on the repeated acquisition task and on the time estimation task was still impaired.

Ramsey and associates[30] demonstrated that in an industrial workplace situation, ambient temperatures above and below the temperature range preferred by most workers were associated with a higher incidence of unsafe behaviors and potential accidents. Nunneley and associates[10] took the unusual approach of heating the human head directly and studied the derived effect of elevated head temperature on reaction time and cognitive performance. Shortened reaction times and diminished performance accuracy were observed.

Psychomotor Performance

In general, there seems to be agreement that psychomotor tasks (eg, tracking) quickly become impaired above a temperature of 85°F WBGT. The upper limits of heat exposure for unimpaired psychomotor performance is about 90°F WBGT regardless of the length of time the task is required to be performed—out to 4 hours (Figure 4-3). As is the case with mental tasks, continuous repetitive tasks

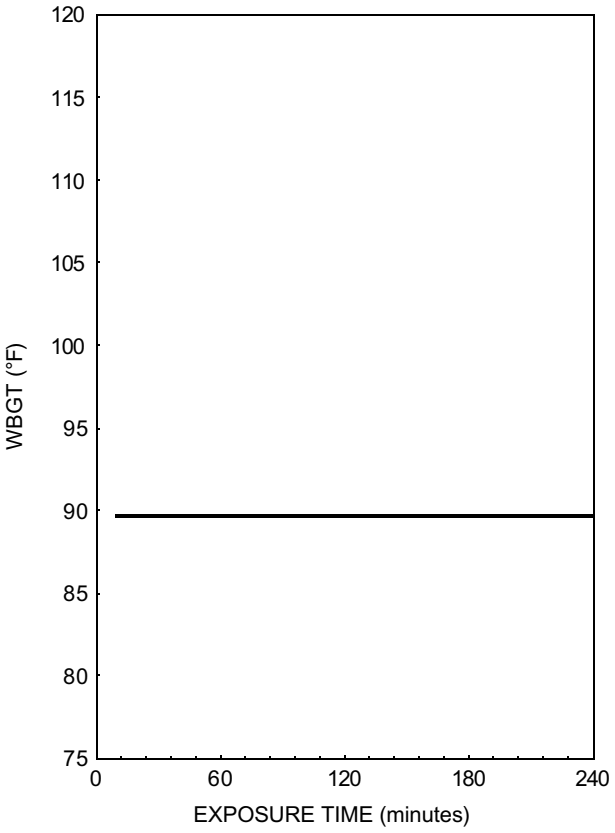

Fig. 4-3. Upper limits of thermal stress (wet bulb globe temperature, WBGT) for unimpaired psychomotor performance, as a function of exposure time.

with relatively low arousal value tend to be affected most. Interesting tasks with high arousal value requiring frequent and varied responses to the environment are less affected.

Dexterity, Steadiness, and Aiming

Military operations require the performance of numerous tasks involving psychomotor components, such as object manipulation, manual dexterity, steadiness, aiming, and rifle marksmanship. Studies that have explored the effects of ambient heat on psychomotor activity have also included the influence of other factors, such as chemical protective clothing, fatigue, and drugs of military interest.

Although heat effects on psychomotor performance have been reported, there are contradictions among the findings. An early study[31] found that ability to align pointers was poorer at ambient temperatures above 90°F, but a later study[6] reported better steadiness at high temperature (126°F). A more recent study by Johnson and Kobrick[32] reported poorer steadiness and rifle marksmanship, but better manual dexterity, at high temperature (95°F, 60% rh). Another study involving high heat and humidity (95°F, 60% rh), chemical protective clothing, and drug antidotes (atropine and 2-PAM Cl [2-pyridine aldoxime methyl chloride, also called pralidoxime chloride])[33] reported impairment of several psychomotor tasks (steadiness in aiming, gross and fine manual dexterity, and gross body coordination in rail-walking) at 95°F, 60% rh, regardless of clothing or drug condition. Fine and Kobrick[28] found that 6 hours of ambient heat (91°F, 61% rh) exposure led to impairments in plotting, arm and hand steadiness, peripheral vision, and rifle-firing accuracy. Thus, the measured effects of ambient heat on psychomotor tasks show a general overall trend of impairment, but the findings have not been uniformly consistent.

Other studies suggest that heat may interact with gender differences and accompanying personal factors, such as level of comfort. Beshir and Ramsey[34] reported that while performing certain perceptual-motor tasks, women were more uncomfortable and reported more fatigue and boredom at higher temperatures than did men. Wyon[35] showed that, regardless of gender, typists did more and better work at lower (66°F, 50% rh) than at higher (75°F, 50% rh) air temperatures. Meese and associates[36] compared the performance of 1,000 factory workers (men and women, black and white) on simulated factory tasks involving psychomotor performance, which were studied at ambient temperatures of 68°F, 79°F, 90°F, and 100°F with "low humidities" for all exposures. Performance at 90°F improved for all groups but white women, and was best for both white and black men. The performance of white women improved at 68°F and 79°F, was worst at 90°F, and improved again at 100°F. These results suggest complex gender and race differences in the effects of heat on psychomotor performance.

Tracking

Tracking typically involves the manual coordination of devices and controls with targets of various kinds, and is a very common military activity. Visual tracking is probably the most common type, although tracking can also involve the use of other senses.

Studies of the effects of ambient heat on tracking have shown decrements in performance and a general cumulative effect of heat with continued exposure. Pepler[20,37–40] showed consistent impair-

ment of a tracking task based on pointer alignment, even when the subjects were heat acclimatized. Two later studies also showed impairment of tracking tasks in the heat. Bell[41] showed impairment of a subsidiary task but not of a primary task performed concurrently. Beshir, El Sabagh, and El Nawawi[42] showed that impaired performance was directly related to task duration. However, Nunneley[43] found that a simple tracking task showed some improvement in the heat, but that two more complex tasks showed no change. Neither Russell[17] nor Grether and associates[4] found any impairment of tracking tasks in the heat.

Based on available data, effects of heat on tracking appear to be interactively related to task complexity, task duration, and operator skill. This position is supported in review articles by Hancock,[44] and Enander and Hygge,[12] and by the findings of Poulton and Edwards.[21,45]

Concurrent Tasks

The effects of heat on performance of two or more tasks performed concurrently have received less attention because such studies are more complicated and, therefore, more difficult to conduct and analyze. Studies indicate that heat generally causes decrements in concurrent task performance of various types. Bell[46] studied the combined performance of a primary pursuit-rotor task and a subsidiary number-processing task under ambient temperatures of 72°F, 84°F, or 95°F (all at 45% rh) and noise levels of either 55 or 95 dB. Performance decrements occurred for both high noise and high temperatures for the subsidiary task, but not for the primary task, and were attributed to overload factors. Mackie and O'Hanlon[47] found that heat-impaired steering caused more errors in a driving task and reduced sensitivity of brightness discrimination. Iampietro and associates[48] found that heat impaired a number of aviation flight tasks. Bell[46] found decrements in one task but no change in performance of other concurrent tasks, the implication being that performance was maintained on a chosen task at the cost of lowered performance on others. Provins and Bell[49] observed that heat led to initial improvement on one task but not on others, followed later by impaired performance. Dean and McGlothlen[50] found no change in concurrent tasks due to heat exposure. Two reviewers have concluded that heat stress generates systematic decrements in mental and cognitive skills, tracking and psychomotor performance, and multitask activities. Hancock[24] has proposed that such decrements are due not only to heat but also to task complexity, and that the decrements are correlated with heat stresses that are physiologically uncompensable, so that deep body temperature continues to rise. Enander and Hygge[12] have taken a similar position but pointed out that empirical research is difficult to interpret because of differences in methodologies and operational definitions of the basic stimuli.

Subjective Response

The perception of bodily sensations associated with heat exposure and the accompanying feelings of discomfort are often considered aspects of the hot environment that have to be tolerated. However, in this section we shall see that subjective responses affect behavior (eg, sleep quality and the nature of social interactions) and that accurate perception of our bodily sensations is important in judging the onset of heat injury.

Symptomatology

Studies have shown that 6 or more hours of ambient heat (91°F–95°F, 60% rh) exposure leads to typical symptomatic reactions[51] such as weakness, "rubbery legs," dizziness, restlessness, hyperirritability, feeling hot and sweaty, loss of appetite, vomiting, and nausea. In addition, heat-stressed individuals tend to be unwilling to drink adequate water and make up for salt depletion. (For further discussion of this "voluntary" dehydration, see Montain's chapter, Hydration, in Volume 3 of *Medical Aspects of Harsh Environments*.)

Salt depletion is a major factor in the development of heat symptoms. During the first several days that military personnel are rapidly deployed to the field, their dietary salt consumption is often reduced because salt content in field rations is altered and total ration consumption is generally reduced.[52] In a jungle, in a desert, or at sea in the tropics, decreased salt consumption becomes particularly problematic because of salt losses associated with sweating.

The minimum salt requirements for acquiring and sustaining heat acclimatization under desert conditions were established in a study using 17 healthy soldiers. After completing 7 days of optimum control conditions, the soldiers underwent 10 days of heat acclimation (106°F, 20% rh) while receiving diets containing low-normal (8 grains [5.2 mg]) or low (4 grains [2.6 mg]) levels of daily salt.[53,54] The data were analyzed for incidence of heat symptoms using a questionnaire-based index of subjec-

tive heat illness.[53,55] The analysis disclosed that regardless of diet group, the predominant symptoms during heat acclimation were warmth, sweatiness, weakness, irritability and restlessness, and rapid heart beat. Dizziness and disturbed coordination also occurred, most often during the first 2 days of heat acclimation. It is clear that greater salt intake significantly reduced reports of subjective heat strain (ie, the physiological change produced by a disturbance) during the first 2 days of heat acclimation but not thereafter. This study assumes paramount importance when we consider that a service member's *perception* of heat illness symptoms is the sole basis for judging the onset of heat injury.[56]

Discomfort

Discomfort due to heat is a familiar experience to everyone. However, the range of reactions varies widely among people in both the kind and the severity of symptoms. Although this is commonly understood, it is difficult to measure and systematically classify the symptoms.

In an attempt to measure subjective reactions to environmental stress, including heat discomfort, Sampson and associates[55] developed a combination inventory and rating scale called the Environmental Symptoms Questionnaire (ESQ). Kobrick and associates[51] employed the ESQ in a study mentioned previously to assess subjective reactions to heat while wearing impermeable chemical protective clothing (mission-oriented protective posture, MOPP 4). The ESQ dramatically reflected the incidence and increase in severity of discomfort reactions in the heat and MOPP 4 condition.

Other approaches have also been used to assess subjective reactions to heat. Beshir and Ramsey[34] compared the reactions of men and women to heat as it was judged to affect their performance of perceptual-motor tasks. The participants rated their thermal reactions, drowsiness, boredom, and fatigue while performing the tasks. The study found that men preferred significantly lower temperatures than women did, but women were more uncomfortable at the temperature extremes. Women reported greater drowsiness and boredom overall, but men became more fatigued than women at higher temperatures.

Another study examined subjective discomfort during the process of recovery from heat stress. Shitzer and associates[57] first exposed men to a standard heat condition, and then allowed them to choose recovery temperatures at will from either (*a*) a personally preferred temperature, which each participant had individually chosen prior to the heat exposure condition, or (*b*) varied schemes starting either nine Fahrenheit degrees above or nine Fahrenheit degrees below the preferred temperature. The recovery conditions with the highest overall temperature were judged the most comfortable, implying that subsequent comfort reactions are biased by previous exposure conditions.

Price and Hennigan[58] asked each of 24 undergraduate men to estimate the immediate temperature of the heat chamber in which he sat. The researchers found that temperature estimates were closer to the dry bulb air temperature than to either the WBGT or a temperature–humidity index. The participants' everyday experience with dry bulb air temperature as an index of heat was concluded to be the dominant factor in temperature estimation.

In summary, these findings reflect the highly subjective quality of personal discomfort reactions and show that experience is a strong factor in the kinds of discomfort reactions that occur. In effect, previous heat experience can influence later heat tolerability. It also seems clear that people judge discomfort on the basis of familiar indices, primarily dry bulb temperature, which is the most commonly cited everyday index.

Changes in Sleep Behavior

Another common belief is that heat disrupts normal sleep. Restlessness due to discomfort is a familiar reaction at night during a heat wave, and there is considerable research evidence[59–61] to indicate that hot conditions do seriously disrupt ordinary restful sleep.

Libert and associates[62] studied the effects of continued heat exposure (5 days and nights at 95°F) on actual sleep during that period using unacclimatized participants. The time distribution of sleep stages and recovery effects at subsequent normal temperatures were examined. It was found that effectiveness and quality of sleep were generally reduced at high temperature; also, there were no indications of improvement in sleep with continuing heat exposure, despite increased physiological heat adaptation. Libert and associates[63] then examined the effects of heat exposure and hot sleeping conditions combined with traffic noise on sleep activity. Heat was found to be more disruptive to sleep than was noise. Sleep time decreased in the heat, the pattern of sleep stages was disrupted, and time awake increased. In general, the available findings indicate that attempting to sleep in the heat is not only unpleasant, it also affects sleep patterns.

Social Effects

It is a common belief that motivation and activity levels tend to be lower in hot and hot–humid regions, whereas the opposite is believed to be true for aggression. A major problem in studying the influence of climate on social groups is that the effects of heat on behavior are interrelated with other potential causal factors, such as social influence; differences in individual reactivity to heat; crowded living conditions associated with hot, urban areas; cultural beliefs and practices; and seasonal climatic changes.

In a study by Bell and associates,[8] 64 men and 64 women undergraduates performed an easy or a complex reaction time task, either alone or in pairs (coaction), while exposed to ambient temperatures of either 72°F, 46% rh; or 98°F, 43% rh. Reaction time was faster for men than for women, faster for the easy than for the complex task, and faster for coacting than for individually acting participants. Heat increased reaction time, although its effect was statistically "weak." All of these factors were essentially additive, with task difficulty having the strongest effect.

Bell and Doyle[64] studied the effects of heat and noise on helping behavior. Ninety-six undergraduates were given an opportunity to aid a confederate both before and after exposure to ambient temperatures of either 73°F, 46% rh; or 95°F, 46% rh; and to noise levels of 55 dB, 95 dB without perceived control, or 95 dB with perceived control. Although heat produced discomfort and led to negative evaluations of the environment, and noise generally increased response to stimuli (arousal), neither heat nor noise influenced any measures of helping behavior.

Megargee[65] addressed concerns about the influence of overcrowding, cramped space, and uncomfortable temperatures in prisons on inmate misconduct. The relation of population size, amount of personal space, and population density were compared with the incidence of disruptive behavior over a 3-year period in a correctional institution for young male offenders. Population size was significantly correlated with the number but not the rate of disciplinary violations. The total amount of personal space and the index of population density were significantly correlated with both the number and the rate of violations. A post hoc analysis showed that the incidence of uncomfortably hot days had no relation to disruptive behavior.

On the other hand, it is well documented that aggression toward neighbors and family members increases in crowded urban areas during summer heat waves.[66,67] Although these data have not gone unchallenged, it is clear that heat increases the likelihood of heightened anger and irritability, which in turn, leads to quarreling among neighbors, family members, and coworkers. Analysis of the effects of hot living conditions on overt social behavior is blocked by interactions of heat with crowding, urban problems, and socioeconomic factors.

UNDERLYING MECHANISMS

To explain the effects of the thermal environment on psychological performance, several underlying mechanisms have been proposed. We consider three here: the influence of heightened physiological arousal accompanying heat exposure, the influence of a stable (static) versus a changing (dynamic) deep body temperature, and the influence of emotional reactions to heat exposure.

Arousal

In emergency situations, an organism's responsiveness to external and internal stimuli is heightened. Emotions such as fear and anger motivate us to act. Arousal reactions are accompanied by measurable changes such as an activated electroencephalogram (EEG), increased heart rate, and elevated blood pressure. One theory to explain changes in performance that accompany heat stress centers on an individual's arousal level.[68,69] In this theory, exposure to ambient heat is considered a stressor that increases arousal, and the relationship between arousal and performance is seen as an inverted U function (Figure 4-4). Too little or too much arousal may impair performance. The theory states that (1) psychological performance improves as arousal increases above a comfortable resting level (as can be caused by mild heat), and (2) performance degrades when arousal either drops below or rises above this optimal level. Presumably, performance is best when one is fully awake, poorer when one is less than fully awake (just waking up or falling asleep), and poorest (or nonexistent) when one is either asleep or hyperalert. Hyperalertness occurs during panic situations such as those associated with extreme danger or extreme pressure (eg, imminent or actual combat). For poorly learned tasks, performance is best when arousal is low. A beginning marksman, for example, will likely fire more accurately when in a quiet area (low arousal) than

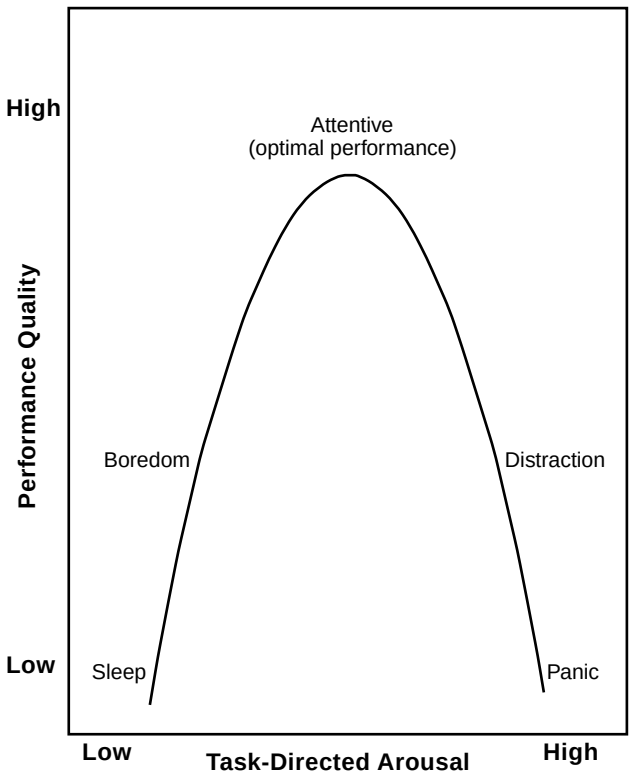

Fig. 4-4. The arousal–performance function for a moderately difficult task.

in an area where an instructor is yelling (high arousal). On the other hand, an excellent marksman is less likely to fire poorly under high arousal conditions. This relationship between performance, arousal, and task difficulty is known as the Yerkes-Dodson Law[70] and predicts that for highly skilled (trained) individuals, performance deteriorates less rapidly as arousal increases.

If ambient heat is intense enough, it may be perceived as dangerous to health, high arousal will occur, and performance will likely be extremely poor. Grether[71] has suggested that performance is optimal when ambient temperature is 80°F ET, or just above the thermal comfort level. Grether's selection of 80°F ET as the optimal level for psychological performance was based on studies of vigilance and of Morse code operation during ambient heat exposure.[20,31]

Static Versus Dynamic Deep Body Temperature

In a review of the effects of the thermal environment on vigilance, Hancock[1] has argued that the data in support of the arousal theory interpretation actually contradict the notion that performance is facilitated in ambient temperatures that exceed a comfortable level. Rather, Hancock argues that performance is degraded as the thermal homeostasis of the individual is disturbed; that is, deep body temperature becomes dynamic. Hancock points out that in the original experiments of Mackworth[31] and of Pepler,[20] two variables—ambient temperature and the "thermal state" of the individual—were confounded. Specifically, in Mackworth's study, the optimal ambient condition, 79°F ET, happened to be the only ambient condition selected for study that did not cause a change in deep body temperature of the individuals being tested; that is, deep body temperature was static. On the other hand, both of Mackworth's heat stress conditions, 87.5°F and 97°F ET, were above the ambient temperature level, 85°F ET, which results in heat stresses that are physiologically uncompensable, so that deep body temperature continues to rise. Hancock argues that a significant breakdown in performance efficiency does not necessarily occur when the ambient temperature is above a certain level, but occurs when deep body temperature is disturbed or changing; in other words, when an individual's deep body temperature is in a dynamic as opposed to a static state.

If Hancock is correct, then performance should remain unaffected as long as deep body temperature is stabilized and is neither rising nor falling. In a study of combinations of ambient heat (up to 93°F ET) and ambient noise (110 dB), Dean and McGlothen[50] found no change in vigilance performance during a 30-minute exposure to heat and noise. These data support Hancock's contention that it is not the actual ambient temperature that affects performance, but it is a combination of exposure time and ambient temperature sufficient to change deep body temperature that will lead to degradations in performance. Although the ambient condition was high enough to produce an uncompensable rise in deep body temperature, the 30-minute exposure was not long enough to induce a measurable change in body temperature.

Improvement in psychological performance as a result of thermal stress has been found when the individual has been stabilized in a static hyperthermic state. For example, Wilkinson and associates[72] assessed auditory vigilance in subjects who had been stabilized at either 1.4, 2.5, or 3.6 Fahrenheit degrees above preexposure deep body temperature. This was accomplished by first exposing them to 109.4°F and 100% rh until their body temperature reached one of the target levels. They were then removed from the heat chamber; put into a heated, impermeable suit to maintain body temperature; and tested on

the vigilance performance task. At body temperatures more than 1.4 Fahrenheit degrees above preexisting levels, vigilance performance improved as measured by speed of response to targets and correctness of target identification. Hancock points out that the difference between this study and the others was the establishment of a static hyperthermic state that lacked the stress associated with the constant rise in body temperature. Thus, the data are in line with the notion that performance degradations resulting from heat exposure are associated with dynamic deep body temperature and not with static deep body temperature.

Emotional Arousal and Experience

Stressful situations, including heat stress, tend to elicit emotional responses that were linked to stressful situations in the past. In high-stress situations, the emotional response (activated EEG, rapid heart rate, elevated blood pressure) is, at a minimum, distracting, and at most, incapacitating (panic reaction). These emotional responses have become associated with stress and now become the dominant responses during stress. Behaviors associated with calmer times are less likely to be remembered and performed. If an appropriate adaptive response is not well practiced, it is extremely difficult to remember it and perform it well while under stress. Research has shown that if a response is practiced so that it becomes automatic, it has a better chance of being expressed during crises.[73] Because military personnel are required to respond quickly to changing events during stressful situations, such as those that exist on the battlefield or during heat stress, when arousal is sure to be high, military tasks have to be overlearned if they are to be proficiently performed when needed most. For these reasons, all military tasks, from the most basic to the most complex, need to be practiced during times that are not stressful as well as during times of high stress if they are to have a chance to become the appropriate first response (or at least a not-too-belated second response) in the repertoire of emotional responses. Using data from validated heat stress situations,[20,31,73] we have constructed Figure 4-5, which illustrates how well tasks are likely to be performed under varying levels of ambient heat. Performance decrements are likely to be minimal when skill level is high, and considerable when skill level is medium or low.

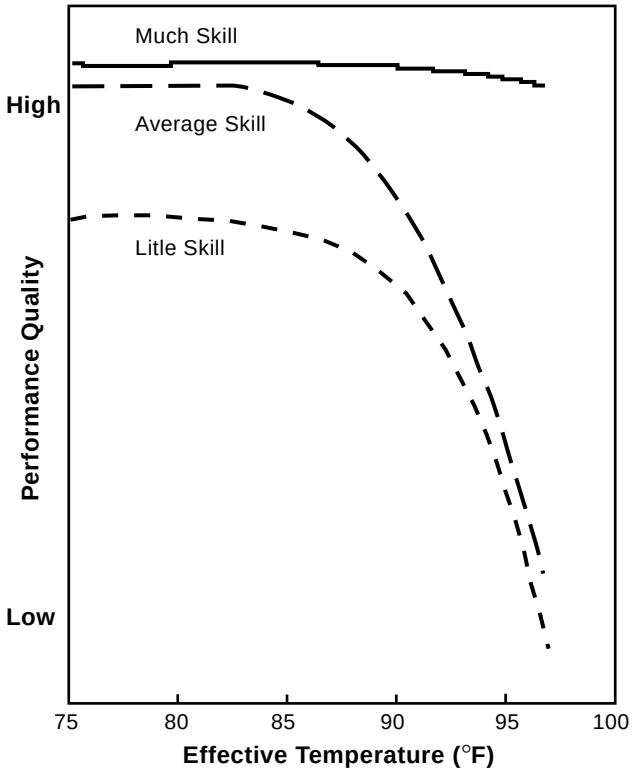

Fig. 4-5. The performance–heat exposure function for different skill or training levels.

CHANGES IN PERFORMANCE UNIQUE TO THE MILITARY

Some aspects of the military situation uniquely influence psychological performance. Uniforms and equipment, for example, which are designed to protect the soldier from both chemical contamination and ballistic fragments, often impose an added heat load due to impairments in normal evaporative cooling. Medications designed to protect a soldier from nerve agents often impair thermoregulation by inhibiting sweat production. Acclimatization and appropriate training in hot environments may help to attenuate limitations imposed by clothing, equipment, and medications.

Performance as a Function of Clothing and Equipment

Military clothing and equipment are inherently involved in ambient heat exposure because, on the one hand, shelters, vehicles, and clothing provide environmental protection (eg, shade, wind protec-

tion, and thermal insulation). On the other hand, shelters and vehicles trap heat and retard ventilation, and clothing, especially impermeable encapsulating ensembles, traps body moisture. Also, evaporative cooling is hampered by other devices such as load-carrying systems, which block moisture release.

Most of the research on heat stress involving clothing and personal equipment systems has directly examined the effects on operator performance while wearing various clothing and equipment items and systems in the heat. Major attention has been directed to problems due to wearing chemical protective ensembles, particularly MOPP 4, which, by necessity, is totally encapsulating and impermeable. Kobrick and associates[51,74] compared performance over a target period of 6 hours on a variety of psychomotor, cognitive, visual, and locomotor tasks; and subjective reactions, while subjects wore the MOPP 4 ensemble and the standard military battle dress uniform (BDU) under hot–humid (91°F, 35% rh) and temperate (70°F, 30% rh) conditions. All participants completed the 6-hour sessions while wearing the BDU ensemble, and showed only minimal performance impairment and few subjective reactions. In contrast, the MOPP 4 heat sessions could not be continued beyond 2 hours, and all tasks were significantly impaired. Subjective reactions were numerous and severe. Kobrick and associates[15] also measured the effects of the same test conditions on several indices of visual performance (acuity, phoria, stereopsis, contrast sensitivity). All indices showed significant impairment under continued heat exposure. Kobrick and Sleeper[16] also studied the effects of the same test conditions on sensitivity for detecting signals distributed at various locations throughout the visual field, and found significant systematic increases in response time to all signals.

Fine and Kobrick[28] examined the effects of similar heat conditions on sustained cognitive performance while wearing MOPP 4 chemical protective clothing. Cognitive performance began to deteriorate markedly after 4 to 5 hours in the heat. By the end of 7 hours of exposure to heat, increases in the percentage of group error on investigator-paced tasks ranged from 17% to 23% over control conditions. Most of the decrements were due to increases in errors of omission. The productivity of the group on a self-paced task (map plotting) diminished by approximately 40% from control conditions after 6 hours in the heat in protective clothing; accuracy of plotting was not markedly affected.

Carter and Cammermeyer[75] observed the task performance of 195 medical unit personnel participating in a 3-day simulated chemical warfare field training exercise under very hot conditions. A heat injury checklist was used to assess physiological parameters of heat stress. Participants performed under simulated scenarios: (1) conventional warfare, (2) chemical casualties, and (3) hostile and chemical attack. Participants in the second scenario appeared to experience the most stress. Participants became increasingly disorganized in the third scenario. Five real casualties emerged as a result of heavy activity, medical problems, limited food and fluid intake, overweight, or prior difficulty wearing gear.

In a similar type of study approach, White and associates[76] examined physiological and subjective responses of nine men (aged 21–33 y) who worked while wearing two types of protective ensembles (chemical protective clothing and scuba gear) in each of three thermal environments. Even at a low work intensity, individuals who wore chemical protective clothing in the heat required progressively shorter work periods and more frequent and longer rest periods. Subjective responses indicated that the participants perceived the chemical protective clothing as less acceptable than the scuba gear.

Arad and associates[77] investigated the effects of pyridostigmine pretreatment; nuclear, biological, and chemical (NBC) protective gear; and heat-exercise exposure on psychomotor performance and subjective sensations in eight healthy, heat-acclimatized men. Exercise in heat enhanced the performance of vertical addition but prolonged reaction time. The effects of pyridostigmine, protective gear, and the interactions between the various stressors were not significant. Cognitive performance was not dependent on body core temperature. Multiple complaints of subjective discomfort arose from wearing the protective garment. Results suggest the existence of significant subjective discomfort but absence of major cognitive decrements.

These studies indicate that heat stress generated by wearing clothing and equipment systems adds to that created by the same ambient environment alone. These effects cause impairment of a wide range of tasks involving sensory, psychomotor, cognitive, and subjective aspects of performance. It should also be noted that there may be additional hampering effects caused by the physical characteristics of the equipment itself (eg, visually restricting face masks, glove liners that ball up when wet with perspiration, restriction of manual dexterity by stiff and thick glove material, and hearing restriction due to encapsulating headgear).

Johnson and Kobrick[78] evaluated the findings of numerous studies on the effects of wearing chemical protective clothing on rifle marksmanship and on selected sensory and psychomotor tasks. Task performance in the BDU under thermoneutral conditions was used as a standard for comparison based on the percentage of change to evaluate the separate and combined effects of wearing chemical protective clothing, exposure to ambient heat, and test duration. The findings indicated that wearing chemical protective clothing resulted in an early overall impairment of task performance (less than 2 h), but that the magnitudes of impairment did not increase progressively over time (up to 6 h) beyond the initial impairment levels. Although wearing chemical protective clothing in hot conditions led to the development of heat stress (and thus limited test time to less than 2 h), it did not degrade sensory or psychomotor performance beyond that observed under thermoneutral conditions for the same period. Wearing chemical protective clothing during heat exposure did, however, degrade rifle firing accuracy during the first 2 hours (mainly due, however, to the poor compatibility of the face mask with the rifle, which caused poor alignment of the eye with the sights and the target). Tasks involved in simulated sentry duty showed that wearing chemical protective clothing intensified vigilance decrements and degraded rifle marksmanship. (For further information, interested readers can consult Taylor and Orlansky.[79])

Influence of Operational Requirements: Medications

The effects of drugs and other chemical substances on military performance during heat exposure are of high interest to military unit commanders. Available studies have explored effects of substances either of some pragmatic interest, or of a known relationship to some heat-mediating body function.

Kobrick and associates[15,51,74] conducted a multifactorial investigation of the effects of a standard US Army nerve agent antidote (2 mg atropine combined with 600 mg 2-PAM Cl vs a placebo) and heat exposure (95°F, 60% rh vs 70°F, 30% rh) on repeated performance of militarily relevant psychological tasks while wearing the BDU and while wearing chemical protective clothing (MOPP 4). Twenty-three men, aged 18–23 years, volunteered. All BDU sessions (6 h in duration) were completed, but with some task impairments and a few subjective discomfort reactions. MOPP 4 heat sessions could not be continued beyond 2 hours; all tasks were impaired, and significant subjective reactions were numerous and severe. The combination of atropine and 2-PAM Cl significantly shortened endurance time for heat sessions in MOPP 4. If we assume that heat exposure combined with wearing MOPP 4 chemical protective clothing constitutes a very severe form of heat experience, these investigations indicate a sizable drug-related impairment to several types of behavior, which increases with heat exposure.

In another study, Arad and associates[77] examined effects of heat exposure combined with pyridostigmine, a nerve agent pretreatment, on varieties of psychomotor performance and subjective reactions. Eight healthy, heat-acclimatized men were tested on addition and reaction time tasks while they exercised in the heat. Performance of addition was enhanced, but reaction time increased. The effects of pyridostigmine, protective gear, and the interactions between the various stressors were not significant. Cognitive performance was not dependent on core body temperature. Multiple complaints of subjective discomfort arose from wearing the protective garment. Results suggest the existence of significant subjective discomfort but an absence of major cognitive decrements in a multiple-stress state of chemical warfare alertness.

One study examined the effects of substances believed to be related to physiological heat-regulation mechanisms. Bakharev[80] studied the action of the synthetic posterior pituitary hormone, arginine vasopressin (AVP), a neuromodulator of long-term memory, on human tolerance to the short but intensive action of a hot–dry environment, using eight participants who were 29 through 35 years old. No direct effect of AVP on the dynamics of the heat balance of the body could be found. However, AVP improved participants' subjective sensations with respect to most parameters studied.

Acclimatization and Training

The beneficial influence of heat acclimatization on subsequent physical performance in the heat is well documented. It is also generally understood that heat acclimatization is best achieved by exercise during continued heat exposure. However, the available information on heat acclimatization effects pertains mainly to physical activity and allied physiological functions; related factors involving training in the heat also primarily address physical performance. In contrast, knowledge of the effects of heat acclimatization and training on psychologi-

cal and cognitive performance is much more limited and, in fact, considerably speculative.

Mackworth[31] studied the decoding of Morse code messages by military personnel of varying skill and training levels at ambient heat levels of 79°F, 83°F, 87.5°F, 92°F, and 97°F ET. He found that personnel who were highly skilled in decoding were least affected by the heat, showing fewer than 10 errors per hour regardless of the ambient heat level to which they were exposed. Those with medium and minimal skill levels showed their usual error rates (< 10 and < 40 errors per hour) at 79°F and 83°F; at 92°F, they began to show increased errors over the lower temperature conditions; and at 97°F both groups showed dramatic impairments in performance (80 and 90 errors per hour, respectively). Skill and training levels are obviously an important factor in determining the extent to which performance is impaired by ambient heat exposure.

Razmjou and Kjellberg[9] studied the effects of heat (104°F dry bulb, 77°F wet bulb) on sustained attention and serial response in a simple reaction time task and a serial four-choice reaction time task. They found that in unacclimatized subjects, simple reaction time performance as well as accuracy in the serial choice reaction time task deteriorated significantly in heat. In a study of performance during heat acclimatization, Shvartz and associates[7] found that simple reaction times to a visual stimulus obtained over 10 consecutive days during exercise in heat decreased, so that they were only 10% higher than resting reaction times on the last heat stress day, and were the same as resting reaction times on the last (room temperature) day. In other studies, Curley and Hawkins[29] and Curley and associates[81] assessed cognitive performance during a 10-day heat-acclimatization regimen using repeated acquisition tasks. On the first day of exposure, performance on the repeated acquisition tasks yielded slight decrements compared with preacclimatization baseline values recorded at moderate temperatures. By the 10th day of heat exposure, all participants demonstrated significant heat acclimatization. However, mean performance on the repeated acquisition task was still impaired. Results suggest that tasks requiring the acquisition of new behaviors may be difficult to perform in a hot environment, even by partially acclimatized individuals. Thus, the available studies have produced somewhat conflicting results, which have not dealt experimentally with the influence of training level and skill of the participants. In a review article, Hancock[82] reflected this concern, noting that individuals who are skillful at a task may be better able to withstand the detrimental effect of the stress exposure than their unskilled counterparts.

The potential effects of experience with heat exposure are considered in three other studies of effects of heat adaptation on perceptual reactions. Khudaiberdiev and associates[83,84] and Khudaiberdiev and Pokormyakha[85] used psychophysical scaling to assess temperature judgments of individuals during summer and winter seasons, and found sensitivity to smaller temperature increments in summer than in winter. They concluded that temperature judgment sensitivity becomes reorganized during prolonged exposure to higher seasonal temperature; however, the authors did not propose a causal mechanism for the change in sensitivity. Two reviews from a similar aspect consider the heat adaptation effects of sauna bathing on perception and performance[86,87] and note that the psychological effects of the sauna have rarely been studied. Nevertheless, they speculate that users undergo (1) autonomic changes and build up resistance to the temperature extremes, as well as (2) subjective adaptation to the experience. Sulanov and Freink[88] argue that to adapt to arid heat, humans adopt an avoidance strategy that incorporates specific work and rest patterns, use of shade, air conditioning, and clothing that screens the body from the sun's radiation.

MILITARY APPLICATIONS AND CONSIDERATIONS

We now have considerable knowledge about human reactions to heat exposure and can make some useful recommendations about military performance in the heat, based on available research findings and our own practical experience. This section is intended to provide concise recommendations for dealing with heat effects (Exhibit 4-1). The guidelines are organized according to the behavior categories that we have used above, and in the context of military operations wherever possible.

Mental Performance

In the military, mental performance includes the performance of those tasks that are mainly intellectual, from the most basic decision (whether or not to react to the sudden appearance of a target), to

EXHIBIT 4-1

GUIDELINES: PSYCHOLOGICAL EFFECTS OF HEAT ON MILITARY PERFORMANCE

These useful conclusions are intended to help military personnel address psychological effects on performance of military tasks due to heat exposure:

1. Simple reactions can be performed as quickly in the heat as under normal conditions. More complicated reactions involving compound actions or some form of judgmental choice will take longer and show more variability as heat increases.
2. Although vision is not directly affected by heat, seeing clearly is impaired by secondary factors such as sweat running into the eyes and moisture obscuring optics and lens surfaces.
3. Visual distortions due to heat, such as mirages, optical illusions, shimmer and glare, reduce accuracy in performing visual tasks such as rifle aiming and distance judgment.
4. Equipment controls properly designed for use under optimum conditions can interfere with efficient manual operation when they become too hot to handle comfortably.
5. Sweating causes headgear and headphones to become unstable and slide on the head, interfering with hearing, vision, and the performance of other tasks.
6. Tasks requiring sustained attention, such as sentry duty, watch keeping, and instrument monitoring, usually deteriorate after 90 minutes, even under normal conditions. Heat exposure will shorten effective performance time to about 30 minutes. However, attention losses can be significantly counteracted by using various forms of caffeine and by taking intermittent breaks to reduce boredom.
7. Complex mental tasks, such as mathematical reasoning and decoding of messages, deteriorate in heat above 90°F after about 3 hours. Acclimatization to the heat and training in the heat reduce such decrements by increasing tolerable heat level and extending endurance time.
8. Protective handwear worn in the heat can cause increased sweating, as well as swelling of the hands themselves. These lead to slipperiness of gloves on the hands, bunching up of glove liners, and impairment of dexterity and tactility.
9. Continuing heat exposure causes progressive motor instability, leading to impaired steadiness and reduced rifle firing accuracy due to increased variability.
10. Target tracking, in which the operator must judge differences in continuous target alignment, deteriorates under hot conditions.
11. Simple tasks are less affected by heat than are highly complex tasks. Moderately complex tasks tend to be the most resistant to heat effects because they tend to sustain attention while placing only moderate demands on the operator's overall performance.
12. Multiple tasks (ie, two or more tasks being performed concurrently) are more affected by heat than any of the same tasks performed individually.
13. Discomfort reactions are widely different among individuals, and heat acclimatization and experience greatly influence degrees of discomfort. People are typically most uncomfortable and most distracted from their military tasks during conditions of heat and high humidity.
14. Symptoms of heat illness seriously degrade soldier performance, and symptom intensity varies widely among individuals. Military personnel should not be returned to duty prematurely because residual effects, although not directly obvious, can still affect military performance.
15. Crowded conditions commonly aggravate the effects of heat exposure, leading to increased aggression and irritability among groups in settings such as confined crew compartments and crowded troop transports.

(**Exhibit 4- 1** *continues*)

Exhibit 4-1 *continued*

16. Typical disruptions of sleep patterns during military operations are further aggravated by hot conditions. The resulting sleep deprivation leads to difficulty in concentrating, thinking clearly, and reacting quickly and appropriately.
17. Although chemical protective clothing temporarily shields the body from direct (mainly radiant) heat, it also creates a microenvironment around the body that inhibits dissipation of heat and humidity generated by the wearer. In effect, a servicemember wearing chemical protective clothing in the desert may really be suffering instead from the effects of wet, jungle-like heat conditions. Mandatory requirements for encapsulation unavoidably intensify heat loads.
18. Wearing chemical protective clothing in the heat can be expected to cause almost immediate moderate impairment in military performance but with no additional significant increase for up to 6 hours thereafter. The decrements apparently are due mainly to the physical encumbrance of the clothing itself rather than to the heat effects per se.
19. The main effects of the nerve agent antidote, atropine, are slowed reaction time and photophobia. At the same time, heat causes reduced steadiness. These separate effects of drug and heat combine to impair tasks such as rifle firing, which require motor stability, quick reaction, and clear vision under bright conditions.
20. Commanders should take every opportunity to train their troops under hot conditions to avoid the disrupting effects of experiencing heat for the first time in the field. In other words, tasks to be performed in the heat should be learned and practiced in the heat. The beneficial effects of heat acclimatization and training can act to reduce the performance impairments, which would otherwise occur without such heat experience.

the maintenance of vigilance through determination (sentry duty), to the complex (map plotting, decoding of messages).

Reaction Time

During brief heat exposure, tasks involving simple reactions, such as straightforward target detection, are largely unaffected. In fact, reaction time in general seems to be quite durable during heat exposure; even choice reaction time is relatively unaffected. The severity of heat (dry bulb temperature ≥ 120°F) seems to be the driving factor rather than the duration of exposure. Nevertheless, complicated reactions can be expected to deteriorate sooner than simpler ones during heat exposure.

Despite the length of heat exposure, simple military tasks such as the operation of toggles will be performed with minimal delay. However, when choices are required, such as discriminating friend from foe, some lag will occur, but not to an important degree. This general relationship will hold throughout the tolerable temperature range, but deterioration will increase as temperature rises to the point of heat incapacitation. Personnel will also become more variable in their reaction speeds at higher temperatures and when performing more complicated tasks. Heat acclimatization, however, will still act to mitigate these heat effects.

Sensation

Vision is clearly the most important human sense because it provides the most information for guiding overall behavior. Therefore, factors that affect vision, either directly or indirectly, are crucial considerations for efficient military performance. The available data seem to indicate that heat does not affect the visual process directly. However, heat exposure can indirectly interfere with normal visual function by causing disruptive conditions, such as condensation collecting on eyeglass or mask lenses, and sweat that runs into the eyes and causes headgear and headphones to become unstable and slide around on the head.[89]

Service members can be expected to have visual problems whenever severe sweating occurs. Sweat that runs into the eyes is especially troublesome when encapsulating clothing is worn because the sweat cannot be wiped away without breaking the seal of the protective mask. Collection of moisture on the eyepieces of the mask is a further problem because it

obscures the visual field. We have seen soldiers dressed in the MOPP 4 ensemble trying to look through trickle patterns in the condensed moisture on the eye ports—their only means of seeing clearly. Whether or not encapsulating clothing is involved, sweat in the eyes can cause excessive tearing, leading to reduced acuity. Thus, even though heat exposure does not directly affect vision, it can produce other major problems that lead to impaired sight.

Heat also can interfere with other normal sensory actions and, as a result, can cause sensations that are quite different from those experienced under cool conditions. Such effects are often mistakenly judged to be artifacts, but can be the immediate cause of performance decrements. For example, hot environments can cause mirages, visual distortions, and optical illusions due to atmospheric shimmer and glare, all of which result in reduced or inaccurate performance of visual tasks. Because these phenomena are common, it is strongly recommended that military personnel be given direct experience with them, by being trained in hot conditions where they occur. Similarly, it is important that the training be conducted in an environment most like the actual operational conditions (eg, on terrain similar to what will be encountered during real operations) so that personnel will become familiar with the possible visual effects.

Vigilance

Whether on land, in the air, or at sea, military operations invariably require continual surveillance and attention to current conditions. Therefore, vigilance and alertness are critical military factors, and the effects of heat on them are of serious concern. In tasks involving sustained attention, alertness, or both (eg, sentry duty, vehicle operation, instrument monitoring), vision appears to be the main process affected by heat. However, there is evidence that auditory vigilance tasks are also affected by heat. Interpretation of the findings is complicated by both visual and auditory vigilance studies' having been based on several different temperature indices (eg, dry bulb, WBGT, ET). Nevertheless, deterioration of vigilance seems to begin at around 85°F and higher.

Various studies using watch-keeping tasks have shown that aspects of vigilance significantly deteriorate after 90 to 120 minutes of sustained performance. Heat exposure likely will further shorten effective performance time, but this also will depend on the demand level of the task. Low-demand, boring tasks, such as watch keeping, with little challenge to the operator, can be expected to be the most vulnerable to heat effects, and the reduction in effective performance time can be as much as 60 to 90 minutes.[79] This means that the reliability of watch keeping in the heat will drop after 30 minutes, and that personnel should be relieved as often as possible to maintain effective surveillance. Vigilance can also be maintained fairly effectively by using caffeine in the form of coffee, tea, cola, chocolate, and even over-the-counter stimulants, usually in dosages of 200 mg. Familiar experience suggests that brief interruptions or breaks such as getting "a breath of fresh air," stretching, and walking around seem to help reduce boredom. There are also some data showing that experience, training, and motivation are positive factors in reducing the negative effects of heat on vigilance.

Complex Mental Performance

Virtually every military task requires service members to be able to process information rapidly and accurately, and to remember and use this information effectively. Therefore, the effects of heat on the complex mental processes involved are important considerations. Heat has been shown to affect short-term memory, mathematical reasoning, map plotting, and the coding or decoding of messages, typically after 3 hours at or above 90°F.[27,28,32,33] Acclimatization can be an effective means of reducing the decrements that develop, both by increasing the tolerable heat load and by extending the length of endurable heat exposure. In like manner, training tends to offset the effect of heat by reducing errors and serving to maintain the quality of performance for longer periods.

Every effort should be made to acclimatize military personnel to the prospective temperature conditions they may encounter, and to provide as much training as possible in the specific operational tasks required to accomplish a mission. Overtraining is better in all respects than merely adequate training to accomplish the task in temperate conditions. Even better is training conducted in the actual heat conditions that are expected to be encountered in the field.

Psychomotor Performance

Psychomotor performance includes those tasks that are largely manual. It includes manual dexterity (with and without protective handwear), steadiness, aiming, and visual tracking (the firing of small

arms requires proficiency in this area). Complex tasks often require the concurrent performance of two or more psychomotor tasks, such as in the operation of vehicles, tanks, and helicopters.

Dexterity, Steadiness, and Aiming

Modern mechanized warfare involves many manual operations, such as manipulation of controls, assembly of weapons, and mechanical maintenance. Therefore, the effects of heat exposure on dexterity and psychomotor performance can seriously influence military operations. As with vigilance and perceptual performance, heat levels of 90°F to 95°F in laboratory studies have shown impairments that warrant serious concern for practical operations. High heat levels also raise the surface temperature of devices that personnel operate, rendering them uncomfortable, or even painful, to handle.[90] Many controls that are well-designed for operation under cool conditions cannot be operated when they heat up in the sun, and can even cause thermal burns to the skin.[90] Thus, heat by itself can block the performance of many operational tasks for physical reasons that are entirely unrelated to the physiological effects of heat.

Although gloves will protect the hands from contact burns, they create other problems that result from overheating the hands. The main problem is sweat that is generated and then trapped inside the gloves. The result is slipperiness between the hands and gloves, causing the gloves to slide on the hands, and at the same time causing sensory impairment because of the interference of the glove material. Glove liners invariably become soaked, leading to their bunching up at finger joints, slipping down the hands, or wadding up in clumps.

Finally, hands and feet often become edematous, causing gloves to fit too tightly. This, in turn, leads to reduced joint motility and reduced manual dexterity due to stiffness.

External ambient temperatures of 90°F to 95°F translate to much higher levels inside cramped spaces such as closed metal crew compartments and in encapsulating uniforms. Whenever heat entrapment occurs, heat exposure and its consequent effects can be expected to be much more severe than might otherwise be the case.

As shown in laboratory studies, another issue for serious concern is the impairment of steadiness caused by ambient heat. Unsteadiness can influence many military tasks, rifle marksmanship among others. Such effects lead to loss of accuracy and increased variability of rifle firing accuracy. Applied to military units rather than just one marksman, such effects could be devastating for the outcome of military combat operations.

Tracking

Military tasks involving alignment of weapons or control systems to track moving target positions (eg, machine gun firing; control of tube-launched, optically tracked, wire-guided [TOW] weapons), manipulation of joy-stick controls, and even complex tasks such as piloting aircraft, have been shown to deteriorate in the heat (≥ 90°F). Tracking is a complex skill involving complex coordination, steadiness, and focused attention. Impaired tracking is directly related to reduced steadiness, which also deteriorates under hot conditions, as noted previously. This causes interference with the ability to track targets reliably, particularly in compensatory tracking, when an operator must continuously make up the difference between control position and target position. Beyond the direct expedient of simply keeping the operator cool, some compensation for heat effects may be obtainable by building in more reluctance into control systems (ie, making controls stiffer to operate, thus tending to cancel out deviations due to the operator's unsteadiness and tremor). However, in the field, commanders will still have to cope with the effects of very hot conditions like those encountered in the desert, which will seriously influence the performance of crucial tasks such as operating guidance systems in fully closed tanks, where interior heat levels become nearly unendurable. Although there has been progress in the development of air-conditioned vehicles and practical, microclimate-controlled clothing, older equipment still will be used in combat. As a result, commanders must understand and make allowances for increased variability and unreliability of troop performance in the heat.

Concurrent Tasks

Many military tasks are routinely performed individually in some situations, but at other times may be performed with other tasks. The effects of heat on task performance may be quite different for the component tasks when performed singly or in combination. We must therefore consider the effects of heat on task interactions. The main factors that influence performance where these interactions are involved seem to be the complexity level of each individual task, the combined complexity of the combination of tasks, the skill and experience of the

operator, and the heat load accompanying the situation. Tasks that are performed concurrently are usually prioritized by the operator based on the demands of the mission, the requirements of the separate tasks, and the environmental conditions. It is important to understand the basic principle in such task interactions: that performance of individual tasks in the heat is usually best under moderate levels of heat load and task demand. Conversely, poorest performance will occur with highly complex task combinations under very hot conditions, although degraded performance will be somewhat offset by the motivation of the operator and the perceived importance of the mission. Commanders may be able to preserve the overall efficiency of their units in hot weather conditions by delegating task assignments so that the stress load is shared by all.

Subjective Response

A commander's awareness of the subjective response of the soldier is important to proficient accomplishment of the mission. Discomfort and distraction owing to heat symptoms can be a serious matter for the soldier and impair the completion of minor tasks. Discomfort may also lead to poor unit cohesion when social interactions become strained.

Symptomatology

In addition to physiological reactions to heat, there are characteristic symptomatic reactions that can seriously affect capability. In practical terms, a service member who feels ill is less apt to be an effective warrior. Reactions are highly individual but still can be grouped into common categories that occur frequently. The predominant symptoms include weakness, dizziness, hyperirritability, discomfort from feeling hot and sweaty, loss of appetite, and vomiting and nausea. These symptoms may not be intense enough to require medical attention, but they can be serious enough to interfere directly with military performance, or indirectly by influencing morale and motivation and by distracting the individual from the tasks to be performed. Although the tendency might be to return military personnel to duty when the physical signs of heat illness have diminished, there may still be residual symptoms that are not obvious but need to be taken into account. Commanders and medical personnel should therefore be warned about returning service members to duty too soon after they have suffered heat injuries.

Discomfort

Discomfort due to the heat can have a strong effect on individual capability. In combat situations and emergencies, discomfort may be ignored by the service member, but otherwise it can become severe enough to actually incapacitate. Heat combined with high humidity intensifies discomfort for most people. Nevertheless, discomfort is a highly individual subjective reaction; one person's idea of intolerable heat may be considerably different from another's. We can speculate that soldiers who grew up in hot regions should be more accustomed to hot conditions and will probably endure heat somewhat better, overall, than those who grew up in colder parts of the world. Acclimatization and experience with heat will help to remedy such differences by accustoming troops to heat exposure. Nevertheless, military leaders must realize that the urgency of discomfort during severe heat exposure can be a major factor in the willingness and motivation of individuals to perform their assignments. Military personnel who are trained in the heat should be able to endure higher heat conditions for longer periods than those with the same military task experience but who were trained under more comfortable conditions.

A considerable amount of research data[59–61] shows clearly that heat exposure seriously disrupts normal sleep. Sleep deprivation leads to disturbances of logical reasoning and creative ability, inability to concentrate effectively and react quickly, and weariness and irritability, all of which are clearly impediments to military performance. Service members who are exposed to hot conditions, particularly for prolonged periods when sleep deprivation can build up, should be expected to show impaired performance of many military activities involving the above factors. They may be at risk when driving vehicles, and may show lapses of performance during monitoring and surveillance assignments, and while performing mental activities ranging from simple substitution tasks, such as decoding messages, to complex planning of tactical operations. It may not be possible to counteract such effects, but military commanders and medical officers need to know about such impairments, and to expect them to be potential hazards during hot-weather operations.

Social Effects

Although there is considerable literature on both individual and group subjective reactions to heat

exposure, specific or quantitative conclusions about such reactions cannot yet be made from available research findings. Still, there is a common belief that heat, humidity, and crowding affect human behavior, probably as much from discomfort as from reducing physical capability in the heat. The common expression, "It's not only the heat, it's the humidity," conveys its own meaning and, for the most part, is probably true. However, the transition from this truism to practical recommendations for military operations in the heat is hard to make based on our knowledge of how people react to hot, humid, and crowded conditions. Common sense tells us to provide shelter from the sun, make sure that adequate cool water is available and that soldiers drink required amounts, and to attempt to ease crowded conditions. It may also be necessary to inform military leaders that the old idea of water discipline education (ie, that personnel can be trained to get along on less water by denying it to them), is dangerous and wrong. Another equally dangerous misconception is the idea that "sucking pebbles" to stimulate salivation somehow reduces thirst.

There is a solid database on the effects of heat and its relationship to violence and aggression in crowded situations. This research has clear implications for military team situations. Commanders should expect that personnel will be more argumentative and irritable during hot weather, and especially in crowded conditions, such as on troop ships, and in confined areas, such as tanks and foxholes. However, it may be possible to avoid or reduce such reactions by informing troops that they are natural and typical results of heat and overcrowding, to expect them to happen, and to try to keep them under control.

SUMMARY

The military needs to understand how mental performance, psychomotor performance, and subjective responses vary with severity of heat stress. Understanding this relationship is important because heat stress can significantly impair military performance and because psychological changes often precede the onset of critical physiological changes. Establishing well-defined relationships between climatic conditions and psychological performance has been difficult. Thermal stress researchers have attempted to identify psychological breaking points in performance, but the environmental conditions employed to simulate the natural world (combinations of temperature, humidity, wind speed, and exposure time) do not lend themselves to systematic, real-world organization. Therefore, it is difficult to make broad generalizations about the effects of heat stress on psychological performance. Nevertheless, there is general agreement that

- the upper limit of heat exposure for unimpaired psychomotor performance is 90°F WBGT;
- the upper limit of heat exposure for unimpaired mental performance is 85°F WBGT if the service member is required to perform the task for 2 hours or longer; at less than 1 hour on the task, individuals can perform proficiently at higher ambient temperatures approaching 109°F WBGT; and
- continuous repetitive tasks with relatively low arousal value tend to be the most affected.

Psychological performance during ambient heat exposure is compounded for military personnel because they are often encumbered by mission-essential clothing and equipment, including, for example, chemical protective clothing or medications such as nerve-agent antidotes, or both. Realistic military training in hot environments followed by persistent practice of military tasks in hot environments will attenuate otherwise severe impairments in performance. Humans differ in their predispositions, ranges of capability, motivations, and expectations for success. Although for convenience we tend to conceptualize human performance in terms of averages, military personnel still respond as individuals with different talents, initiatives, and attitudes.

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Chapter 5

PATHOPHYSIOLOGY OF HEATSTROKE

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INTRODUCTION

If the pathophysiology of heat stress and heatstroke were well understood, then this entire chapter could be written in only three or four pages. The length of this chapter indicates that although many facts are known, the relative importance of their roles is poorly understood. If there is a hierarchy of homeostatic mechanisms, then which is more important: the need to control elevated body temperature by sweating, or the need to maintain plasma volume by not sweating? Heat alters the physiology of all the systems in the body. Here are presented the facts, and our interpretations of them, based on current knowledge (Figure 5-1). Certainly, most observers agree that work in the heat is an enormous regulatory challenge. The existence of heat illness within a given population at risk suggests four simple observations or assumptions:

1. Excess physiological strain results in homeostatic failure.
2. There is a natural variation between individuals in the response to heat and exercise.
3. Hemodynamic failure (ie, syncope, hypotension, and heat exhaustion) may not prevent severe hyperthermia.
4. Because hyperthermia is not painful and may even be euphoric, volitional behavior, expressed as continued performance of exercise, is often maintained even as the risk of heat injury increases (ie, an individual sees others collapse but rejects the notion of personal risk).

More complete clinical and theoretical descriptions of heat illnesses are contained in other chapters in this textbook and in Chapter 8 of *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*.[1]

Four thousand years ago, denial of water to the enemy through siege was a well-recognized form of warfare. However, siege in those days was considered the least desirable and most expensive method of the routes of attacking a walled city (going over, through, or under the wall; by ruse; or any combination).[2] The defending forces safely within the walls could minimize their exposure to the sun and heat, thus reducing their requirements for water, which was stored in large cisterns, in addition to any obtained from springs or wells. As a result, a siege could last for months or even years, and severely damage the attacker's economy. Eventually, however, a combination of dehydration, starvation, and disease could sufficiently weaken defenses for a successful attack, or could lower morale enough for surrender. Modern sieges similarly last for months and often fail because the defender usually has sufficient water in storage (eg, Jerusalem, 1947) or a "back door" for supplies (eg, Iraq, Desert Shield, 1990/91).

Modern warfare is usually characterized by high-intensity battles with rapid troop movements, and may occur in tropical countries with high heat loads. Therefore, soldiers lose a great amount of water through sweating, which must be made up by extensive diversion of logistical resources. Because each soldier requires a minimum of 4.5 gal/day (~ 16 L/d) during summer combat, for a 10,000-man division, at least 160 tons of water or more per day would be required, which must be carefully distributed and dispensed. The consequences of insufficient water can be avoided even in a desert, but soldiers must be instructed where and how to look for underground supplies (Exhibit 5-1).

Heatstroke, a potentially lethal illness, is important even among the lower animals, where it is sometimes used (seemingly deliberately) in interspecies conflict. If hornets attack the hives of Japanese honey bees, scores of bees closely crowd around the invading hornet, forming a "ball of bees" several layers thick.[3] The bees rapidly vibrate their thoracic muscles, heating themselves and the hornet to approximately 116°F (46.7°C). But while the bees are relatively heat-tolerant and survive, the invading hornet collapses and dies of "heatstroke." There is a circuitous military lesson here: try to raise the core temperature of the enemy as much as possible, and by any means possible, such as by forcing them to be on the move, or to wear encapsulation gear, or to prevent their consumption of water.

The following historical narrative is a classic description of the devastating impact of the failure of logistics to provide water to a military unit, this one deployed in Texas. This excerpt is from the official report to the Medical Director, Department of Texas, September 1877, titled "A Cavalry Detachment Three and a Half Days Without Water," by Captain J. H. T. King, Assistant Surgeon, US Army Post Surgeon, Fort Concho, Texas[4]:

> The next day found them still marching onwards, and the mid-day tropical heat causing great suffering. The desire for water now became uncontrollable. The most loathsome fluid would now have been accepted to moisten their swollen tongues and supply their inward craving. The salivary and mucous secretions had long been absent, their mouths and throats were so parched that they could

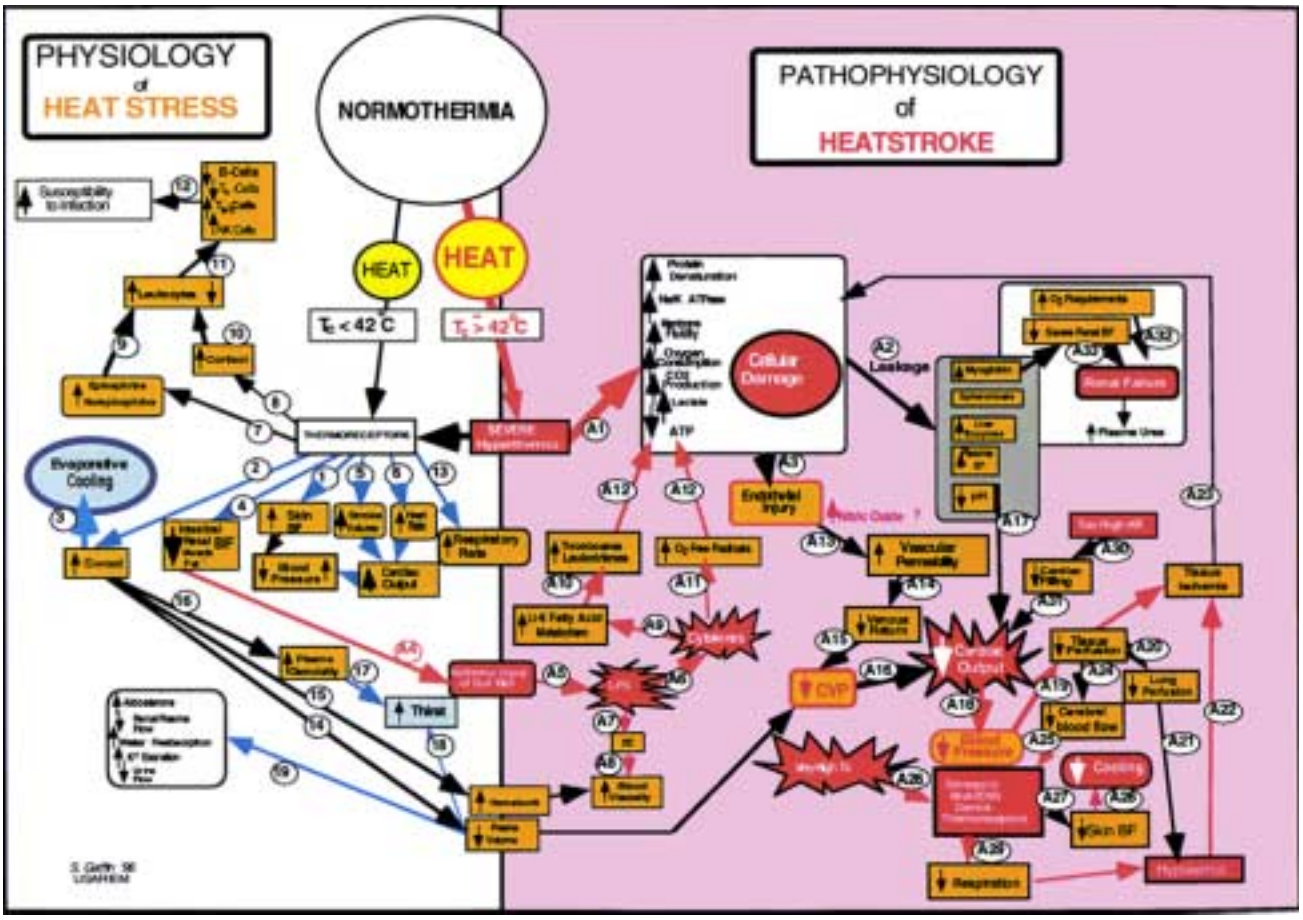

Fig. 5-1. A model of the physiology and pathophysiology of heat stress and heatstroke. To clarify physiological mechanisms, core temperature (T_c) has been divided into two regions: normal thermoregulation (left, 1–19) and impaired homeostasis (right, A1–A33).

Normal Thermoregulation. Under a moderate heat load, as skin or T_c or both rise, thermoreceptors (1) increase skin blood flow (skin BF) and (2) cause the secretion of sweat to (3) result in evaporative cooling. To prevent a drop in blood pressure (4), blood flow to the splanchnic regions and to muscle are reduced and (5) stroke volume and then (6) heart rate are increased. Then (7) catecholamines are secreted, followed by corticotropin releasing factor (CRF), which leads to the secretion of adrenocorticotropic hormone (ACTH), followed by (8) cortisol. Catecholamines (9) cause a leukocytosis, while cortisol (10) causes a leukopenia, leading to (11) changes in the amounts of leukocyte subsets. If the heat stress is prolonged and severe, the immunosuppression could (12) lead to subsequent increased susceptibility to infections. As temperature rises (13), the respiratory rate increases. As a result of sweating, (14) plasma volume decreases and (15) hematocrit and (16) plasma osmolality rise, leading to (17,18) the sensation of thirst and release of antidiuretic hormone (ADH). Reduced plasma volume (14), together with reduced renal blood flow, lead to (19) rises in water-sparing hormones and reduced kidney function.

Impaired Homeostasis. In the event or prior or concurrent exercise, these events occur at lower T_c and earlier. As T_c continues to rise above approximately 40°C to 42°C (104°F–107.6°F), direct hyperthermic damage (A1) to cells commences with increases in membrane fluidity and permeability, increases in metabolic rate, including the activity of the Na^+-K^+ adenosine triphosphatase (ATPase) pump, increases in a variety of metabolites and decreases in cellular adenosine triphosphate (ATP) content. At the same time, the reduction in intestinal blood flow (4) becomes more severe, leading to (A4) ischemic injury of the gut wall. This in turn leads to rises in (A5) circulating toxic lipopolysaccharides (LPS) and (A6) cytokines. By activating a blood factor (A7), LPS causes (A8) disseminated intravascular coagulation (DIC) and its consequent rise in blood viscosity. Thermal injury of endothelia (A3), together with elevated cytokines, leads to (A9) enhanced metabolism of omega-6 fatty acids, including (A10) the production of thromboxanes and leukotrienes, (A11) oxygen free radicals and (A12) further cellular injury, probably production of toxic nitric oxide, and (A13) increased vascular permeability. This leads to the loss of fluids into the tissues and thus (A14) reduced venous return and (A15) consequent reduced central venous pressure (CVP). Through Starling's Law of the Heart (A16), cardiac output begins to fall. This is exacerbated (A17) by electrolyte changes in the blood. Eventually, (A18) blood pressure falls, leading (A19) to reduced tissue perfusion. In lung (A20), reduced perfusion leads to (A21) systemic hypoxemia and, eventually, (A22) ischemia of various tissues and organs and its consequent (A23) contribution to further cellular damage. (A24) Reduced blood flow to the brain (A25), as well as (A26) probable direct thermal denaturation, leads to damage of centrally mediated homeostatic mechanisms, (A27) reduced skin blood flow and (A28) drop in cooling rate, and (A29) a fall in respiration.

In a separate pathway, cardiac output is also depressed as a result of a (A30) too-rapid pulse, causing (A31) incomplete cardiac filling. Electrolyte derangements are made more severe by (A32) an increased metabolic rate and (A33) reductions of renal blood flow.

Adapted with permission from Hubbard RW, Gaffin SL, Squire DL. Heat-related illnesses. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. St. Louis, Mo: Mosby; 1995: 203.

EXHIBIT 5-1

FINDING WATER IN THE DESERT

At the end of the 1973 Yom Kippur War, a large group of Egyptian soldiers was found dead without battle wounds in Sinai near El Arish, undoubtedly heat casualties. Yet adequate amounts of drinkable water were very near, *just a foot and a half below the sand.* If these soldiers had been instructed in desert survival, they probably would have survived.

When rain falls slowly, it enters the soil and gradually percolates downward until it hits solid bedrock, where it accumulates. At this point, as water continues to flow downward, its upper surface within the soil gradually rises, saturates the soil, and becomes the "water table." A hole dug deeply enough into the soil will eventually reach the water table. If the hole is dug deeper than the water table, water will slowly percolate out of the soil (over several minutes to several hours) and fill the hole to the level of the water table. If the underlying bedrock is sloped, then the water may continue slowly moving downward, eventually connecting to a stream; or it may lead to a depression in the soil and create a saturated region or swamp; or the water may remain buried for hundreds and even thousands of years.

The question is, How do you find it? To find underground water in the desert, the soldier should search for the presence of markers such as vegetation growing where there is no visible water. Presumably, the plants' roots have found subsurface water. If the plant is small, then the water would not be expected to be too far down. The soldier should dig at least 5 or 6 feet until wet sand is reached, and then dig a little deeper. Gradually, the hole will fill with water. The amount of water available could be very little or very great.

Appropriate places in the desert to search for plant life indicating the presence of water would be along wadis (dry river beds), or in spots under cliffs where *surfaces have been ground smooth* from rocks in mudslides or from flash floods from the occasional rainstorms. Grinding rocks sometimes wear away deep holes in stone beneath cliffs, which become filled with a mixture of rocks and water, and then are covered with a thick layer of mud. The water remains to be found and used.

Source: Y. Gutterman, PhD; Professor, Blaustein Institute for Desert Research, Ben-Gurion University of the Negev, Sede Boker, Israel. Personal communication, 1980.

not swallow the Government hard bread; after being masticated it accumulated between the teeth and in the palate, whence it had to be extracted with the fingers; the same occurred with mesquite beans and whatever else they attempted to eat. The sensibility of the lingual and buccal mucous membranes was so much impaired that they could not perceive when anything was in their mouths. … [B]rown sugar would not dissolve in their mouths, and it was impossible for them to swallow it. Vertigo and dimness of vision affected all; they had difficulty in speaking, voices weak and strange sounding, and they were troubled with deafness, appearing stupid to each other, questions having to be repeated several times before they could be understood; they were also very feeble and had a tottering gait. Many were delirious. What little sleep they were able to get was disturbed with ever recurring dreams of banquets, feasts, and similar scenes in which they were enjoying every kind of dainty food and delicious drink. At this stage they would in all likelihood have perished had they not resorted to the use of horse blood. As the horses gave out they cut them open and drank their blood. The horses had been so long deprived of every kind of fluid that their blood was thick and coagulated instantly on exposure; nevertheless, at the time it appeared more delicious than anything they had ever tasted; in fact every one was so eager to obtain it that discipline alone prevented them from struggling for more than the stinted share allowable to each. The heart and other viscera were grasped and sucked as if to secure even the semblance of moisture. At first they could not swallow the clotted blood, but had to hold it in their mouths, moving it to and fro between the teeth until it became somewhat broken up, after which they were enabled to force it down their parched throats. This horse blood quickly developed diarrhea, passing though the bowels almost as soon as taken; their own urine, which was very scanty and deep colored, they drank thankfully, first sweetening it with sugar. The inclination to urinate was absent and micturition performed with difficulty. A few drank the horses' urine, although at times it was caught in cups and given to the animals themselves. They became oppressed with dyspnea and a feeling of suffocation as though the sides of the trachea were adhering, to relieve which they closed the lips and breathed through the nose, prolonging the intervals between each inspiration as much as possible, gazing on each other, their lips thus closed were

observed to be covered with a whitish, dry froth and had a ghostly, pale, lifeless appearance as though they would never be opened again. Their fingers and the palms of their hands looked shrivelled and pale; some who had removed their boots suffered from swollen feet and legs.[4(pp194–195)]

PATHOPHYSIOLOGY OF HEATSTROKE

Hyperthermia and dehydration are two different illnesses, but which interact and may cause heatstroke. A given hyperthermic core temperature (T_c) leads to one level of altered cardiovascular functions (cardiac output, stroke volume, blood pressure, systemic vascular resistance) and dehydration, another. However, when a subject is rendered both hyperthermic and dehydrated, the alterations in cardiovascular variables may become even greater than their sum.[5] Soldiers in a hot environment, even at rest, are usually moderately dehydrated (except immediately after a meal) unless they are forced to drink by command. Therefore, in summer, heatstroke is accompanied by some degree of dehydration. For details of the mechanisms of heat production and dissipation see Chapter 2, Human Adaptation to Hot Environments, and Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues.

Effect of Hyperthermia on Cells

Ion Pathways in the Plasma Membrane

Ions do not readily cross lipid bilayers despite their large concentration gradients across plasma membranes. In general, they require specialized channels or carriers to do so (Figure 5-2). Membrane channels are proteins that contain hydrophilic pores that penetrate the lipid bilayer, permitting the diffusion of specific ions down their electrochemical gradients to enter or leave cells.[6] Cotransporters are different types of pathways, which move the ions across the cell membranes *up* their electrochemical gradients by coupling the translocation of at least two ions (eg, K^+ and Cl^-), using the energy stored in preformed chemical gradients such as those of H^+ or Na^+ (rather than the concentration gradients of the transported ions).[6]

The Na-K-2Cl Cotransporter. This important family is present in most cell types, especially in excitable cells and red blood cells (RBCs), and mediates the coupled, electrically neutral movement of Na^+, K^+, and Cl^-, and water. The driving force is solely the chemical gradients of the three ions. Under normal conditions, on the one hand the force mediates an inward movement of Na^+, K^+, and Cl^-, and, on the other hand, interacts with K^+ channels and the Na^+/K^+ adenosine triphosphatase (ATPase) pump for the secretion of salt.[7]

The K-Cl Cotransporter. This pathway is present in many cell types but is not normally active. However, heat and osmotic swelling activate this cotransporter and lead to a *net efflux of K^+, Cl^-, and water,* with a consequent decrease in cell volume. Furthermore, it may be involved in the heat-induced exacerbation of sickle-cell disease. In that disease, there is a genetic defect in the hemoglobin (Hb) ß-chain, leading to polymerization of the hemoglobin and causing characteristic sickling of the cells. The rate and extent of polymerization greatly increase with small decreases in RBC volume. Patients with sickle-cell anemia have very active K-Cl cotransport mechanisms in their RBCs. As a result, during heat stress the K-Cl cotransporter is activated, resulting in smaller volumes in those RBCs, increased polymerization of hemoglobin, and, consequently, enhanced pathology.[8,9]

The Na^+/H^+ Exchanger. This is an electrically neutral transporter that exchanges *extra*cellular Na^+ with *intra*cellular H^+ (ie, Na^+ *enters* and H^+ *leaves*). In many cell types it regulates (*a*) both intracellular pH (pH_i) and plasma H^+, (*b*) the concentration of intracellular sodium ($[Na^+]_i$), and (*c*) cell volume. The exchanger is normally "silent" and is activated by intracellular acidosis, returning pH_i to resting levels, and only indirectly regulates $[Na^+]_i$. It is powerfully stimulated by cell shrinkage (which occurs during the hypovolemia associated with heat stress) and regulates cell volume together with the Na^+-K^+-$2Cl^-$ cotransporter.

The Na^+/Ca^{2+} Exchanger. Normally, the Na^+/Ca^{2+} exchanger in the "forward" mode transports Ca^{2+} out of cells coupled to Na^+ influx with a usual stoichiometry of 3 Na^+ to 1 Ca^{2+}.[10] However, in some cell types, particularly in excitable cells, elevated $[Na^+]_i$ may cause the exchanger to operate in the "reverse" mode and transport Na^+ out of the cell, coupled with the entry of Ca^{2+}.[10] The Na^+/Ca^{2+} exchanger normally operates at only a very small fraction of its maximum capacity and is capable of great activity, fast enough so that *during an action potential in muscle, the transient local elevation of $[Na^+]_i$ triggers the Na^+/Ca^{2+} exchanger, speeding Ca^{2+} influx* and causing the subsequent regenerative release of Ca^{2+} from the sarcoplasmic reticulum.

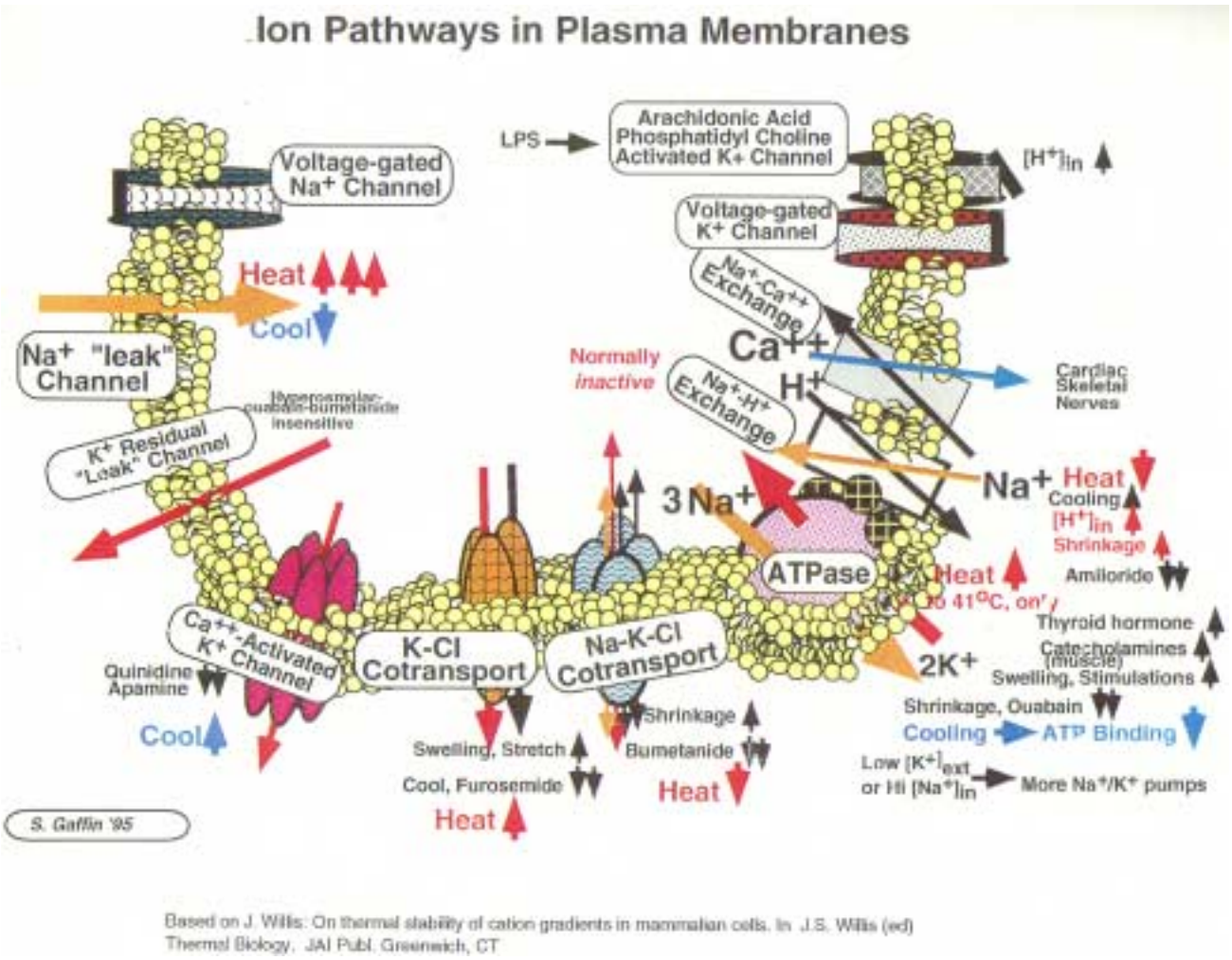

Fig. 5-2. Membrane pumps and ion pathways. Cell membranes contain a variety of specialized proteins that mediate ion transport across the plasma membrane and intracellular ion concentrations. These proteins may form channels through which ions travel passively, according to their electrochemical gradients, and may have gates "opened" or "closed" by elevated or reduced transmembrane potential, or the presence of specific intracellular ions, metabolites, or extracellular hormones (voltage-gated Na^+ channel, Na^+ leak channel, K^+ residual "leak" channel, Ca^{2+} voltage– or arachidonic acid–activated K^+ channel). Other substances may be transported across cell membranes by a "pump" mechanism (Na^+K^+ATPase) requiring a chemical energy source (adenosine triphosphate), by using the energy of preformed gradients (Na^+–H^+ exchanger, Na^+–Ca^{2+} exchanger, or by carrying along other ions (Na–K–Cl cotransporters). Temperature changes alter the transport rate of each process, which, in turn, can alter intracellular (in) and extracellular (ext) ion concentrations.
Source: Willis JS. Thermal compensation of passive membrane transport of cations: A lesson from nonhibernators. In: Geiser F, Hulbert AJ, Nicol SC, eds. *Adaptations to the Cold: 10th International Hibernation Symposium*. Armidale, New South Wales, Australia: University of New England Press; 1996: 253.

Other Pathways. Other pathways present in most cells include the Na^+/K^+ ATPase pump, Ca^{2+} ATPase pump, and Ca^{2+} channels, as well as the passive diffusion channels for Na^+, K^+, and Cl^- (see Figure 5-2).[11] Heat alters the rate and function of some of those pathways, leading to changes in intracellular concentrations of Na^+, K^+, Ca^{2+}, and Cl^-, all of which may alter cellular metabolism, tissue function, and reflexes, and lead to dyshomeostasis and heat illness. Specifically, both $[Na^+]_i$ and the concentration of intracellular calcium ($[Ca^{2+}]_i$) increase with heating.[12,13]

Cell Shape

Heat stress causes changes in the structure of subcellular organelles in many cell types including

detachment of cortical microfilaments from the plasma membrane,[14] collapse of the cytoskeleton, swelling of the mitochondria and the endoplasmic reticulum,[15] and disaggregation of polyribosomes[16,17] and nucleoli.[17] Furthermore, heat causes the plasma membrane to undergo gross deformations, forming bulges known as blebs.[18] Blebs result from heat-induced increases in membrane fluidity,[19,20] altered membrane function,[21] increased permeability[22] with solute leakage,[23–27] and alterations in the linkage of the cytoskeleton to the plasma membrane.[28] Such changes are not necessarily lethal and, up to a point, bleb formation may be adaptive, leading to increased survival of the cell.[29–31] The more severely the cells are heated in vitro, the greater the extent of blebbing and greater is the proportion of cells killed.[30,32] For additional information, see the review by Hales, Hubbard, and Gaffin in the *Handbook of Physiology*.[33]

RBCs also undergo rearrangements in the cytoskeleton with heat and, rather than forming blebs, they form spheroids at elevated temperatures. These enlarged, spherically shaped cells are much less efficient at gas exchange than normal RBCs and probably contribute to reduced partial pressure of oxygen (Po_2) in the tissues at elevated temperatures. Spheroid formation has been found in athletes during long-distance running and may contribute to their physical collapse during exercise.[34]

Increased membrane permeability is indicated by rises in circulating enzymes. Creatine kinase is the first enzyme detected at a T_c as low as 39.5°C in monkeys,[35] and 42.5°C in rats, followed by lactate dehydrogenase.[36]

Apoptosis

Suicide of certain individual cells in the body (apoptosis) is genetically programmed and necessary during embryogenesis, development, metamorphosis, normal cell turnover, and tissue repair. In this process, cells are broken into small vesicles containing characteristic highly condensed chromatin surrounded by intact cell membranes (apoptotic bodies), which become phagocytosed by macrophages or neighboring cells. Apoptosis differs from necrosis in that the latter involves early swelling, destruction of the plasma membrane, and spewing of cell contents into the extracellular milieu. Furthermore, necrosis may provoke an inflammatory response and lead to damage or death of neighboring cells.[37]

Whereas apoptosis had been considered to be a normal, genetically programmed event, high temperatures also cause apoptosis in experimental animals and cultured cells. Hyperthermia to 41.5°C to 42.0°C for a few minutes to an hour or two caused some cells in a variety of mammalian cell types to undergo apoptosis during the next several hours.[38,39] The greatest level of whole-body hyperthermia-induced apoptosis occurs in the thymus, spleen, and lymph nodes, and in the small intestinal mucosa. Resistance to inappropriate apoptosis can be experimentally induced in cultured cells by stresses that induce heat shock proteins (see discussion below).[40] In polymorphonuclear leukocytes, the presence of interleukin-6 delays normal apoptosis following inflammation, and in that case, may lead to tissue injury through excess local production of active oxygen species from the too-old polymorphonuclear leukocytes.[41–43] Long-distance running or marching with a heavy load in the summer can lead to a T_c of 41.5°C to 42.0°C. In principle, this rise in temperature may lead to inappropriate apoptosis in some cells and render a soldier at risk during the following day. Table 5-1 displays the rise in core (rectal, T_{re}) temperatures of eight acclimatized Israeli soldiers, each carrying a 35-kg pack, after they marched 8 hours on an "ordinary" summer day; short rest periods were taken during the march. The core temperatures of all soldiers rose, the lowest by 0.9 Centigrade degrees and the highest by 5.0 Centigrade degrees.

In response to a rising local temperature, the least stable proteins within the body denature and their functional activities fall. However, because of the presence of diverse alternative metabolic pathways within individual cells, the cells are able to survive for minutes to hours or longer, or until temperature rises so much that even those pathways are compro-

TABLE 5-1

ISRAELI SOLDIERS' CORE TEMPERATURE RISE DURING AN 8-HOUR MARCH*

Soldier No.	T_{re} Before March	T_{re} at End of March
1	37.6	39.9
2	37.3	40.6
3	37.4	38.3
4	37.4	42.1
5	37.8	41.5
6	37.4	42.4
7	37.6	42.2
8	37.3	41.5

*Carrying a 35-kg pack
T_{re}: Rectal temperature
Reprinted with permission from Gilat T, Shibolet S, Sohar E. The mechanism of heatstroke. *J Trop Med Hyg.* 1963;66:208.

mised. Membrane pumps may increase their activities with temperature, but eventually they peak and then decline, leading to alterations in the concentrations of intracellular electrolytes (see Figure 5-2). There is probably also a time–temperature relationship at which the pump deteriorates at lower temperatures, if held there for sufficient time. In addition, the increasing fluidity of the membrane with temperature further alters membrane permeability and function.

Rises in temperature increase rates of chemical reactions including adenosine 5'-triphosphate (ATP) hydrolysis, thereby requiring greater O_2 and nutrient delivery to each cell, and faster transport of CO_2 and waste products to the lungs, liver, and kidneys. Consequently, a soldier's respiratory rate rises with temperature but eventually peaks and then declines, resulting in reduced arterial O_2 concentration, rising CO_2 concentration, and falling pH.[44] (The rise in respiratory rate leads to deleterious alterations in blood chemistry and an increase in O_2 demand by the muscles of respiration, which in extreme situations, can lead to respiratory failure. Consequently, respiratory failure is often a component of the pathophysiology of severe heatstroke.) Such conditions lead to increased lactate production by anaerobic pathways and acidosis. Furthermore, if the temperature is sufficiently elevated, then additional lactate may be produced even in the presence of normal O_2 delivery.[44] Overall, in response to hyperthermia and elevated rates of ATP hydrolysis, there occur relative and absolute reductions in O_2 and nutrient delivery, acidosis, and a drop in intracellular ATP concentration.

Calcium

Because so many metabolic pathways are activated by transient high $[Ca^{2+}]_i$, including the opening of Ca^{2+}-activated K^+ channels for K^+ efflux, Ca^{2+} regulation is critical for cell survival. Because of its double charge, Ca^{2+} cannot "leak" through a normal plasma membrane by diffusion but requires a variety of Ca^{2+} channels, exchangers, and pumps. Because its electrochemical gradient permits inward diffusion, influx of Ca^{2+} is mediated by both the reverse mode of the Na^+/Ca^{2+} exchanger and by calcium channels. Ionic calcium efflux, however, is strongly impeded by both unfavorable concentration gradient and charge. Ca^{2+} has two mechanisms for exiting from the cell[10]:

1. a rapid, high-capacity but low-affinity Na^+/Ca^{2+} exchanger, mentioned above, and
2. a slower but high-affinity membrane Ca^{2+} ATPase pump, which is ATP-dependent.

Heating a variety of cell types causes an elevation of $[Ca^{2+}]_i$ from a "resting" level of approximately 100 to 300 nmol, to 700 to 1,000 nmol by the entry of Ca^{2+} from external solution and from the release of Ca^{2+} from the intracellular calcium stores.[13] This rise appears to be greater in cells obtained from black individuals than in those from white individuals, but the biochemical and physiological implications are unclear.[45] Heat acclimation of certain cell lines blunts this heat-induced rise in $[Ca^{2+}]_i$.[46]

Sodium

Because of the well-known pH-dependence of chemical reactions, the maintenance of intracellular pH within very narrow limits is an important cellular requirement. During cellular acidosis a major pathway for proton removal is the Na^+/H^+ exchanger (Na^+ enters, H^+ leaves), which is "quiet" at normal pH but is activated by acidification and by cell shrinkage.[11,47] Heat activates the exchanger indirectly through both pathways. The temperatures observed in heat illnesses alter metabolism and acidify cells by 0.1 to 0.4 pH units, and sweating caused by hyperthermia leads to hyperosmolality, causing cells to shrink. Both the acidosis and shrinkage pathways activate the Na^+/H^+ exchanger, leading to a rise in $[Na^+]_i$ as protons are removed. The activity of the Na^+/H^+ exchanger may also be seasonally adjusted (presumably it is more active in summer), as part of a more general acclimatization response, because the Na^+/H^+ exchanger is only minimally active in bears during hibernation.[11] The increase in $[Na^+]_i$ in severe hyperthermia is not a transient event but probably is sustained for several minutes to hours after return to normothermia, until it is eventually returned to normal values by other pathways such as the Na^+/K^+ ATPase pump.

Heating cells to 42°C to 43°C causes a rise in $[Na^+]_i$, which may activate the Na^+/Ca^{2+} exchanger, thus indirectly altering $[Ca^{2+}]_i$. This rise in $[Na^+]_i$ persists for minutes to hours after a cell returns to 37°C, and thus alters cell physiology for a prolonged period of time. This temperature seems to be a "critical" temperature in heatstroke pathophysiology because significant deaths in passive heatstroke (ie, in a resting or anesthetized experimental animal) also commence at rectal temperatures of 42°C to 43°C. However, the temperature leading to injury and death depends on a variety of factors and is lower during exercise and varies with health status, hydration, and recent illness. A highly motivated soldier may not admit any illness, and this is a particularly serious risk factor.

Possibly, elevated $[Na^+]_i$ is a "trigger," which through elevated $[Ca^{2+}]_i$, activates cell metabolic pathways and consequent pathophysiological actions, leading to irreversibility and heatstroke death. A rise in $[Na^+]_i$ in excitable cells, according to the Nernst relationship, leads to a reduced magnitude of action potential. In the brain, this would reduce the amount of neurotransmitter released, which in turn, would alter synaptic-dependent events such as cognition, initiation of reflexes, possibly causing inappropriate changes in skin blood flow (BF_{sk}) and sweat secretion, and lead to an overall rise in T_c and death. High temperatures due to infection would also be expected to show rises in $[Na^+]_i$ with similar alterations in brain function.

This rise in $[Na^+]_i$ due to hyperthermia may have important biochemical, metabolic, and physiological effects:

1. Because of the 3:1 stoichiometry of Na^+:Ca^{2+} in the Na^+/Ca^{2+} exchanger, its energetics and direction depend on the cube of the Na^+ concentration gradient. Therefore, a small change in $[Na^+]_i$ would greatly alter the driving force of Ca^{2+} movements via the Na^+/Ca^{2+} exchanger. With a typical $[Na^+]_i$ of 6 mmol and a resting membrane potential (Em) of –70 mV, the driving force is in the direction of net Ca^{2+} efflux, as is normally required by the cell. However, if $[Na^+]_i$ should rise to 15 mmol, then the exchanger would catalyze net *Ca^{2+}influx* in resting cells, obviously leading to major metabolic changes, including the lowering of $[K^+]_i$ through the opening of Ca^{2+}-activated K^+ channels.[48]
2. A rise in $[Na^+]_i$ in nerve and muscle cells would reduce the magnitude of action potentials, according to the Nernst relationship, and in the brain would reduce the amount of neurotransmitter released in synapses. Alterations in synaptic activity could affect reflexes and sensoria and could account for the dyshomeostasis in much of the pathophysiology described here, as well as explain the altered mental activity of heatstroke.

This pathway may be involved in a positive feedback loop, in which high T_c (through acidosis) increases $[Na^+]_i$, which elevates the Na^+-K^+ ATPase activity, and results in still greater production of heat. In addition, dehydration would lead to cell shrinkage, in turn activating the Na^+-H^+ exchange, leading to a positive feedback loop of more rapid entry of Na^+, raised $[Na^+]_i$, consequent speed-up of the Na^+/K^+ ATPase pump, and even more elevated heat production.

Potassium

Experimentally, heating causes a *net loss* of potassium ions from cultured cells in vitro and causes a *rise* in plasma K^+ in vivo.[26,49,50] Up to a T_c of approximately 41°C, $[K^+]_i$ and $[Na^+]_i$ remain approximately constant despite their increased influxes and effluxes through diffusion and the Na^+/K^+ ATPase pump.[51] However, the 3:2 stoichiometry of the Na^+/K^+ ATPase pump should lead to a net rise in $[K^+]_i$. Because this does not occur, excess K^+ must be transferred out of the cell by other pathways, which may include the K^+-Cl^- cotransporter, Na^+-K^+-$2Cl^-$ cotransporter, diffusion through Ca^{2+}-activated K^+ channels, or arachidonic acid (AA)–activated K^+ channels. Because the K^+-Cl^- cotransporter is activated by warming, this is probably the main pathway by which K^+ leaves cells during hyperthermia.

The decline in $[K^+]_i$ indicates the ultimate inability of the Na^+/K^+ ATPase pump and Na^+-K^+-$2Cl^-$ cotransporter to balance the increased K^+ efflux through the K-Cl cotransporter. Because a good deal of wasted heat is liberated during the hydrolysis of ATP by the Na^+/K^+ ATPase pump, the benefits of rapid cooling in cases of mammalian heatstroke are due, in part, to the slowing of this pump.

Hyperthermia as well as physical exercise leads to a progressive rise in plasma K^+ concentration, reaching almost twice the normal value in some studies.[52] Such elevated plasma values during heat and exercise suggest that in tissues, concentrations may reach as high as 16 mEq/L, enough to substantially depolarize cell membranes, alter nerve and muscle function, and increase metabolic activity.[53] This rise in ionic potassium concentration outside the cell ($[K^+]_o$) might contribute to any pathophysiology induced by elevated $[Na^+]_i$ causing heat illnesses.

During low-intensity exercise, for each contraction of skeletal muscle, 7 to 11 nmol/g of Na^+ and K^+ enter and leave the cell, with a net gain of Na^+ and loss of K^+ before the Na^+/K^+ ATPase pumps are activated.[54] During near-tetanic and tetanic contractions, these changes in ion concentrations may overwhelm the pumps' capacity, even when operating at maximum rates. As a result, during exercise at normal or only moderately elevated T_c, plasma $[K^+]$ rises.[52,55] Because heat alone causes hyperkalemia, the additional K^+ due to exercise in the heat may place an insurmountable burden on a soldier, ren-

dering him susceptible to muscle weakness, fatigue, and exertional heatstroke. Elevating $[K^+]_i$ to 7 mmol causes profound weakness in humans by reducing the propagation velocities of action potentials and reducing the amplitude of the action potentials (because the membrane potentials depolarize from a less negative voltage), which reduces the amount of Ca^{2+} released into the cytoplasm of a muscle fiber.[54,56] This weakness is a protective negative feedback process, as it inhibits further muscular activity. On the other hand, certain pharmaceutical agents can reverse these effects. Treating K^+-depressed muscle with insulin, $ß_2$ blockers, and calcitonin gene-related peptide activates the Na^+/K^+ ATPase pumps within minutes, reducing $[Na^+]_i$; effects hyperpolarization; and restores muscle strength. In the future, these treatments may be found to be therapeutic for muscle weakness, fatigue, and heatstroke.

Elevations in K^+ concentrations may not be all bad. Elevated extracellular $[K^+]$ would affect nerve endings, thereby altering their sensitivities, and may be involved in a neural feedback mechanism that modulates the firing rate of motor neurons during exercise, especially exercise in the heat. This feedback mechanism could initiate protective cardiovascular reflexes such as postexercise hyperemia and rapid heart rate.[57] After exercise has ceased, reuptake of K^+ into the exercising muscles may take minutes of increased blood flow, but restoration of overall water and electrolyte balances with the various body compartments could take much longer.[54]

Energy Depletion Model

During heat stress and elevated T_c, the Na^+/K^+ ATPase pumps operate at increasing rates, hence hydrolyzing ATP more rapidly and liberating waste heat into the body faster. If the body cannot dissipate all this heat though radiation, conduction, convection, and evaporation of sweat, then, according to the laws of thermodynamics, T_c must rise, leading to still greater rates of the Na^+/K^+ ATPase pumps—an ominous positive feedback loop. The total amount of energy available to a cell is limited. Therefore, at a certain elevated temperature, ATP utilized by the activated Na^+/K^+ ATPase pump is no longer available for normal cellular processes and the cell becomes energy depleted. Experimental support for this concept is the presence of swollen cells (implying a slowing of ion pumps, which affect water transport) and the rapid development of rigor mortis (caused by depletion of ATP) at the end stage of heatstroke pathophysiology.[58] Furthermore, this concept also explains the occurrence of heatstroke deaths at lower T_c in persons exercising in the heat.[59]

Because plasma $[K^+]$ eventually rises during heat stress, and the main store of K^+ is in cells, then $[K^+]_i$ must fall. This fall would be mediated by such means as the K-Cl cotransporter and Ca^{2+}-activated K^+ channels. Overall, the rise in activity of the various ion pumps provides more waste heat, which, near the limit of sweat secretion, increases T_c still more.

Pathological Manifestations of Heatstroke in Mammals

In contrast to threshold hyperthermia mortality temperature of 42°C to 43°C for animals at rest, in exercised, heat-stressed rats, mortality occurs at a lower temperature: 40.4°C.[60] What may account for this critical temperature? Between 37°C and 40.4°C and higher, two fundamental cellular functions are altered: (1) the rate of transmembrane ion flux is increased, and (2) the rate of protein synthesis is inhibited.

As T_c rises toward and above 42°C, most of the homeostatic systems within the body are near crucial limits for proper functioning. Severe hyperthermia acts on thermoreceptors to maximize their inputs to the hypothalamus to increase stroke volume and heart rate; BF_{sk} has reached its maximum; sweating has caused plasma osmolality to rise well above the thirst threshold, and the victim experiences severe thirst (unless there has been adequate ingestion of fluids by command). Plasma volume is greatly reduced, leading to high hematocrit values and greatly increased blood viscosity, which in turn, require the heart to use more energy to maintain the same cardiac output. As plasma volume continues to fall and osmolality to rise, the secretion of sweat falls, leading to reduced rates of evaporation and cooling and even faster rises in T_c—a second ominous positive feedback loop. Eventually, BF_{sk} as well as sweat secretion fall, and heat dissipation fails.

The pathological features of heatstroke are similar no matter what the cause of the heat illness. They are manifested by (1) swelling and degeneration of tissue and cell structures, and (2) widespread microscopic to massive hemorrhages.[61] The organs are congested, with increased weights and swollen cells.

Kidney

During moderate hyperthermia and normovolemia, the "excess" plasma K^+ released from the cells is excreted in the kidneys, thus maintaining normokalemia. In response to moderate hyperthermia and gradually decreasing blood volumes, blood flow to the kid-

neys is reduced, leading to reduced filtration and reduced urinary flow. Nevertheless, K^+ excretion persists and plasma K^+ is maintained near normal levels until blood pressure has fallen significantly. Eventually, as T_c rises above approximately 42°C and mean arterial pressure falls, renal blood flow is greatly reduced, and kidney function fails.[44] At this point, the K^+ released from cells in response to hyperthermia is no longer excreted; it accumulates in the plasma, leading to frank hyperkalemia. Plasma K^+ concentration may rise as high as 8 mmol, approximately double baseline levels. Eight millimoles of potassium in the blood implies the presence of 16 mmol K^+ in extracellular fluid in the tissues, a value high enough to depolarize some excitable tissues almost to threshold.

When T_c has risen so high that mean arterial pressure (MAP) falls, the O_2 content of arterial blood also falls.[44] At approximately the same time, renal blood flow drops still further and may be reduced to such low levels that O_2 delivery is below the minimum required for survival, and tissue destruction follows. Renal failure is an often-reported consequence of heatstroke.

At postmortem examination, the kidneys are found congested with macroscopic hemorrhages in 20% of cases.[61(p288)] Pigmented casts were present in the distal convoluted tubules when heatstroke victims survived more than 24 hours before dying.[61]

Circulation: Electrolyte Imbalance

In experimental studies using isolated cells, hyperthermia leads to a rise in $[Na^+]_i$ and a fall in $[K^+]_i$, but the results are far from clear in patients in the clinical setting. On admission to a hospital, victims of heat illnesses may have elevated, normal, or reduced concentrations of serum Na^+ or K^+. The interpretation of these results has generated controversy. However, on the basis of recent studies, the results may be explained as follows[44]:

1. Hyperthermia leads to a primary loss of K^+ from tissues into the blood circulation.
2. In a victim of heatstroke, the kidney would excrete this "excess" K^+ into the urine, thus maintaining normal or near-normal serum K^+.
3. When the combination of elevated T_c and dehydration is sufficiently severe, renal blood flow, filtration, and urine output all fall, with a consequent rise in serum K^+.
4. However, if the hyperkalemic victim is infused with liters of crystalloid in the field, thus raising systemic blood pressure, then normal renal function may return, causing a rapid excretion of the elevated plasma K^+ into the urine.
5. Cooling of the victim to near 37°C would terminate loss of K^+ from the tissues and cause the reuptake of K^+ by the cells from extracellular fluid. The K-Cl exchanger becomes *in*activated by cooler temperatures, while the Na^+/K^+ ATPase pump continues to pump K^+ inward from interstitial fluid and plasma.
6. As a result, by the time the victim reaches a hospital, serum K^+ may be reduced to normokalemia or even *hypo*kalemia.

Prior conditions, such as intense training in the heat over a period of several days, tend to lower body potassium, thus worsening hypokalemia. Similarly, acute intense hyperthermia could mask preexisting hypokalemia by acutely raising plasma levels. On treatment, potassium levels would return to normal ranges, effectively producing misleading clinical indications. Thus, to arrive at a better diagnosis and prognosis, it is important for the medical officer to know the thermal exposure and nutritional history of the soldier before collapse. Hyperkalemia is usually a grave clinical sign.

Studies of sodium concentration are less clear and represent an equally complex diagnostic situation because there is only a small absolute rise in $[Na^+]_i$, and this loss from the circulation represents a small percentage of the background serum sodium. Elevations in serum sodium are often secondary to depletion of body water.

Within certain temperature limits, normal thermoregulatory mechanisms are capable of restoring T_c to 37°C from either hyperthermia or hypothermia. However, if T_c rises to approximately 42.5°C, then metabolic pathways may be so affected that inappropriate physiological responses occur. Vascular collapse, shock, and death can follow unless countermeasures such as cooling and therapy to increase plasma volume are initiated. It is not certain at present whether there is a *single* critical intracellular derangement occurring at 42.5°C that ultimately leads to the activation of many harmful metabolic pathways and heatstroke death, or if *many* harmful pathways become independently established at about the same time in response to the given temperature. Probably it is the latter. In any case, the *beneficial impact of rapid cooling* on the underlying causes of energy depleting reactions *cannot be overstated,* and correlates well with reduced injury and improved recovery.

If a soldier is expending considerable metabolic energy and hydrolyzing ATP at a high rate during severe exercise, then he or she may develop the form of heatstroke called *exertional heatstroke* at a lower temperature than if the soldier had been at rest. In exertional heatstroke, metabolic heat is produced faster than the body's normal ability to dissipate it, a situation that is made worse by a high solar heat load, high ambient temperature, or both. This is in contrast to classic heatstroke, in which the heat-dissipation mechanisms are depressed. This differential mortality and morbidity, with exercise-related factors clearly demonstrated in animal research and certain series of human data, speaks to the existence of other causative parameters such as O_2 availability, energy status, and pH effects rather than direct thermal effects per se. Clearly, the local effect of exercise-induced ischemia and acidosis on certain tissues, and their reaction to heat, on the ultimate outcome of exertional heatstroke makes prediction and prognosis based entirely on thermal history alone very unlikely or exceedingly complex. If a substantial fraction of available high-energy phosphate stores (creatine phosphate plus ATP) within a cell is consumed by exercise, then less remains available for normal cell metabolism, for activating cooling mechanisms (eg, increased cardiac output), and for the repair of damage caused by elevated temperature.

Gut

Postmortem examination of heatstroke victims often shows engorged intestinal vessels, and the gastrointestinal tract may show massive ulcerations and hemorrhages.[61] The gut is an important focus in any discussion of heat illness for two important reasons:

1. its function determines whether ingested fluid and solutes are delivered to the systemic circulation to correct losses and thereby attenuate hyperthermia, dehydration, reductions in splanchnic blood flow, and gut distress; and
2. heatstroke may result from or be exacerbated by gastrointestinal dysfunction, leading to endotoxemia and circulatory collapse.

Current research indicates excellent parity between the rehydration demand and the capacity of the stomach to deliver ingested fluids (gastric emptying under optimal conditions can provide 1.8 L/h) and the intestines' capacity to absorb it (1.4–2.2 L/h). This degree of rehydration capacity should accommodate the average sweating rate of most endurance athletes, given that reductions in splanchnic blood flow (up to 80% with maximal exercise intensity) could not be maintained for long periods before other factors such as fatigue or hyperthermia intervened.

Intense and prolonged running is a common cause of gastrointestinal bleeding, and estimates as high as 85% of ultramarathoners demonstrate guaiac-positive stools from a 100-km race.[62] This bleeding varies in intensity from runner to runner and is due to gut ischemia, trauma to the gut wall, and the use of nonsteroidal antiinflammatory drugs. Exercise-induced gastrointestinal bleeding is most often mild but can be serious, and the serious cases speak to the more important issue of periodic bouts of decreased gut barrier function, especially when combined with nausea, diarrhea, and abdominal cramps. Nonsteroidal antiinflammatory drugs are widely used for training and to treat competition injuries and are known to enhance intestinal permeability and bleeding susceptibility.[63] The gastrointestinal tract has been dubbed the "canary of the body," reinforcing the concept that the presence of signs and symptoms in the gastrointestinal tract suggest an underlying disorder likely to be aggravated by exercise.[64]

The lumens of the small and large intestines contain considerable quantities of Gram-negative bacteria and their cell-wall component, highly toxic (10^{-12} mol is lethal) lipopolysaccharide (LPS, also called endotoxin). The combination of high temperature and consequent reduced intestinal blood flow injure the intestinal wall, compromising its ability to prevent LPS and other bacterial toxins from leaking out into the systemic circulation via the portal vein and intraperitoneal space.[65] If the plasma LPS concentration is high enough to overcome all the safeguards that normally inactivate it, then some LPS is complexed with a blood component, LPS-binding protein (LBP), and is bound to the cluster of differentiation 14 (CD14) receptor of macrophages and other cell types. In turn, it induces the inflammatory cytokines, tumor necrosis factor (TNF), and interleukin-1 (IL-1). These, in turn, cause the formation of oxygen free radicals and activate omega-6 fatty acid metabolism, leading to the formation of toxic prostaglandins (PGs), leukotrienes (LTs), and thromboxanes (TXs). Collectively, these agents directly damage cells and reduce blood flow to the tissues, exacerbating the pathophysiology induced by the heat.

LPS causes a second effect mediated by a completely different pathway. LPS binds to the Hageman

factor of blood, activating it, and leads to disseminated intravascular coagulation (DIC), a common complication of heatstroke. As a result of reduced blood clotting, conjunctival hemorrhage; melena; purpura; bloody diarrhea; and lung, renal, or myocardial bleeding frequently occur.[61] It is interesting that the effect of altering these pathways through enrichment of omega-3 fatty acid metabolism could provide future research avenues of investigation. (Interested readers can see Gaffin and Hubbard's 1996 article in *Wilderness and Environmental Medicine*[66] for further information on this topic.)

Liver

Hyperthermia reduces blood flow to the liver at the same time it reduces blood flow to the intestines. Therefore, delivery of O_2 and nutrients to the hepatocytes and Kupffer cells falls as T_c rises. The combination of thermal injury and local ischemia depresses liver function, which may persist even after heatstroke has been resolved. Postmortem examination of a heatstroke victim who died 30 hours after admission to a medical treatment facility showed centrolobular necrosis and, in biopsy specimens, hepatocellular degeneration around the widened centrolobular veins as well as swelling, cholestasis, and leukocytic cholangitis.[67]

Lungs and Respiratory Function

As T_c rises, tidal volume, respiratory rate, heart rate, and, to a lesser extent, stroke volume, also rise to meet the increased metabolic demands. In humans, there is a closely related rise in minute volume and heart rate. Ethical considerations usually limit studies on hyperthermic volunteers to a T_c of 39.5°C or lower. However, the temperatures reached in actual heatstroke may be several Centigrade degrees higher. In animal studies performed at those higher temperatures, respiration increases much faster than cardiac output, and the respiration rate may reach such high levels that the CO_2 is driven off too fast, resulting in respiratory alkalosis. Consistent with this, respiratory alkalosis is common in soldiers suffering from exertional heatstroke or exertional heat exhaustion with syncope and cramps.

When T_c rises sufficiently, the minute ventilation reaches a maximum and then rapidly declines to low values and may cease altogether.[44] As a result, blood becomes hypercapnic and acidotic and, if not rapidly treated, can lead to death with final pH approaching 6.9 to 6.8. In other words, heatstroke is first characterized by a respiratory alkalosis, and then is followed by a metabolic and respiratory acidosis.

Central Venous Pressure and Baroreceptors

In addition to the maintenance of heart function, proper function of the central nervous system (CNS) is probably most critical for survival during heatstroke. During early hyperthermia, arterial blood pressure remains approximately constant even though central venous pressure (CVP) progressively falls. This lowered CVP helps maintain adequate blood perfusion through the CNS by increasing the driving pressure, reducing the CNS vascular resistance by means of local vasodilation, or both. The unloading of baroreceptors in the great veins and atria is the stimulus for maintaining or raising CNS blood flow. When central blood volume was experimentally reduced in steps, arterioles in the forearm constricted first, then flow to the periphery was reduced, and the volume in the peripheral compliance vessels was reduced.[68] When the central blood volume was sufficiently reduced, and with CVP and MAP sufficiently low, then baroreceptor responses caused the forearm veins to contract to aid in the central mobilization of blood volume to resist decompensation.[69–71]

At moderate hyperthermia, thermoreceptors acting through the hypothalamus cause a dominant drive within the body toward vasodilation, leading to increasingly elevated BF_{sk} and secretion of sweat, resulting in a reduced plasma volume and reduced CVP.[72,73] However, once a certain T_c is exceeded[74,75] there is an altered sensitivity of BF_{sk} to increasing T_c. The central venous baroreceptors now initiate vasoconstrictor drives, which "override" the vasodilator drives from the thermoreceptors. At that T_c, the reduced CVP unloaded baroreceptors located in the great veins and atria. This initiates the changes that are critical to precipitation of heatstroke, such as the reduction in BF_{sk}.[76] Possible interaction between baroreflexes and thermal reflexes is an important consideration.[77] The thermoreceptor system appears somewhat more potent than the baroreceptor system.[78] Normally, CVP is lowered as a result of the high BF_{sk}. That is, at this early stage, BF_{sk} determines CVP, whereas at the final stage of severe heat stress, CVP controls BF_{sk}.[77–79]

To maximize heat loss from the skin, hyperthermic blood should pass only slowly through the cutaneous circulation. Vasodilation and venodilation of the skin provide a highly compliant circuit in the skin, which, owing to the large volume of blood that can be stored in skin, reduces venous return and lowers CVP during heat stress.[72,73] Excessively low CVP, however, leads to reduced cardiac filling, re-

duced stroke volume, and reduced cardiac output. Experimental maintenance of the central blood volume during heat stress increases stroke volume and cardiac output.[80,81] This observation indicates that the defense of CVP, acting via the low-pressure baroreceptors in the thoracic region, causes the late reduction in both skin blood flow and skin blood volume during severe heat stress.

To provide adequate skin perfusion and maintain heat tolerance, it is necessary to sustain elevated cardiac output, and, hence, stroke volume.[72,82–84] Classic heatstroke and exertional heatstroke (discussed later) may differ in this regard, among others (Table 5-2): in exertional heatstroke, stroke volume increases with acute heat stress and exercise, whereas in chronic heat stress (ie, classic heatstroke), stroke volume is initially reduced. Interested readers can consult the 1986 article by L. C. Senay, Jr., in the *Yale Journal of Biology and Medicine*[85] for further information on classic heatstroke.

This discussion describes the physiology of humans and most animals. However, because of their frequent use in experimental studies, it should be mentioned that in rats, CVP is not the critical determinant of heat tolerance.[86] The reason for this is not known.

Stress Hormones

During the Archeozoic period, organisms developed an immune system that destroys microorganisms. This nonspecific immunity is basic to all phyla today. In the original immune mechanism, the

TABLE 5-2

COMPARISONS BETWEEN CLASSIC AND EXERTIONAL HEATSTROKE

Patient Characeteristics	Heatstroke	
	Classic	**Exertional**
Age group	Elderly	Men (15–45 y)
Health status	Chronically ill	Healthy
Concurrent activity	Sedentary	Strenuous exercise
Drug use	Diuretics, antidepressants, antihypertensives, anticholinergics, antipsychotics	Usually none
Sweating	May be absent	Usually present
Lactic acidosis	Usually absent, poor prognosis if present	Common
Hyperkalemia	Usually absent	Often present
Hypocalcemia	Uncommon	Frequent
Creatine phosphokinase/aldolase	Mildly elevated	Markedly elevated
Rhabdomyolysis	Unusual	Frequently severe
Hyperuricemia	Mild	Severe
Acute renal failure	< 5% of patients	25%–30% of patients
Disseminated intravascular coagulation	Mild	Marked; poor prognosis
Mechanism of heatstroke	Poor dissipation of environmental heat	Excessive endogenous heat production and overwhelming of heat-loss mechanisms

Adapted with permission from Knochel JP, Reed G. Disorders of heat regulation. In: Kleeman CR, Maxwell MH, Narin RG, eds. *Clinical Disorders of Fluid and Electrolyte Metabolism.* New York, NY: McGraw Hill; 1987: 1212.

invader's cytoplasm was chemically destroyed by active oxygen species, including free radicals, produced by the host. During evolution over geological time, a rich variety of defense mechanisms arose with great variation in use and importance of particular mechanisms, and in which specific antibodies play no part.[41] In modern mammalian species, specialized inflammatory cells such as macrophages and natural killer (NK) cells are especially enriched in the enzymes and chemical pathways that produce the toxic free radicals; they also contain additional pathways that allow these inflammatory cells to recognize invaders and penetrate tight epithelial junctions, so the invaders can be easily reached and destroyed. The immune cells are activated by means of circulating and locally produced TNF and IL-1 as part of a stress reaction mediated by the hypothalamus. Natural selection generated a common stress reaction in vertebrates that is activated by stresses other than infections, such as hyperthermia, and activates pathways that produce toxic species, which may now be inappropriate and even toxic to the host. Interested readers can find further information in S. L. Gaffin's chapter in *Adaptation Biology and Medicine* (1999),[42] and M. J. Kluger's 1991 article in *Physiological Reviews*.[43]

The hypothalamus is ordinarily programmed to defend a T_c of approximately 37°C by initiating conventional thermoregulatory vasomotor effector responses to the skin for moderate hypothermia. The presence of circulating pyrogens raises this set point, resulting in fever, which is the defense of an elevated T_c, and not hyperthermia, which is a consequence of excess heat load or insufficient cooling capacity. When the hypothalamus receives thermal information that it interprets as severe hyperthermia, then it initiates a generalized programmed response. Within the anterior pituitary of an unstressed person are normally produced up-regulators of protein and hormone synthesis. These include prolactin, growth hormone (GH), and gonadotropin-releasing hormone (GRH). GRH induces follicle stimulating hormone, luteinizing hormone, and testosterone. Severe stress causes the hypothalamus to secrete corticotropin-releasing factor (CRF), which circulates to the anterior pituitary and down-regulates the synthesis of GH, GRH, and prolactin. At the same time, CRF up-regulates the immunosuppressors ß-endorphin, melanocyte-stimulating hormone, IL-1, TNF, and adrenocorticotropic hormone (ACTH). ACTH, in turn, circulates to the adrenal gland and up-regulates the secretion of the immunosuppressor, cortisol. IL-1 and TNF are part of a positive feedback loop, which causes further secretion of CRF.

Overall, hyperthermic stress initiates pathways that lead to the secretion of immunosuppressors, which can reduce overall resistance to infections. The same pathways are also activated in response to other severe stresses such as exercise, sleep deprivation, or infection and cause further secretion of CRF, lowering overall resistance to heat stress, and increasing the incidence of heat illnesses in general. Simultaneous severe stressors, such as those that occur during combat, become approximately additive in their immunosuppressive effects. (For a review of the effects of psychological factors on the immune system, interested readers should see S. Cohen and T. B. Herbert's 1996 article in the *Annual Review of Psychology*.[87]) Consistent with the general effect of stressors on the immune system, during World War II and the Korean War, the incidence of diseases increased with the intensity of combat.[88]

Both IL-1 and TNF are involved in the regulation of sleep; they are somnogenic, in addition to their role in inflammation. IL-10 inhibits the synthesis of these cytokines and reduces the amount of rapid eye movement sleep in mice. Therefore, rises in IL-10 by stress may alter sleep pattern and times, ultimately increasing the risk for heat illnesses.[89]

Strenuous exercise by athletes increases IL-1, TNF, and IL-6, and decreases interferon-γ (IFN-γ).[90] Changes in cytokine concentrations from exercise may persist for hours to days, and render a soldier at risk to heat the day *after* severe exercise.

Arachidonic Acid–Induced Dysfunctions

TNF and IL-1 exert their toxicity through the production of highly toxic phospholipase A_2 (PLA_2). The binding of TNF and IL-1 to their specific membrane receptors activates G-proteins, which increases the intracellular concentration of cyclic adenosine monophosphate (cAMP), which in turn, stimulates the formation of PLA_2 phosphatase (PLAP). This enzyme then activates the membrane toxin, PLA_2, which hydrolyzes membrane phospholipids, forming small lesions in plasma membranes. Some of those membrane phospholipids contain omega-6 fatty acid bases, producing AA on hydrolysis. AA is a *key metabolite,* which may be acted on by either of two enzymes, lipoxygenase or cyclo-oxygenase, leading to the production of toxic products through two main pathways (Figure 5-3). The lipoxygenase pathway leads to the production of the toxic leukotrienes B_4 (LTB_4) and D_4 (LTD_4), and the cyclo-oxygenase pathway to the production of

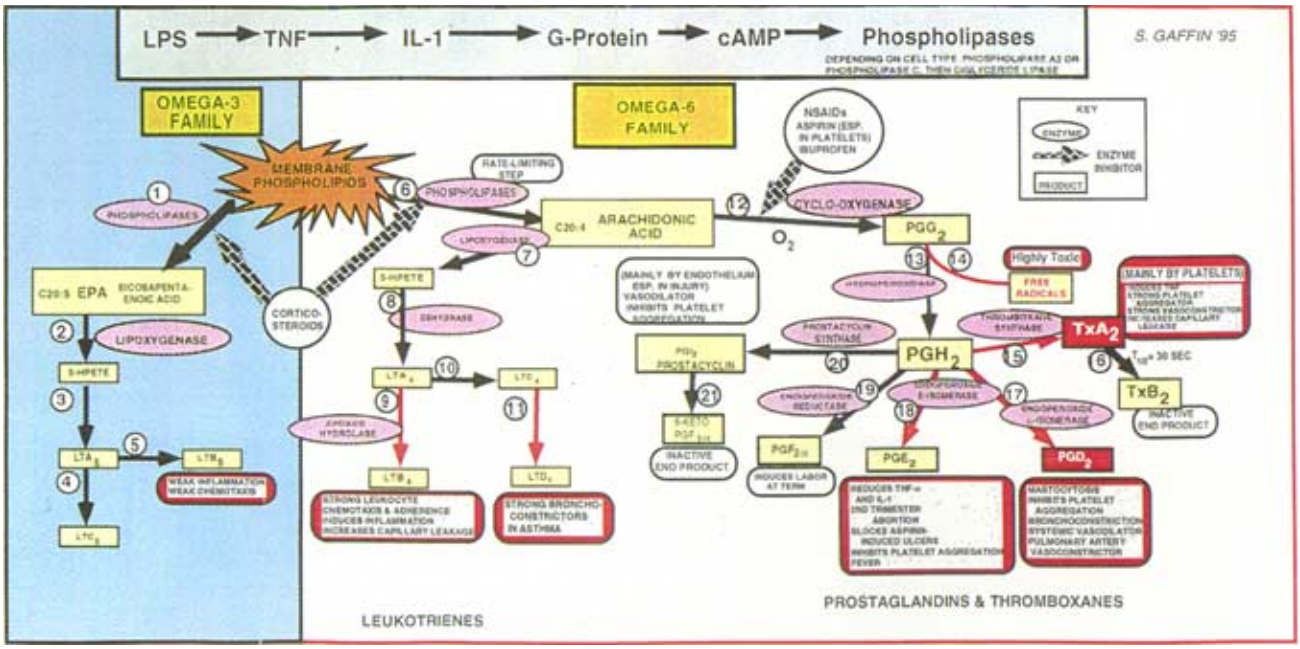

Fig. 5-3. Eicosanoid pathways; metabolism of omega-3 and omega-6 fatty acids induced by lipopolysaccharide (LPS) or cytokines. LPS binds to the CD14 receptor on macrophages, leading to the induction of tumor necrosis factor (TNF) and interleukin 1 (IL-1). These lead to the activation of membrane-associated phospholipase A_2, a critical step in the pathophysiology.

Omega-3 Family. Membrane phospholipids of the omega-3 family are (1) hydrolyzed by phospholipase A_2 into eicosapentaenoic acid (EPA), which is rapidly converted (2–4) into the benign leukotriene C_5 (LTC_5) or (5) weakly inflammatory leukotriene B_5 (LTB_5).

Omega-6 Family. Those phospholipids of the omega-6 family are (6) hydrolyzed by phospholipase A_2, into arachidonic acid, which may be converted by (7) lipoxygenase and (8) dehydrase into the powerful and toxic leukotrienes (9) LTB_4 and (10, 11) LTD_4. Arachidonic acid may also be converted by (12) oxygen and cyclo-oxygenase into prostaglandin G_2 (PGG_2). This is converted with (13) hyperperoxidase into the pivotal PGH_2, producing (14) toxic free radicals. PGH_2 may be converted by (15) thromboxane synthase into the highly toxic, but (16) short-lived thromboxane (TXA_2), or by (17) endoperoxide a-isomerase into the highly toxic PGD_2. Other prostaglandins formed vary (18–21) from mildly toxic to beneficial.

Adapted with permission from Hubbard RW, Gaffin SL, Squire DL. Heat-related illnesses. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. St. Louis, Mo: Mosby; 1995: 182.

toxic free radicals, thromboxane A_2 (TXA_2), and the prostaglandins D_2 (PGD_2) and E_2 (PGE_2).[91] Artificially raising the concentration of the intermediates PLAP or AA increased the plasma concentration of the cytokines, IL-1 and TNF. Therefore, not only can PLA_2 and AA synthesis be stimulated by cytokines, but cytokine concentrations can also be up-regulated by PLAP and AA in an inflammatory response.

LTs and PGs produced from AA cause the following toxic effects:

- LTB_4 and LTD_4 cause leukocyte adherence; induce inflammation and increase capillary leakage, thus reducing plasma volume; and cause the strong bronchoconstriction of asthma.[92,93]
- PGD_2 and PGE_2 cause mastocytosis, bronchoconstriction, vasoconstriction of the pulmonary artery, fever, abortion, and inhibit platelet aggregation.
- TxA_2 induces TNF, increases capillary leakage, and is a strong vasoconstrictor.
- The oxygen free radicals produced in the cyclo-oxygenase pathway damage cellular DNA and may cause cell death.

To a person in shock or with other circulatory disturbances, such agents could convert a serious but treatable condition into a lethal one. For more complete discussions of these pathways, see the 1988

articles in the *New England Journal of Medicine* by J. A. Oates and colleagues[94,95] and the 1992 article by G. Bottoms and R. Adams in the *Journal of the American Veterinary Medical Association*.[96]

The production of the key compound, AA, depends on the activation of PLAs and the presence of omega-6 fatty acids in plasma membranes.[97,98] The PLAs break down phospholipid esters of fatty acids, including the omega-6 fatty acids, which predominate in cell membranes of an individual on a normal Western diet.[99,100]

Fever and Infections

Normal T_c is maintained by a variety of homeostatic pathways. When there is a large rise in metabolism or in absorption of heat from the environment relative to heat-dissipating mechanisms, the rise in T_c is known as hyperthermia. Fever is different. Fever occurs when the temperature set point of the hypothalamus is reset by the actions of pyrogens released by bacteria or viruses,[101] which provoke the secretion of cytokines IL-1, TNF,[102,103] IL-2, or IFN-γ.[104] These secreted cytokines, in turn, alter homeostatic pathways to defend a new, higher temperature, or thermal set point.

A number of disease states lead to the induction of cytokines, which alter a variety of cellular enzymatic paths and functions. Physiologically, cytokines cause nausea, vomiting, diarrhea, fever, and at least one cytokine, IL-1, causes the feeling of tiredness and somnogenesis during many diseases. Several observers have noted that persons suffering from intercurrent illnesses, even very minor ones, are at much greater risk of heat illnesses.[105] Neutralizing antibodies to IL-1 and TNF are present in the sera of normal and sick individuals and may play a role in the regulation of those cytokines and of fever.[106]

Aspirin has long been known to reduce fever. Aspirin-like compounds inhibit cyclo-oxygenase (which synthesizes PGs from AA) and, by so doing, interfere with the fever induced by IL-1.[107] Circulating pyrogens eventually reach the thermoregulatory control center in the anterior hypothalamus of the brain, and induce the synthesis of PGs.[108,109] At the onset of a fever, patients often feel chilled and shiver (which increases metabolic rate), show reduced BF_{sk} (which reduces cooling), and may wrap themselves with blankets (which improves their insulation by behavior) to establish a new, elevated, and *preferred* T_c.[110] Once a new set point temperature is established, the thermoregulatory center uses all available homeostatic mechanisms to maintain it. Therefore, attempting to lower the elevated T_c *of fever* by active cooling measures causes sensations of extreme discomfort and violent shivering. *If attempts at cooling cause chills and violent shivering in a patient with suspected heat illness, a coexistent infection or disease should be suspected.*

The set point temperature mediated by PGs is responsible for fever, pathological elevations in temperature, and temperature elevations related to stress; and it contributes to normal circadian variations in temperature, at least in nonhuman animals.[111–114] Treatment for fever should be directed at agents that block the action of the pyrogen at the hypothalamic receptor sites (although some pyrogens act independently of PGs).[115,116] These include aspirin, acetaminophen, indomethacin, ibuprofen, and other newer nonsteroidal antiinflammatory compounds.[117]

Should antipyretic therapy be routine during an episode of fever? Because normal febrile response is generally self-limited in both magnitude and duration, there is no urgent need for active pharmacological measures.[118] Fever has long been recognized both as a manifestation of disease and potentially a serious problem in itself.[119] However, it is both illogical[120] and usually ineffective[121] to actively cool a patient with true fever, and antipyretic therapy should not be routinely instituted for every febrile episode.[122,123] Moderate (but not extreme) fever aids in host defense by promoting phagocytic engulfment of microorganisms and chemotaxis *up to approximately 40°C (104°F)*.[124,125] Routine antipyretic therapy for moderate fever may be counterproductive.[126] Rather, treatment should be based on the relative risks in individual cases,[126–128] particularly in those patients whose high T_c is not due to exercise, and should be reevaluated if expected benefits are not achieved.[120]

In simple cases of mild heat stress or hyperthermia, when exercise ceases and environmental heat is no longer absorbed, then body temperature will spontaneously fall toward normal levels.[102] However, observations in experimental animals suggest that in extreme hyperthermia (> 40°C) administration of vasopressin or melanotropin or their analogues may prove to be beneficial, by acting centrally to suppress temperature elevation.[129,130]

Lipopolysaccharides and Cytokines

There appears to be a relationship between hyperthermia, reduced splanchnic blood flow, and lethal heatstroke that mimics sepsis. To appreciate this relationship, it is necessary to consider the contents of the intestinal lumen and the likely results

of its leaking into the systemic circulation.[131] In healthy individuals, the intestinal lumen from the jejunum to the sigmoid contains large amounts of Gram-negative bacteria and LPS, the highly toxic endotoxin coat that sloughs from its walls.[132] LPS can cause death at plasma concentrations as low as 1 ng/mL. Whereas LPS remains within the intestines, no toxicity occurs; however, when it enters the blood, then pathology similar to sepsis ensues.[133] LPS is not a contact poison, but instead, induces in a variety of cell types excessive amounts of toxic inflammatory agents, including TNF and IL-1. These two can themselves produce most of the symptoms of septic shock (except DIC, which is caused directly by the activation of a clotting factor by LPS). At low concentrations, LPS alone, or in combination with TNF and IL-1, causes fever, somnogenesis, nausea, vomiting, diarrhea, and headache, but at high concentrations can cause vascular collapse, shock, and death.[134,135] At the onset of heat illnesses, there is probably a specific etiology for each case, but one that is unrelated to LPS. However, at a certain combination of elevated temperature, hydration status, and time, splanchnic blood flow is reduced so severely as to cause ischemic damage to the gut wall, which is followed by translocation of LPS from the gut into the circulation. The patient then becomes endotoxic as well as hyperthermic.[65,66]

Besides altering AA pathways and forming active oxygen species, IL-1 causes cachexia. IL-1 causes muscle proteolysis by inducing branched-chain α-keto acid dehydrogenase, a rate-limiting enzyme for the oxidation of amino acids in skeletal muscle. As this enzyme increases in concentration, amino acids are progressively oxidized, leading to the breakdown of muscle proteins. TNF induces IL-1 and also depolarizes the plasma membrane in skeletal muscles.[136] Consequently, alterations in physical performance are expected when TNF is present.

There is a reciprocal relationship between the brain and the immune system. Cytokines such as IL-1_β that are produced by immune cells in the periphery also alter neural activity.[137] IL-1_β is a chemical signal between immune cells during infection and injury, and is also a critical mediator of immune-to-brain communication. IL-1_β can activate sensory nerves to trigger sickness responses by the brain including fever, reduced food and water intake, increased sleep, exaggerated pain, and the like. IL-1_β in the periphery can increase IL-1_β within the CNS, which in turn, initiates classic sickness responses.[138] Furthermore, the integrity of the blood–brain barrier is normally compromised in several small areas of the brain, especially in the hypothalamus.[137,139–143] Thus, it is possible that cytokines produced elsewhere may circulate, cross the deficient blood–brain barrier in those regions, bind to specific cytokine receptors present in hypothalamic neurons, and alter CNS function.[144,145] Research related to the function of barrier systems within the body during heat and exercise appears clearly warranted.

Septicemia has been characterized by fever, vascular collapse, DIC, shock, and death. Fine[65] and others[133] noted almost identical symptoms in a heatstroke victim and suggested that part of the pathophysiology of heatstroke involved the same mechanisms as those of septic shock. Because most of the symptoms of Gram-negative bacteremia are produced by LPS,[134,135] they suggest that LPS participates in the pathophysiology of heatstroke.[65] Experiments in nonhuman primates suggest that LPS becomes increasingly important as a pathological agent as T_{re} approaches 43.5°C, but above this temperature direct thermal damage to brain tissue is probably most important.[146]

The intestinal walls are rendered reversibly permeable to LPS by reducing the intestinal blood flow to below approximately 50% of normal. Hyperthermia leads to a reduced splanchnic blood flow, and when severe enough, LPS leaks out of the gut lumen at a high rate and principally enters the portal vein. LPS may be removed from the circulation by the liver, or it may bind to a number of protective plasma proteins such as LBP, anti-LPS antibodies (anti-LPS), or soluble CD14, a fragment of the LPS binding ligand present on macrophages. When macrophage CD14 is activated by LPS, the production of TNF and IL-1 is induced, causing symptoms. Although many cell types produce cytokines in response to LPS challenge, bound macrophages within the liver may be the major source of the cytokines in circulation during endotoxemia.[147]

Maintenance of health requires adequate barrier function of the intestinal wall. Virtually any significant damage of the gut wall by ionizing radiation, trauma, viral scouring, bacterial overgrowth, and parasites permits LPS to leak into the systemic circulation. Elevated LPS leakage is also caused by major blocks of the splanchnic arteries (eg, occlusion of the superior mesenteric artery, severe hemorrhage or hypotension, and even temporary reductions in splanchnic blood flow caused by the reflex sympathetic activation from the breathing of a hypoxic gas mixture).[148] Probably, most stimuli leading to the activation of the sympathetic nervous system, including excitement, fear, and infection, can reduce splanchnic blood flow and may, especially in concert

with additional stressors, lead to endotoxemia. Cytokines may be produced by mechanisms other than LPS activity. IL-1 is also produced as a result of sleep deprivation, and may, in principle, contribute to endotoxicity.[149]

Heatstroke in experimental animals leads to an endotoxemia at 42°C to 43°C, and probably at a lower T_c in exercising humans, which probably results from a combination of ischemic injury and direct thermal damage to the gut wall.[150] The temperature at which heatstroke becomes irreversible depends on a complex relationship among T_c, exercise intensity, time, original state of hydration, and health. At temperatures below 42°C, heatstroke probably involves classical pathways not involving LPS, but at 43°C and higher, for a person at rest, clinical endotoxemia may be a complication.

Runners who collapsed during or at the end of an 89.5-km ultramarathon run on a warm day, all appeared superficially similar. However, based on their plasma LPS levels, they could be classified into two groups.[150] Most (80%) showed elevated levels of LPS, including two in the 1 ng/mL lethal range. (NB: For short periods the body can tolerate much higher LPS concentrations. The widely reported lethal concentration of 1 ng/mL refers to a long-term plasma level after all host defenses are activated.[132,151]) The second group, with lower, normal levels of LPS, showed better performance (ie, faster times to the finish line), had higher levels of natural anti-LPS immunoglobulin G (IgG; almost everyone has some measurable anti-LPS IgG in their plasma[152]), and lower morbidity indexes (combined scores of nausea, vomiting, diarrhea, and headache). In other words,[150]

- those with normal low levels of LPS, who usually had high plasma levels of anti-LPS IgG, recovered within 3 hours, and
- those with elevated levels of LPS, who usually had low levels of anti-LPS IgG, required up to 3 days to fully recover.

In a pilot study, the plasma of surviving heatstroke patients had been cleaned by 3 to 5 days of venovenous hemofiltration with a polyacrylonitrile hemofilter that nonspecifically binds to LPS, and exchanged with 40 units of fresh frozen plasma, in addition to conventional therapy. Those treated with conventional therapy alone died.[153] The exchanged plasma may have contained sufficient endogenous anti-LPS antibodies to neutralize any LPS remaining in the blood. Overall, the anti-LPS antibodies appeared protective.

The question is, Who has high levels of anti-LPS? A study of triathletes suggested that very hard training increased levels of anti-LPS, because those who had the highest combined miles of running, bicycling, and swimming 3 weeks before the race had the highest levels of anti-LPS.[154] Untrained thoroughbred horses had low levels of anti-LPS, which rose during the training period and fell immediately after a race.[155] It may be that a part of the benefit of physical training is the increase in levels of natural anti-LPS antibodies. This possibility is currently under active investigation.

Classic Heatstroke and Cytokines

Several studies of heatstroke victims during the Hajj in Saudi Arabia indicate that cytokine levels become elevated during heatstroke. However, these patients are mainly elderly, classic heatstroke victims and not the much younger, exertional heatstroke victims seen during combat and training in hot weather. Eight of 17 victims admitted to a hospital within 2 hours from the onset of heatstroke, suffering from delirium, confusion, and coma, had elevated levels of plasma LPS, TNF, and IL-1.[132,156–158] Those cytokines probably induced PGs, which exacerbated the hyperthermia of heatstroke, because IL-1, in particular, is known to cause fever.[159]

The rapidity of cooling and entry to a hospital intensive care unit is important in determining survival of heatstroke patients.[59,60] Heatstroke mortality is related to the total heat stress (ie, during heating *and* cooling). This speed may be important because of the time required for the production of cytokines, which is in the range of minutes to hours.

On the battlefield in summer, soldiers expend energy at a high rate, leading to rises in T_c despite substantial sweat losses. In addition to the usual condition of moderate hypovolemia, a number of other factors capable of reducing BF_{sk} interact (eg, fear, excitement, and sleep deprivation). These factors may be exacerbated from any additional LPS entering the circulation as a result of mild or subclinical salmonellosis or shigellosis from drinking contaminated water.[160]

The presence of circulating LPS, whether translocated from the gut or from mild infection, can alter the activities of normal heat dissipation mechanisms and exacerbate the pathophysiology that simple elevated T_c causes. Injection of LPS into healthy subjects at elevated ambient temperature caused a PG-mediated complete cessation of sweating from 1 to 1.5 hours after the injection, lasting from 7 to 60 minutes.[161] It is not known whether this

cessation of sweating is a direct effect or mediated via other mechanisms such as baroreceptor override of peripheral circulation and vasodilation. The subjects also showed the symptoms of fever, headache, fatigue, nausea, skin vasoconstriction with "gooseflesh" appearance, sensation of chill, and hypotension.

Exercise and Cytokines

It is possible for circulating cytokines to increase with exercise and independent of any rise in LPS. Strenuous exercise leads to mild increases in plasma IL-1, TNFα, IL-6, IFN, and soluble IL-2 receptor.[90] Eccentric exercise, which usually damages skeletal muscle, causes a rise in IL-1.[162]

In general, during severe exercise, B and T lymphocytes and subpopulations of NK cells are recruited into the blood from lymph nodes. Both the number and the activity of NK cells—a heterogeneous group—are enhanced.[163] The NK cell subsets that increased in number the most were those most responsive to IL-2 activity from 2 to 4 hours after exercise. Prostaglandins released by monocytes during severe exercise suppress NK activity for several hours. Afterward, NK activity returns to baseline. During that several-hour period of immunodepression, some studies suggest that there is an elevated susceptibility to viral or bacterial agents.[163] Because aspirin is PG-mediated, its administration would possibly prevent this temporary immunosuppression.

Monocytes produced in the bone marrow are released into the circulation in response to interleukins and LPS.[164] They are phagocytic and produce cytokines. Nevertheless, circulating monocytes are actually immature cells and have half-lives of only 2 to 3 days in transit to tissues. Once they have reached their tissue sites, they mature into the larger and more capable macrophages, with half-lives measured in months. Macrophages exist in different functional states, depending on local signaling such as the ranges of local concentrations of cytokines and stress hormones. Macrophages may have low functional activities, be up-regulated and primed for further activation as in inflammation, or may be fully activated.

As described earlier, acute, moderate exercise causes a transient increase in monocytes, which lasts minutes to hours, but exercise training does not increase the monocyte count in resting subjects.[164,165] On the contrary, exhaustive exercise for 7 consecutive days decreased resting monocyte counts. However, over a period of days or weeks, physical training alters metabolic parameters of macrophages, such as increased insulin-mediated glucose uptake and O_2 consumption[164,166] and an increased affinity of insulin receptors.[167]

Overtraining may be very harmful. Quantitative laboratory determinations of blood parameters will, hopefully, eventually be able to show at what point any benefit from exercise training becomes less important than the disadvantages of immunosuppression, and indicate, therefore, when exercise should be curtailed.[168]

HEAT ILLNESSES

Heat illnesses and heat-related illnesses may be induced by hyperthermia, dehydration, salt depletion (from sweat loss or inadequate diet), exercise, and hyperventilation, in any combination. Before 1950, the mortality rate of heatstroke was 40% to 75%, with long-term survival directly related to the speed with which cooling and volume therapy were instituted. Because hyperthermia leads to subjective feelings of weakness and exhaustion but is not actually painful, highly motivated soldiers may persist exercising in the heat even when they feel exhausted. As a result, they may develop a wide range of clinical illnesses, from fatigue to heat exhaustion to heatstroke. The distinction between various clinical diagnoses is somewhat artificial, and a differential diagnosis may be difficult to establish in the field or even in a hospital (Table 5-3). Heat exhaustion and heatstroke are two extremes of a continuum of disorders, and they probably share many common pathways and factors. The boundary between heat exhaustion and heatstroke is usually arbitrarily defined as a T_c of 41°C plus alterations in sensoria. However, the seriousness of the illness is an *individual* matter for each patient, and if a person collapses during or after exercise with a T_{re} higher than 39°C, the physician should consider possible heatstroke, institute cooling procedures immediately, and transport the patient to a hospital. Moreover, during the period of transporting an undiagnosed patient to a hospital, T_{re} may have fallen to 39°C or even less, which might render doubtful a hospital diagnosis of heatstroke. Typical symptoms of heatstroke include belligerence toward friends and authority, a vacant stare, confusion, babbling, and aimless running or crashing into objects.

Until relatively recently, cessation of sweating was considered critical to the diagnosis of heatstroke; today, however, it is recognized that sweating may persist until late in heatstroke. Documentation of

TABLE 5-3

SIGNS AND SYMPTOMS OF SALT- AND WATER-DEPLETION HEAT EXHAUSTION

Signs and Symptoms	Salt-Depletion Heat Exhaustion	Water-Depletion Heat Exhaustion	Dilutional Hyponatremia
Recent weight gain	No	No	Yes
Thirst	Not prominent	Yes	Sometimes
Muscle cramps	In most cases	No	Sometimes
Nausea	Yes	Yes	Usually
Vomiting	In most cases	No	Usually
Muscle fatigue or weakness	Yes	Yes	No
Loss of skin turgor	Yes	Yes	No
Mental dullness, apathy	Yes	Yes	Yes
Orthostatic rise in pulse rate	Yes	Yes	No
Tachycardia	Yes	Yes	No
Dry mucous membranes	Yes	Yes	No
Increased rectal temperature	Yes	In most cases	No
Urine Na^+/Cl^-	Negligible	Normal	Low
Plasma Na^+/Cl^-	Below average	Above average	Below average

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only mild elevation in T_c should not preclude the diagnosis of heatstroke because T_c may have fallen before measurement. Unless an alternative etiology is obvious, a previously healthy soldier who collapses after physical exertion in hot weather should be diagnosed with exertional heatstroke, even if body temperature was not found to be markedly elevated several hours after collapse.

The early symptoms of heat illness are often unrecognized, and casualties may present to a medical treatment facility with nausea and visual disturbances that mimic the prodrome of a migraine headache. Failure to attend to these symptoms puts an individual at great risk for thermal injury. Individuals who anticipate exposure to hot environmental conditions should be thoroughly versed in the manifestations of heat illness.

Heat Exhaustion

Heat exhaustion is the most common clinical syndrome resulting from heat stress and exercise. It tends to develop over several days of exposure, thus providing ample opportunity for electrolyte and water imbalance to occur. However, under conditions of rapid sweating (high temperature, humidity, or work rates), heat exhaustion can occur in hours rather than days. Three forms of heat exhaustion are recognized: (1) water-depletion heat exhaustion, better known as dehydration; (2) salt-depletion heat exhaustion; and (3) the combination of salt depletion and dehydration. The form of heat exhaustion that manifests is determined by the relative and absolute losses of water or salt in sweat. Victims usually show common symptoms, including intense thirst (although not necessarily in salt-depletion heat exhaustion), fatigue, weakness, anxiety, restlessness, mental confusion, and poor muscle coordination. In salt-depletion heat exhaustion, the body temperature may remain normal or even subnormal. But when victims stand up, blood pressure falls and they may faint.

In general, the loss of plasma volume is more severe in relatively unacclimated individuals because their sweat contains relatively high concentrations of salt. On the other hand, in those who are relatively more acclimated and are secreting a more dilute sweat (approaching a water deficit), there is a greater rise in plasma osmolality and sodium, and

thirst is greater. For further information, see Chapter 8 in *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*.[169]

Water-Depletion Heat Exhaustion

The symptoms of water-depletion heat exhaustion include hyperthermia, thirst, hyperventilation, tachycardia, fatigue, weakness, discomfort, and some impairment of mental state such as confusion, poor judgment, disorientation, paraesthesia, and muscular incoordination. Delirium and coma occur in advanced cases. A patient may collapse with either a normal or an elevated temperature (in severe cases, around 40°C). Spontaneous cooling is normal if the patient is sweating. Oliguria is common and urinary contents of sodium and urea may reflect normal renal conservation mechanisms. Clinical evaluation is necessary, as *serum sodium is always elevated* and may approach 170 mEq/L in severe dehydration. If left untreated, the dehydration could lead to heatstroke. Particular caution must accompany the lowering of serum sodium levels to avoid the serious risk of cerebral edema. The cautious administration of 1 to 2 liters of normal saline over 3 to 6 hours is followed by the judicious administration of 5% dextrose. *Under no circumstances should serum sodium be permitted to fall more rapidly than 2 mEq/h.*[170]

Salt-Depletion Heat Exhaustion

The hyponatremia and hypochloremia of salt-depletion heat exhaustion usually occur in nonacclimatized individuals who have not fully developed salt-conserving mechanisms. Therefore, it may even occur in temperate weather, such as in spring; or a cold environment, such as Alaska even while one is encapsulated in heavy clothing, when extraordinary work periods cause large sweating losses of salt and water. Even prolonged insensible sweat (and electrolyte) losses that are unreplaced, such as waiting in the sun for delayed transportation, predispose to salt-depletion heat exhaustion if cumulative salt losses are significant. In contrast, predominantly *water*-depletion heat exhaustion, or dehydration, tends to occur when water intake is inadequate owing to voluntary dehydration or the discredited, misinformed concept of deliberate restriction, "water discipline." Among 44 summertime hikers who requested medical help in Grand Canyon National Park in June through September 1993, and whose serum samples were analyzed, 7 had frank hyponatremia with serum sodium levels lower than 130 mmol/L, and had clinically significant symptoms: 3 had grand mal seizures, 2 had other major CNS disorders, and 2 had minor neurological symptoms.[171]

Casualties with salt-depletion heat exhaustion present with hyponatremia and hypochloremia. Symptoms include profound weakness, giddiness, nausea, vomiting, diarrhea, and skeletal muscle cramps in a high percentage of cases (50%–70%). *These patients are much sicker* than those with simple muscle cramps. The skin is pallid and clammy, and the T_c may be normal or even subnormal. Orthostatic hypotension, tachycardia, and syncope are common. Owing to dehydration, laboratory values reflect volume depletion and hemoconcentration. Mild cases of hyponatremic heat exhaustion are generally well managed with the administration of salt and water. Patients who are confused, disoriented, orthostatic, febrile, or vomiting require laboratory evaluations and should be seen by a medical officer.

Salt-Depletion Dehydration

In hot weather and when water supplies are adequate but food is being rationed, an insidious form of dehydration occurs. Over a prolonged period, the body's salt content is gradually depleted from sweating. The salt lost in sweat is not replaced, owing to inadequate dietary replacement. This condition is worsened by both the anorexia accompanying mild dehydration and any diarrheal disorder. Each would shorten the onset and increase the severity of this disorder, which is called *salt-depletion dehydration*. This disorder often contributes to the physiological strain seen in unacclimatized troops who are suddenly exposed to heat and exertion. Salt-depletion dehydration could be used as a weapon, by forcing the enemy to move during daylight hours while his food supplies are limited. For further information, consult Chapter 8 in *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*.[169]

Heat Cramps

Heat cramps are a frequent complication of heat exhaustion and occur in about 60% of casualties with heat exhaustion, but a preexhaustion period of exercise is also always required. Heat cramps are characterized by brief or intermittent, often excruciating, contractions of voluntary skeletal muscles of the legs, arms, and abdomen, and usually occur during or after prolonged work in the heat. The cramp consists of a painful, localized contraction that sometimes appears to wander over the affected muscle as adjacent muscle bundles contract. Cramps

of the rectus abdominus may cause frank abdominal rigidity. In general, cramps usually occur later in the day, sometimes while showering, and occasionally in the evening. The incidence has been estimated at about 1% of foundry workers exposed to a hot work environment. For further information, see Chapter 8 in *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*.[169]

The exact mechanism of heat cramps and other similar maladies, such as exercise-induced muscle cramps, nocturnal cramps, or even writer's cramp, is unknown. Heat cramps sometimes occur as the only complaint, with minimal systemic symptoms. Factors common to most reports of cramps are (1) a prior period of several hours of sustained effort, (2) heavy sweating in a hot environment, and (3) the ingestion of large volumes of water. Their appearance while showering suggests that muscle cooling may be involved. The consumption of large amounts of fluid with inadequate intake of salt, or in the presence of inadequate salt-conserving mechanisms (eg, lack of heat acclimatization), may explain the clinical findings of hyponatremia and hypochloremia, and reduced levels of sodium and chloride in the urine, which is indicative of negative salt balance. Both hyponatremia and hypochloremia disturb calcium regulation (by means of the Na^+/Ca^{2+} exchanger) within muscle cells, resulting in spontaneous contractions of the skeletal muscle, nausea, and vomiting.

Mild muscle cramps respond to a 0.1% NaCl solution taken by mouth (two crushed salt tablets in a liter of water) and, in severe cases, to intravenous (IV) administration of saline solutions (10–15 mL/kg body weight). Heat cramps tend to be rare in individuals who are fully acclimatized to their thermal and working environments and are almost nonexistent in the armies of Israel and India. It is not known why the incidence of heat cramps is only 50% to 70% as great as salt-depletion heat exhaustion. Patients with heat exhaustion are very sick, as opposed to those with heat cramps, in which a combination of heavy work and the drinking of large volumes of pure water tends toward dilutional hyponatremia and cramping.

Heat-Induced Tetany. Heat-induced tetany *at rest* is caused by hyperventilation in extremely hot environments, with consequent alterations in CO_2 concentrations and pH. Deliberate overbreathing can also induce this condition. The onset of symptoms varies from slight tingling sensations in the hands and feet to more severe carpopedal spasms. Symptoms are more related to the *rate* of change in CO_2, PCO_2, pH, and T_{re} rather than the absolute change. Classic heat cramps are distinguished from hyperventilation tetany because they (1) generally involve voluntary skeletal muscle subjected to prior exertion and (2) usually only affect a few muscle bundles at a time, persisting from 1 to 3 minutes, and with excruciating pain in severe cases.

The Syndrome of Cramps, Syncope, and Respiratory Alkalosis. There is a rarely described but not uncommon form of nonclassic heat exhaustion seen in military training settings obtained by running or speed marching in the heat (Table 5-4). Patients are characterized by hyperventilation, moderate-to-marked respiratory alkalosis, and syncope. Nearly all casualties have abdominal or extremity muscle cramps, and nearly 50% experience tetany with carpopedal spasm. The majority of these patients are not depleted of either water or salt. Hyperventilation with its resulting decrease in cerebral blood flow could account for a significant number of cases of exercise-induced syncope.

Recumbency, rest, cooling, and oral replacement of potential fluid and electrolyte deficits are usually recommended. If rebreathing of expired air is initiated to elevate arterial blood CO_2, it should be done with extreme caution because of its hypoxemic effect. Classic syncope is usually associated with postural hypotension, whereas heat exhaustion and heat cramps are not. The latter two are usually associated with water and electrolyte imbalance.

Syncope

Hyperventilation dizziness is a result of slight but prolonged increase in respiratory rate or tidal volume. It can accompany an increase in anxiety and represent some degree of generalized cerebrovascular vasoconstriction.

Vasovagal syncope is the cause of about 3% of emergency department visits and 6% of hospital admissions throughout a typical year. Syncope resulting from diminished venous return due to the pooling of blood in the peripheral circulation is caused not only by heat, but also by (1) psychological disturbances activating an autonomic vasodilation response; (2) reflexes initiated by heavy coughing, micturition, or pressure on an irritable carotid sinus; or (3) reduced vasomotor tone induced by hypotensive drugs or alcohol.

Heat syncope that is characterized by the transient or temporary loss of consciousness has its origins primarily in the cardiovascular system. In an upright and stationary person, blood is displaced into dependent limbs by gravity, which if combined with the filling of capacious peripheral veins to support heat transfer of the skin, can temporarily compromise

TABLE 5-4

CLINICAL DATA ON 17 HEAT CASUALTIES, FORT POLK, LOUISIANA, 12–23 JULY 1970

Soldier[*] No.	Activity	Syncopy (Fainting)	Cramps	Temperature[†] (°F)	Respiration Rate[‡] (Breaths/min)	Blood[§] pH	Na^+	K^+	Cl^-	Lactate	Creatinine
1	Marching	Yes	Abdomen	99.6	24	—	142	4.5	100	—	1.15
2	Running a mile	Yes	Legs, abdomen	98.4	30	—	145	4.2	102	—	1.50
3	Rifle range	No	No	99.4	24	—	143	4.7	102	—	
4[¥]	Marching, running	Yes	Hands	100.4	22	7.47	162	4.3	116	0.75	1.05
5	Marching	No	Legs, abdomen	102.4	35	7.50	141	4.3	102	1.48	1.84
6[¥]	Marching	No	Legs	100.0	22	7.70	152	4.3	—	—	1.40
7	Rifle range	No	Mild	100.0	22	—	140	4.1	100	1.36	1.30
8	Marching	Yes	Legs, abdomen	100.8	30	7.52	145	3.8	102	0.86	0.95
9	Marching	No	Abdomen	101.4	24	7.69	—	—	—	0.62	1.20
10	Marching	Yes	Chest	100.8	18	7.56	140	4.0	100	0.62	—
11[¥]	Marching	Yes	Tetany	101.5	30	7.44	160	4.9	106	1.44	1.30
12	Marching	Yes	Severe	100.6	30	7.71	130	4.5	100	—	1.15
13	Marching	No	Mild	98.6	26	7.77	141	4.0	104	0.91	1.30
14	Marching	Yes	Legs, abdomen	100.7	30	7.76	145	4.8	102	0.49	1.05
15[¥]	Rifle range	Yes	Legs, abdomen	101.0	32	7.66	148	3.7	104	1.05	1.65
16	Marching	Yes	Chest, legs	101.2	28	7.78	148	4.3	105	1.25	1.15
17	Marching	Yes	Abdomen	101.6	22	7.53	146	5.1	102	0.67	1.05

[*]All heat casualties among military recruits during training in the field presenting to a heat ward with heat exhaustion, heat syncope, or heat cramps
[†]Core (rectal) temperature was elevated (> 100.0°F) in only 11 of 17 casualties
[‡]All casualties had increased respiratory rates on admission to the medical treatment facility
[§]Serum electrolytes reported in mEq/L; creatinine in mg%; lactate in mmol/L
[¥]Hemoconcentration was seen in patients 4, 6 ,11, and 15
Data source: Boyd AE, Beller GA. Acid base changes in heat exhaustion during basic training. *Army Science Conference Proceedings*. 1972;1:114–125.

venous return, cardiac output, and cerebral perfusion. Prodromal symptoms include restlessness, sighing, yawning, and dysphoria. The hypotension results predominantly from vasodilation and bradycardia. Fainting and the assumption of a horizontal position is usually self-limiting. Although startling to onlookers and the casualty, the effects appear readily reversible with improved venous return. The casualty should be allowed to rest in cooler, shadier surroundings and offered replacement fluids. Orthostatic pooling and predisposition to syncope is counteracted by avoiding protracted standing in hot environments and by repeatedly flexing leg muscles to promote venous return. Patients should be advised to sit or

lie down at the onset of prodromal symptoms.

The incidence of syncope falls rapidly with increasing number of days in the heat, suggesting the importance of water intake and salt retention in preventing the disorder. Individuals who take diuretics would be at increased risk, and hypokalemia could lower blood pressure and blunt cardiovascular responsiveness. For further information, see Chapter 8 in *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*.[169]

Rhabdomyolysis

Rhabdomyolysis (1) is a complication of exhaustive exercise even in well-trained athletes, (2) often occurs in association with exertional heatstroke, and (3) probably would affect everyone if the exercise were sufficiently severe.[172–174] Rhabdomyolysis most commonly occurs in athletes who force themselves to continue to run maximally despite cramping pain, disorientation, and confusion. Rhabdomyolysis occurs more often in runners with white-collar jobs, and rarely in those with blue-collar occupations.[172] In this devastating disease, the skeletal muscle plasma membrane becomes so severely injured (the mechanism is unknown but may be due to depletion of high-energy phosphate reserves) that cell contents leak out into the blood, as shown by rises in concentration of muscle enzymes (eg, creatine kinase-MM, aldolase) and leads to myoglobinuria. But more severe problems may develop over the next 24 hours, including potentially fatal hyperkalemia, metabolic acidosis, DIC, and adult respiratory disorder syndrome (ARDS). Conventional thinking attributes the sequelae of rhabdomyolysis to the release of the contents of damaged muscle cells. However, similar pathophysiology can occur if LPS is released from the lumen of intestines owing to ischemic damage from a reduced flow of blood to the intestines due to severe exercise.

The severity of symptoms (except for eventual DIC) can vary greatly, from an asymptomatic rise in skeletal muscle enzymes, to muscle tenderness and weakness, to muscle pain, myoglobinuria, and even acute renal failure. The usual sequence of events is a *long* race on a warm, but not necessarily hot, day. During the race the runner appears pale and disoriented, with profuse sweating and hyperventilation, but is not necessarily hyperthermic. Over the next 12 to 24 hours, the patient virtually always shows DIC and may exhibit renal failure, hypocalcemia, and possibly ARDS. The DIC is worst on the third to the fifth day, and spontaneous improvement may commence after 10 to 14 days. If cellular damage is extensive, tissue lysis syndrome may present, and lead to infection, especially pneumonia, shock, myocardial ischemia, and progressive renal failure.[175] Extreme care is required in caring for these patients because they may manifest only minimal clinical abnormalities, yet show severe metabolic dysfunctions.

Summary of Heat Exhaustion

Classic heat exhaustion usually develops over several days and primarily involves electrolyte and water imbalance. Classic heat exhaustion results from a prolonged cardiovascular strain in the attempt to maintain normothermia. It is well documented that patients with classic heat illness often show fluid and electrolyte parameters within normal ranges.[173] This suggests that it is the prolonged duration of heat stress per se, and its consequent cardiovascular strain, that is the predominant cause of classic heat illness. The symptoms of heat exhaustion include various combinations of headache, dizziness, fatigue, hyperirritability, anxiety, piloerection, chills, nausea, vomiting, heat cramps, as well as heat sensations in the head and upper torso. A patient may collapse with either normal or elevated T_c, usually with profuse sweating and tachycardia, hyperventilation, hypotension, and syncope. Spontaneous body cooling can occur with heat exhaustion. This is not prominent in severe heatstroke, thus the clinical determination of heat exhaustion is primarily a diagnosis of exclusion.

The alternate forms of heat exhaustion are characterized by the type of fluid deficit, electrolyte deficit (primarily pure water and/or salt deficiency), or both; their underlying causes (prolonged heat exposure vs intense, short-term exertion); the intensity of the hyperthermia; and the absence or form of CNS disturbance. Intercurrent illness should be suspected if external cooling does not rapidly lower T_c to normal, or, conversely, external cooling precipitates severe shivering. Anecdotal experience suggests that approximately 20% of suspected heat exhaustion cases have some form of viral or bacterial gastroenteritis, especially if nonchlorinated water or ice has been consumed. The term "heat cramps" is a misnomer because the cramps do not usually occur during exposure to heat. The condition is usually precipitated by exhaustive work, ingestion of copious fluids, and cooling of the muscles.

Exertional Heatstroke

Severe exercise appears to cause local disruption of tissues, with the sloughing of tissue fragments into the blood. These fragments circulate, and acti-

vate complement factors of the blood clotting system, forming microscopic polymers that may be filtered out by the kidney and clog glomerular pores. Furthermore, complement activation primes monocytes (ie, renders them responsive to much lower than normal concentrations of activator) for further activation by LPS, or by fragments of tissue subsequently damaged, ultimately leading to DIC. Intense exercise can damage renal function so severely that the excretion rate of proteins rises by 100-fold and can even lead to a depletion of circulating antibodies.[176,177]

Bouchama and colleagues[158,178] further studied the immune systems of 11 patients with classic heatstroke. The responses were not uniform. Nine of the 11 heatstroke victims showed (1) a marked rise in leukocyte numbers that increased with increasing T_c, due to a large increase in the number of T suppressor/cytotoxic cells (CD8) and NK cells, and (2) substantial decreases in the number of T helper cells (CD4) and B cells. Catecholamines also rise during heatstroke; and because epinephrine administration causes leukocytosis with increased NK and CD8 cells, the rise in catecholamines induced by heat stress may have caused some of the changes in lymphocyte subpopulations seen in most heatstroke victims. In a comparison of at least 30 different assays of immune activity, the best marker for immune status was the determination of CD4, CD8, and their ratio.[179] In a study of 14 victims with classic heatstroke and heat exhaustion, the percentage of CD4 cells fell from 43% in controls to 31% in heat exhaustion to 15% in heatstroke; at the same time, CD8 rose from 28% to 42% to 46%, respectively; and the CD4/CD8 ratio fell from 1.54 to 0.738 to 0.326, respectively, indicating that heatstroke is associated with *a large relative rise in T cytotoxic/suppressor cell activity.*[178]

However, in the study by Bouchama and colleagues[158] mentioned above, 2 of those 11 heatstroke patients had a *decreased* number of lymphocytes. As previously described, hyperthermia increases cortisol,[180–182] and cortisol causes lymphocytopenia, the opposite effect of catecholamines.[157] To account for this reduction in lymphocytes in some patients, the effects of cortisol, rather than of catecholamines, were considered to be dominant. Overall, changes in concentrations of subpopulations of lymphocytes in heatstroke may depend, on an individual basis, on the relative rises in concentrations of catecholamines and cortisol, and individual sensitivities to them.

Classic Versus Exertional Heatstroke

Exposure to extremely high ambient temperatures caused a mean of 381 deaths per year in the United States from 1979 to 1996.[183] Nowadays, classic heatstroke is manifested in waves, for example, during the Hajj in Saudi Arabia or in some 500 deaths in Chicago in the 1995 heat wave. Victims of the heat among the Hajji usually collapsed while circling the shrine in Mecca. Following collapse they were usually rapidly cooled nearby and then taken to a local hospital, with T_{re} recorded as high as 47°C. At those high temperatures, survivors usually suffered neurological damage, with the extent of damage related to the duration of elevated T_{re}.[184]

As described by Dill,[185] a heat wave in Cincinnati, Ohio, in July 1936 resulted in hospital admissions of 44 heatstroke victims. Between 8 and 15 July the environmental temperature exceeded 38.3°C (101°F) every day and reached as high as 40.6°C. During that period the daily admission rates were 0, 0, 0, 4, 0, 8, 9, and 14.[186] The increasing number of admissions over time suggests that there was a progressive deterioration of the body with prolonged hot weather. This may be related to elevated cytokine levels. In a different heat wave in the region of Dallas, Texas, lasting from 24 June 1978 through 19 July 1978, half the cases (14 of 28) occurred from 13 to 15 July, but the first case occurred on 3 July and the last on 29 July.[173] Age was a factor, with a mean age of 70.5 years in the patients in Dallas; in a separate heat wave in Boulder City, Nevada, the victims had a mean age of 59 years. Furthermore, 5 of 28 patients in Dallas were older than 80 years, as were 3 of 44 patients in Boulder City. Alcoholism and degenerative diseases were contributing factors in both studies because they were present out of proportion to their numbers. The patients characteristically had high rectal temperatures and dry skin, and half of the patients in Boulder City and 24 of the 28 patients in Dallas were comatose. Twenty-three of the patients in Boulder City showed a fiery red skin rash over the body, particularly over the chest, abdomen, and back.[185] The most common presentation of the Dallas patients was that of respiratory alkalosis, often accompanied by metabolic acidosis.[73] All Dallas patients whose blood lactate was greater than 3.3 mmol/L suffered a poor outcome, whereas all those with initial lactate less than 3 mmol/L did well. That is, what would appear to be only *modest* elevations in blood lactate in exercise studies, become adverse prognostic indicators in classic heatstroke. Furthermore, 9 of 28 classic heatstroke patients arriving with normal serum potassium subsequently became hypokalemic, and all patients were hypokalemic at some point in their course.[173]

Classic heatstroke tends to be a disease of infants, the elderly, the alcoholic, and the infirm. Exertional

heatstroke is different; it typically affects young, healthy, and even euhydrated men and women during exercise, and is a syndrome involving hyperventilation and respiratory alkalosis.[187] The respiratory rate may reach 30 per minute, with a plasma pH as high as 7.78 and elevated arterial lactate.[187] Nevertheless, alcohol may be an indirect factor even in exertional heatstroke. In one incident, a football player became intoxicated the night before practice, and the coach punished him with 50 wind sprints. The athlete completed the exercises, walked off the field, went to his room, and was found dead 3 hours later.[188] Punitive exercises for an exhausted soldier should not be tolerated because the poor performance is probably due to heat exhaustion or impending heatstroke.

Risk Factors

Military Training

A 10-year retrospective study was published in 1996 of heat illnesses among US Marine Corps recruits at Parris Island, South Carolina.[189] A total of 1,454 cases were reported, 89% among men and 11% among women. Heat illnesses occurred during every month of the year, commencing with wet bulb globe temperatures as low as 18.3°C (65°F). In a different study in the British army, heat illness even occurred at an air temperature as low as 10.2°C (50.4°F).[190] Eighty-eight percent of the Parris Island cases occurred during late spring through fall, May through September. In the British army too, most cases occurred during the hot months, with the highest rates found in soldiers stationed in Hong Kong and Cyprus.[191]

At Parris Island, most heat illnesses occurred during the cooler early morning hours, with 60% occurring between 0700 and 0900 hours.[189] This is somewhat surprising, given that early morning is the time for strenuous exercises. That is, endogenous heat produced by exercise was more injurious than environmental heat. It is significant to note that the incidence of heat illnesses did not depend only on the immediate local ambient temperature but was strongly related to the peak temperature the previous day. That is, high environmental temperature caused some unknown biochemical or physiological changes, or both, in the soldiers that persisted for 24 hours and rendered them susceptible to heat illnesses. It is suspected, but not proved, that heat-induced, long-lasting cytokines or inappropriate apoptosis is responsible.

There was a gender difference in heat illness rates among the Marine recruits. Rates for women were higher during the early hot season (May) and rates for men were higher than those for women in the late hot season (September). Furthermore, when considering combined rates, there was a higher overall rate in May even though May was cooler than September. This is probably due to lack of acclimatization in May.

The day of the week also had an effect in the Parris Island study and might affect future allocation of medical resources. Very few heat casualties (1.1%) occurred on Sundays because physical training was limited on weekends. Slightly fewer women developed heat injuries on Mondays and there were fewer cases in men on Tuesdays, both of which may be due to consistent daily differences in their training schedules.[189]

Fitness

A sedentary lifestyle leads to both decreased muscle mass and increased nonmuscle tissue. As a result, for any given amount of exercise, muscles require more energy per gram of body mass. That is, more heat is produced per gram of body mass as overall efficiency declines and the relative metabolic heat load increases.

Physical training improves tolerance to heat not only by improving the efficiency of the cardiovascular system and reducing the amount of excess body fat, but also by reducing the threshold T_c for initiating cooling mechanisms. Whereas physical training increases heat tolerance, a sedentary lifestyle decreases it.[192] Most striking has been the demonstration of a much higher rate of heat-induced mortality in sedentary than in trained rats (Figure 5-4). The key factors of "training" include not only increased efficiency of muscle movement but also increased extracellular volume and better thermoregulatory reactions. Although there are many close similarities with heat acclimation, the maximal O_2 utilization capacity (often defined as relative fitness) of an individual is rivaled only by anthropometric characteristics (eg, body fat, the ratio of surface area to mass) as the principal correlate with reactions to heat stress.[193] The critical determinant of fitness for heat tolerance is probably not O_2 intake capacity per se, because associated physiological adaptations such as changes in organ and cellular or molecular state are very important also. Nevertheless, Gisolfi[194] has warned that because endurance athletes have a greater capacity to sustain circulatory stability at the expense of increasing T_c, they may ultimately be at greater risk of heat injury. Finally, a high level of fitness will slow the loss of heat acclimation and expedite its return if heat exposure is interrupted.[195]

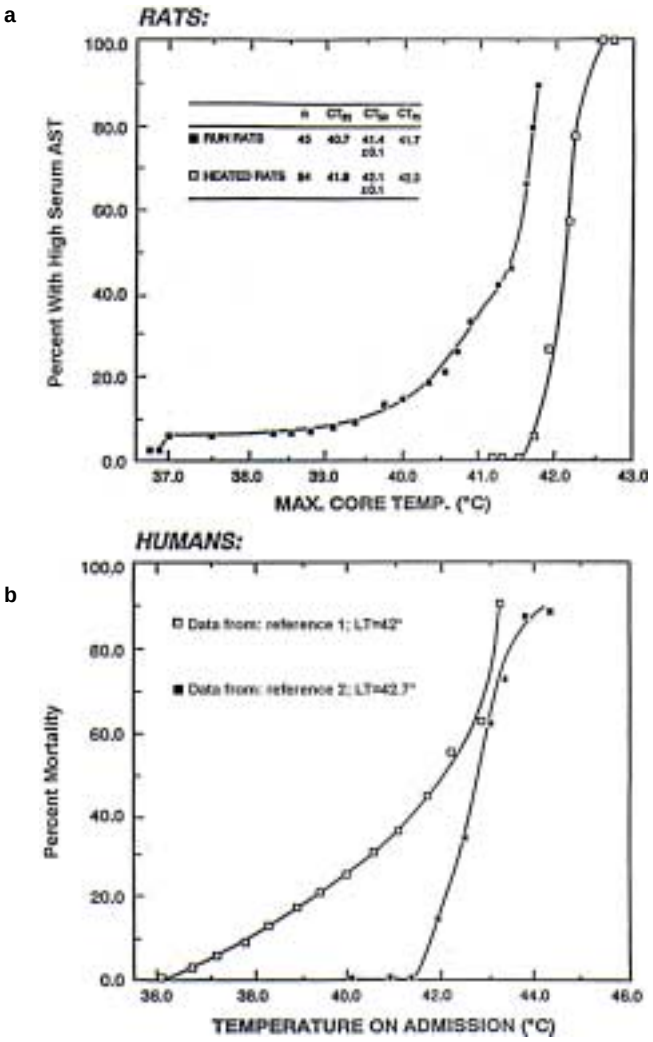

Fig. 5-4. Effect of temperatures on liver damage in rats and mortality in humans. (**a**) Dose-response curve of percentage of surviving rats with serum aspartate aminotransferase (AST, formerly called SGOT) levels in excess of 1,000 IU/L, versus maximum core temperatures of run–exhausted or restrained–heated rats. Values in the insert represent mean ± SE of core temperature at indicated percentages. CT_{50} for run rats was significantly different from CT_{50} for heated rats ($P < 0.025$). Note the increased morbidity (enzyme release) in exercising rats at equivalent core temperatures. (**b**) Dose-response curve of percentage mortality versus temperature was based on hospital records of patients with heatstroke. Note the similarity to mortality data in exertional versus classical heatstroke in humans. Data points in graph b are recalculated from original data in (1) Gauss H, Meyer KA. Heat stroke: Report of one hundred and fifty-eight cases from Cook County Hospital, Chicago. *Am J Med Sci.* 1917;154:554–564 and (2) Ferris EB, Blankenhorn MA, Robinson HW, Cullen GE. Heat stroke: Clinical and chemical observations on 44 cases. *J Clin Invest*. 1938;17:249–262.
Reprinted with permission from Hubbard RW, Matthew WT, Criss REL, et al. Role of physical effort in the etiology of rat heatstroke injury and mortality. *J Appl Physiol*. 1978;45:(graph a) 466, (graph b) 467.

PROPHYLAXIS AND THERAPY

Acclimatization to heat and to physical training is very important in resisting heat illnesses. Hypohydration leads to reduced muscle strength, reduced endurance during exercise, and elevated rates of heating.[196] In response to 1- to 2-hour periods of heat stress per day, over several days, a soldier develops elevated levels of aldosterone, and increased numbers of Na^+/K^+ ATPase plasma membrane pumps in his or her cells. One effect is that the extracellular volume is enlarged and responds to heat stress with a reduced fall in CVP.[86,197,198] Whereas complete acclimatization requires 7 to 10 days, there is significant improvement in response to heat even after 2 days. For a review of cellular adaptations to heat, see the review by Horowitz.[199] Ingestion of fluids enhances heat tolerance by minimizing the decline in central plasma volume from elevated BF_{sk} and sweat loss.[200]

Hydration and Rehydration

"Water discipline," meaning fluid deprivation, is a long-practiced attempt at reducing the body's water requirements during activities in the desert or jungle. However, it is an outdated, incorrect, and dangerous concept that implies that proof of manhood and esprit de corps can overcome the principles of physiology. That concept of voluntarily or involuntarily withholding of water from soldiers in the heat, despite severe thirst, leads to unnecessary deaths and permanent neurological damage in the survivors of heatstroke.[201]

If a soldier is *hypohydrated* when commencing strenuous physical exercise or combat in the heat, he will be at a disadvantage compared with a euhydrated soldier. He will overheat earlier, his physical and mental performance will deteriorate sooner, and he will

be more susceptible to hemorrhage or other trauma. Soldiers should be taught that the "Cost of Work in the Heat = Sweat = Water." Water can be seen as a "tactical weapon" that enhances and extends performance and resistance to heat illness.[202] Water is better carried in the body than unused in a canteen.

Clearly, a soldier should be well-hydrated, but not excessively overhydrated—which is just as debilitating—before beginning physical activity in the heat. Soldiers who are dehydrated for any reason should be prevented from exertion while heat-stressed, and they should drink 8 ounces (1 cup) of water or appropriate fluid-replacement beverage 10 to 15 minutes before starting physical activity. A soldier should be forced by command to drink 8 to 12 ounces of fluid every 20 to 30 minutes during exercise. Reliance on voluntary intake to maintain adequate fluid balance will result in dehydration at high ambient temperatures. The traditional rules of some sports (such as field hockey, soccer, and rugby) unintentionally limit opportunities for hydration by failing to provide for adequate time-outs and maintaining very brief half-times. During extremely hot–humid weather, team physicians, trainers, coaches, and officials should work together to incorporate additional breaks (quarters rather than halves) and provide unlimited access to fluids at the sidelines. For additional information see the 1990 *Report of a Workshop Committee of Military Nutrition*, by R. W. Hubbard, P. C. Szlyc, and L. E. Armstrong.[203]

Nude weights taken before and after exercise can guide the requirements for intake of additional fluids after exercise and before the next trial. For every pound of weight lost during activity, a soldier should consume a pint (2 cups) of liquid. Fluid loss should be replaced before return to activity in the heat; if a weight loss of more than 2% persists, the individual should be withheld from activity. Prehydration will forestall dehydration and enhance long-term performance. Commanders who fail to comply with these standards should be held accountable for any resulting heat injury.[1]

Because most heat illnesses in the field involve dehydration, volume expanders such as Ringer's lactate or normal saline should be administered intravenously immediately after commencement of both intubation, if necessary, and cooling. These solutions expand the intravascular volume without effects on hyponatremia.

The current (year 2000) protocol for treating suspected exertional heatstroke victims among Marine recruits at Parris Island is described in Exhibit 5-2.[204]

EXHIBIT 5-2

PARRIS ISLAND PROTOCOL FOR TREATING SUSPECTED EXERTIONAL HEATSTROKE

1. During physical training, the clinic routinely maintains a dedicated room with two bathtubs full of cold water and ice. In the field, the blouse and pants are removed from a collapsed suspected heatstroke patient; the shorts and T-shirt remain on. Rectal temperature is measured, ice is packed around the groin and axillary areas, and the patient is immediately transported to the clinic on a stretcher. On arrival, the stretcher is placed on top of the iced bathtub above the water and ice, with the carrying handles sticking out at both ends. Mental status and other vital signs are assessed, and blood is drawn for laboratory analyses.
2. One liter of normal saline is administered intravenously, *as a bolus.*
3. Sheets are dipped into the tub's icy water and are used to cover and drench the patient. Copious ice is added to the top of the sheet to cool still further, and the skin is massaged to improve skin blood flow. The sheets are frequently rewetted with the icy water. Concurrently, the head is constantly irrigated with more ice water and a fan is directed at the patient.
4. If rectal temperature is not lowered sufficiently, then the patient is immersed directly into the ice and water. With this procedure rectal temperature usually falls to 102.5°F (39.5°C) within 15 to 20 minutes, and the patient is removed from the stretcher, rinsed, placed on a gurney, and intravenous fluids and laboratory studies are reviewed.

   Supplemental potassium should not be administered until serum electrolytes have been determined. Once they are known, then the choice of additional fluids should be based on the electrolyte status and cardiac and renal functions.

Source: Gaffin SL, Gardner JW, Flinn DS. Cooling method for exertional heatstroke. *Ann Intern Med*. 2000;132:678.

(NB: These victims are young men and women who are otherwise healthy. Victims of classic heatstroke are usually elderly and with concurrent diseases, and those factors must be taken into consideration.)

Fluid Replacement Beverages

One cannot rely on the thirst reflex to prevent dehydration. Simply providing water or other fluids during training or combat or even resting (especially in sunshine) may not be sufficient. Required are (1) breaks in activity during training and (2) drinking on command every 20 to 30 minutes, depending on the degree of heat stress.

What should be the composition of the ideal beverage, given that both electrolytes and water are lost during sweating, and that mainly carbohydrates are used as a fuel source during physical activity? Water and nutrients are absorbed in the upper part of the small intestine. The rate at which fluid is emptied from the stomach or absorbed in the intestines determines the rate at which it can be of value in rehydration. The rate of gastric emptying depends on osmolality and caloric content. Which factor predominates depends on physical activity and the temperature and volume of the beverage. When normal performance is the criterion for the maintenance of plasma volume, some studies suggest that there is no need to add carbohydrates and electrolytes to water.[205] A number of commercial and experimental carbohydrate–electrolyte drinks were tested for their ability to prevent hyponatremia during exercise or to improve performance. Most of the solutions were about equally effective in maintaining water and mineral balance. Furthermore, despite early disagreements, there is little difference in emptying time between beverages that contain 2.5% and those that contain 7% carbohydrate. During exercise, both 5% glucose polymer solutions and water show similar gastric emptying rates. Probably the major benefit of commercially available sports drinks was in their enhanced palatability, which reduced hypohydration through a voluntary increase in fluid intake.[206–212]

Although it is widely believed that drinking cold water causes stomach cramps and inhibits emptying, this is not the case.[213] On the contrary, drinking cold water increases the activity of the smooth muscle in the gastric wall, thereby increasing motility and emptying the stomach more rapidly than drinking warm beverages.

Transport of sodium is the major determinant of water absorption in the proximal small bowel. The active, coupled transport of sodium and glucose creates an osmotic gradient that pulls water from the lumen into the epithelial cells. A frequent argument for including sodium in fluid replacement beverages is enhancement of intestinal absorption. Nevertheless, solutions of carbohydrates and electrolytes are not absorbed more rapidly than pure water.[210]

On the other hand, *feeding* carbohydrates during prolonged exercise *enhances* exercise performance, whether assessed by exercise time-to-exhaustion or by time to complete a predetermined exercise task.[214,215] To do so, a sports drink needs to contain 5% to 10% carbohydrate in the form of glucose or sucrose.[216] However, drinking a solution more concentrated than 10% glucose causes athletes to suffer cramps, nausea, and diarrhea. But glucose can be polymerized, so that a sport drink that is isocaloric to native glucose has only one-fifth the osmotic pressure. With this lower osmolality, one can increase the carbohydrate content without risking the gastrointestinal side effects of a high-osmolar drink. Several commercial polymer solutions are currently available. Glucose polymer solutions given before and during a soccer game resulted in sustained blood glucose and improved performance.[217] Athletes who might benefit are those involved in soccer, field hockey, rugby, and tennis, because the paucity of rest periods and substitutions makes these sports similar to sustained operations in the military. Whether the use of these beverages spares muscle glycogen is a matter of current debate.

The use of carbohydrate solutions is not completely without disadvantages, however. First, these beverages are expensive, and second, the presence of such drinks may attract bees and hornets into the vicinity of the athletes, placing them at risk for sting-induced allergic or anaphylactic reactions. Furthermore, once a carbohydrate has been placed into a canteen, then health risks from potential pathogens growing in the canteen may be more serious than the potential benefit of the carbohydrates, which could probably be better administered in the form of candy bars.

Under usual conditions, electrolyte- and carbohydrate-containing beverages offer no advantage over water in maintaining plasma volume, or electrolyte concentration, or in improving intestinal absorption. Consumption of an electrolyte-containing beverage may be indicated under conditions of caloric restriction or repeated days of sustained sweat losses. For these athletes, use of glucose polymer solutions may be considered. For the vast majority of individuals, however, the primary advantage of electrolyte- or carbohydrate-containing drinks appears to be that they enhance voluntary consumption. This factor should not be considered unimportant if regulated intake is impossible.

Adequacy of Hydration: Urine Test

Because dehydration usually precedes heatstroke, a simple test for hydration status that is easily carried out in the field would be of considerable benefit. In response to dehydration the normal kidney conserves water by excreting small volumes of osmotically concentrated urine, leading to long periods between urinations. Because the bile pigments that give urine its color become more concentrated, urine color darkens to orange or brown. If a soldier is overhydrated (a rare event in the summer) and urinates frequently, then urine color will be faintly yellow or even water-clear. A practical color chart for urine has been developed (Figure 5-5), which shows that for moderate degrees of hypohydration, the color of the urine is a more sensitive index of hypohydration than are blood measurements.[218–220] In short, soldiers should pay attention to the color of their urine. If urine is strongly yellow-colored, they are dehydrated and must drink more water. If it is clear, they are adequately hydrated.

Acclimatization

Acclimatization to heat has a profound relevance to real life problems in the armed forces.[221] Prior heat exposure enhances heat tolerance by (1) expanding circulating plasma volume, (2) increasing the maximum capacity of cutaneous vasodilatation and sweating, and (3) reducing the temperature threshold for increased BF_{sk} and sweating.[204,222] That is, acclimatization is easily shown by the presence of a lower T_c, skin temperature (T_{sk}), and heart rate in response to a standard heat stress. A further important benefit of heat acclimation is reduced sodium loss (both urinary and sweat) and, therefore, a reduced likelihood of salt depletion from high sweat losses.

Because most studies on acclimatization combined heat with exercise, it has been difficult to separate the effects of heat from those of exercise. However, it appears that heat exposure in its own right can markedly improve the tolerance of combined exercise and heat, due to increased thermoregulatory and cardiovascular capacity.[223] On a cellular basis,

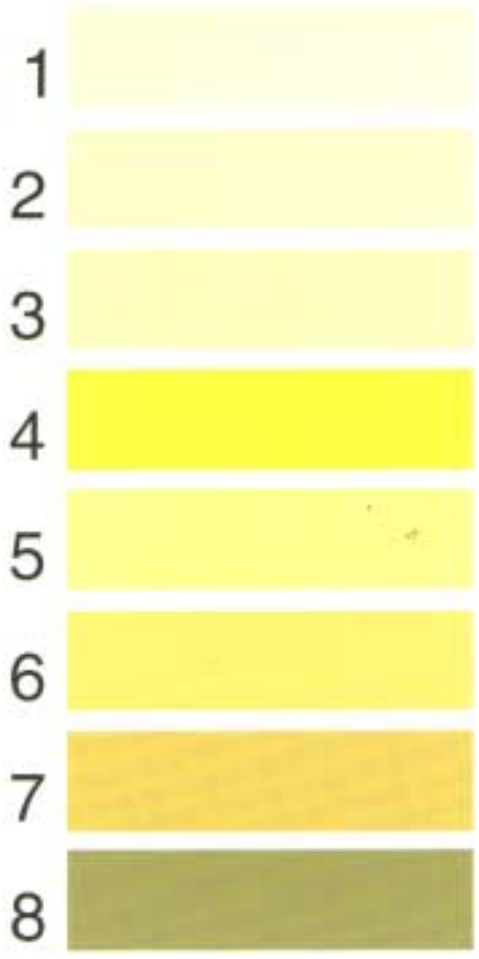

Fig. 5-5. In the presence of normal renal and hepatic function and the absence of significant hemolysis, a soldier's level of dehydration is indicated by the color of the urine. The urine color is matched closest to one in this chart. If the urine sample matches #1, #2, or #3, then the soldier is well hydrated. If the urine sample matches or is darker than #7, then the soldier is substantially dehydrated and should consume fluids.
This chart has been scientifically validated[1,2] but is not at this time officially mandated by the Department of Defense as doctrine. The chart is provided here as an aid to medical officers to consider as one of several possible indicators of hydration status of troops in training or in the field. (1) Armstrong LE, Maresh CM, Castellani JW, et al. Urinary indices of hydration status. *Int J Sport Nutr.* 1994;4:265–279. (2) Armstrong LE, Herrera Soto JA, Hacker FT Jr, Casa DJ, Kavouras SA, Maresh CM. Urinary indices during dehydration, exercise, and rehydration. *Int J Sport Nutr.* 1998;8:345–355. Copyright Lawrence E. Armstrong, 2000. Reprinted with permission from Armstrong LE. *Performing in Extreme Environments*. Champaign, Ill: Human Kinetics Publishers; 2000: 345.

heat exposure leads to a blunted rise in $[Ca^{2+}]_i$ induced by heat. Thus, maximum benefit is derived from exercise combined with heat stress, presumably because only under these conditions are there maximal drives for the required functional and anatomical changes.[194]

Heat Shock Proteins

Almost all cell types in all species can produce proteins to help repair damage caused by stressors. One such group is collectively known as heat shock proteins (HSPs) because they are induced when exposed to moderately high temperatures.[224] Once produced, these HSPs render the cell more tolerant to heat (ie, these cells can survive subsequent temperatures that were previously lethal). HSPs also prevent heat-induced cell death caused by the DNA fragmentation in apoptosis.[225] HSPs are named according to their approximate molecular weights; for example, those with molecular weights around 70 kd are in the HSP-70 family. HSPs are produced intracellularly in response to the stress over a period of minutes to hours. However, even at normal temperatures, many HSPs can be induced by other stresses such as the presence of molecules that alter or denature protein conformation, including increasing concentrations of hydrogen ions, hypoxia, urea, or nonionic detergents and calcium ions,[226] as well as toxic levels of ethanol, arsenite, and heavy metal ions.[227]

The HSPs act as "chaperones," helping translocate important proteins through cell and organelle membranes. However, their protective function in heat stress probably depends on their ability to prevent aggregation of partially denatured proteins, and their ability to help "refold" protein tertiary and quaternary structures that have been partially denatured by the stress.[228,229]

The presence of HSP-70 family members would be expected to be protective against heatstroke in mammals and appear to be involved in the process of acclimatization.[230] Experiments are underway to induce or passively administer HSPs and determine protective effects. Such studies are difficult to carry out because the HSPs are present almost entirely intracellularly, and not in the plasma.

Emergency Medical Treatment and Cooling Modalities

Heatstroke is a medical emergency, with survival depending primarily on the duration and magnitude of the elevated T_c. Therefore, the main therapeutic effort should be directed toward lowering T_c as rapidly as possible.[204,231,232] Nevertheless, it is important to follow the ABCs (airway, breathing, circulation) of stabilization while cooling efforts are initiated.

If a casualty is comatose, a cuffed endotracheal tube should be used to control the airway. Administration of supplemental O_2, if available, may help meet the increased metabolic demands and treat the hypoxia commonly associated with aspiration, pulmonary hemorrhage, pulmonary infarction, pneumonitis, and/or pulmonary edema.[100,233] If hypoxia persists despite the administration of supplemental O_2, then positive pressure ventilation should be applied.

Monitoring and recording rectal temperature on site may be important in the accurate diagnosis of heatstroke. Vital signs should be monitored, with attention to blood pressure and pulse. Although normotension should not be taken as a reassuring sign, hypotension should be recognized for the ominous sign it always is.

After removing the soldier from the heat, loosening clothing, and initiating cooling, a wide-gauge intravenous catheter should be inserted to establish access to the circulation. Intravenous fluids, saline or Ringer's lactate, should be administered as soon as possible. Recommendations regarding the rate of administration of fluids vary widely in the literature.[234] The current (year 2000) procedure at Parris Island for treating exertional heatstroke in Marine recruits is to administer an IV bolus of at least 1,000 mL.[204] The rapidly infused bolus may be more risky in elderly and infirm patients than in a youthful military victim. *Because vigorous fluid resuscitation may precipitate development of pulmonary edema, careful monitoring is indicated. Supplemental potassium should be withheld until serum electrolyte levels are known.* Future choice of fluids should reflect the individual's electrolyte status and cardiac and renal functions.[235]

Although cooling measures should be initiated immediately, cooling techniques are much less effective when the patient is actively seizing. In such cases, convulsions should be controlled by intravenous administration of 5 to 10 mg of diazepam as necessary. Following cooling, the victim should be rapidly evacuated to an emergency medical facility.

Because morbidity is directly related to the duration of the elevated T_c, the efficiency of a given method (how rapidly body temperature is lowered) is most important. In addition, there is the need for unimpeded access to the patient for continuous monitoring. Several methods for cooling heatstroke victims have been described, and there has been

controversy regarding the best approach, including administration of cold intravenous fluids, gastric lavage with cold fluids, cooling the inhaled air, and covering with cooling blankets. Although these therapies may cause lowering of T_c, they are much less effective than ice water immersion or evaporative cooling.[236–240] Ice water immersion provides a very high heat-transfer rate from the skin to the water, and the overall rate of cooling by this method is highest.[204,241] Use of cold water resulted in a rate of cooling similar to that of ice water and is less uncomfortable for the patient than immersion in iced water.[238]

In victims of extreme hyperthermia or exertional heatstroke, the ice water procedure was approximately twice as rapid in lowering T_c as the evaporative spray method (0.20°C/min vs 0.11°C/min).[242] In patients with exertional heatstroke with T_{re} as high as 43.3°C (110°F), cold water or ice water treatment in the clinic reduced T_{re} of Marine victims of heat illnesses to lower than 39°C in 10 to 40 minutes, with *no deaths or renal failure observed in more than 200 cases*.[236] On the other hand, when the evaporative air spray method was used to cool patients with classic heatstroke, 26 to 300 minutes were required to lower T_c to 38°C *and 2 of 18 patients died*.[243]

The following are the most commonly offered criticisms of cooling by ice water immersion[244]:

1. Exposure to severely cold temperatures may cause peripheral vasoconstriction with shunting of the blood away from the skin, resulting in paradoxical rise in body temperature.
2. Induction of shivering (in response to the cold) may cause additional elevation in temperature.
3. Exposure to ice water causes marked patient discomfort.
4. Working in ice water is uncomfortable for medical attendants.
5. Physical access to the patient for monitoring vital signs or administration of cardiopulmonary resuscitation is more difficult.
6. Maintaining sanitary conditions is difficult, should the patient develop vomiting or diarrhea.

While the first two criticisms may appear to be physiologically reasonable, there have been *no* reports of a rise in body temperature following ice water immersion. In fact, decreased vascular resistance has been shown to persist during ice bath cooling to normothermia.[234] Other authors who used ice water immersion for treatment of heatstroke did not find shivering problematic.[241] This is not an unexpected observation; as the hypothalamic set point for temperature regulation is not raised during heatstroke (unlike during febrile illness), the shivering response should occur only if the T_c is allowed to fall below normal. When shivering occurred, IV administration of chlorpromazine (25–50 mg) was effective.[245]

In case cardiopulmonary resuscitation becomes necessary, instead of completely immersing the victim in ice and water, it is usually preferable to place the victim on a stretcher that is placed on top of the tub, with the stretcher handles protruding at each end.[204] The patient is then conveniently drenched with sheets frequently dipped into the ice water, covering the sheets with more ice, massaging the skin, and wetting the head with ice water. The massaging may improve the flow of blood through the skin. In three series of exertional heatstroke victims in a military population (66 patients), there were no fatalities or permanent sequelae after treatment with ice water immersion.[241,246,247] In a second report of 252 cases of heatstroke in Marines, all were successfully treated with ice water immersion.[236] While other cooling methods reduce the rate of mortality, none has been as successful as ice water immersion.

Victims of heatstroke rarely require cardiopulmonary resuscitation, so this concern should not preclude use of ice baths to treat heatstroke. The documented efficacy of ice water immersion in the rapid reduction of body temperature, morbidity, and mortality overrides any consideration of transient personal discomfort for the patient or medical attendants.

An additional benefit of cooling strategies involving water immersion is the physiological shifting of fluid from the periphery to the central region, thus supporting circulation. This is particularly relevant in a tactical situation where intravenous fluids are not available, but access to a poncho-lined hole dug into desert sand and filled with 15 gallons of water is possible.[244]

The most important alternative to ice water immersion is a body cooling unit developed by Khogali and associates,[248] which maximizes evaporative cooling by suspending the patient on a net, spraying him or her from all sides with water at 15°C (59°F), and blowing warm air (45°C–48°C; 113°F–118.4°F) over the victim. Cooling rates of 0.06°C/min have been obtained. Although this method has been widely recommended as the treatment of choice, its rate of cooling is much less

EXHIBIT 5-3

FEATURES THAT HEAT ILLNESSES AND SEPSIS HAVE IN COMMON

Clinical

- Elevated core temperature[1,2–4]
- Neurological symptoms (fatigue, weakness, lethargy, confusion, delirium, stupor, coma, dizziness, paralysis, amnesia, staggering, spasm, paralysis, somnolence, lack of coordination)[1,4–8]
- Hyperventilation[6,9,10]
- Tachycardia[3,8]
- Hypotension[1,3,4]
- Renal failure[11–14]
- Organ failure[6,15]
- Shock[11,12,16,17]
- Edema[8,17,18]
- Hematocrit elevated[6,15]
- Nausea[1,3–7,19]
- Vomiting[5,8,16]
- Diarrhea[3,8,18,20]
- Headache[1,3,4,19,21]
- Myalgia[3,5,6,19,22]

Laboratory

- Leukocytosis or neutropenia[2,8]
- Hypofibrinogenemia, with fibrin degradation products elevated[19]
- Respiratory alkalosis[2,6,10]
- Metabolic acidosis[3,6]
- Decreased systemic vascular resistance[2,23]
- Increased cardiac output[2,24]
- Metabolic acidosis[2,6]
- Ischemia of the bowel, with hemorrhagic necrosis[3,8,25]
- Disseminated intravascular coagulation[17,18,26,27]
- Lactate elevated[6,28]
- Hepatic dysfunction[17,18]
- Cytokines elevated[2,5,29]
- Lipopolysaccharide elevated[29,30]

Sources: (1) Bannister RG. Anhidrosis following intravenous bacterial pyrogen. *Lancet.* 1960;2:118–122. (2) van Deventer SJH. *Endotoxins in the Pathogenesis of Gram-Negative Septicemia* [PhD dissertation]. Amsterdam, The Netherlands: University of Amsterdam; 1988. (3) Franzoni G, Leech J, Jensen G, Brotman S. Tumor necrosis factor alpha: What role in sepsis and organ failure? *J Crit Illnesses*. 1991;6:796–805. (4) Genzyme Corporation. Genzyme: Tumor necrosis factor. In: *1994 Cytokine Research Products*. Cambridge, Mass: Genzyme Corporation; 1994: 207–209. (5) Armstrong LE, Hubbard RW, Kraemer WJ, Deluca JP, Christensen EL. Signs and symptoms of heat exhaustion during strenuous exercise. *Ann Sports Med.* 1987;3:182–189. (6) Howorth PJN. The biochemistry of heat illness. *J R Army Med Corps.* 1995;141:40–41. (7) Axelrod BN, Woodard JL. Neuropsychological sequelae of heatstroke. *Int J Neurosci.* 1993;70:223–232. (8) Johannsen U. Experimental studies on the pathogenesis of Coli-enterotoxemia in swine, IV: Effect of lipopolysaccharide endotoxin on weaned piglets following parenteral administration [in German]. *Arch Exp Veterinärmed*. 1977;31:191–202. (9) Boyd AE, Beller GA. Acid–base changes in heat exhaustion during basic training. *Proc Army Science Conf*. 1972;1:114–125. (10) Boyd AE, Beller GA. Heat exhaustion and respiratory alkalosis. *Ann Intern Med.* 1975;83:835. (11) Shibolet S, Lancaster MC, Danon Y. Heatstroke: A review. *Aviat Space Environ Med.* 1976;47:280–301. (12) Shibolet S, Coll R, Gilat T, Sohar E. Heatstroke: Its clinical picture and mechanism in 36 cases. *Q J Med.* 1967;(New Series)36:525–548. (13) Gitin EL, Demos MA. Acute exertional rhabdomyolysis: A syndrome of increasing importance to the military physician. *Mil Med.* 1974;139:33–36. (14) Harman E, Frykman P, Palmer C, Lammi E, Reynolds K, Backus V. *Effects of a Specifically Designed Physical Conditioning Program on the Load Carriage and Lifting Performance of Female Soldiers.* Natick, Mass: US Army Research Institute of Environmental Medicine; November 1997. USARIEM Technical Report T98-1. (15) Hansbrough J, Moore E, Eiseman B. Shock and multiple organ failure. In: Hardaway RM III, ed. *Shock: The Reversible Step Toward Death.* Littleton, Mass: PSG Publishing Co; 1988: 435–441. (16) Haseeb MA, Amin F. Fatal effects of heat on man. *J Trop Med Hyg*. 1958;61:280–281. (17) Zhi-cheng M, Yi-tang W. Analysis of 411 cases of severe heat stroke in Nanjing. *Chin Med J (Engl).* 1991;104:256–258. (18) Southwick FS, Dalglish PH Jr. Recovery after prolonged asystolic cardiac arrest in profound hypothermia. *JAMA.* 1980;243:1250–1253. (19) Suffredini AF, Harpel PC, Parrillo JE. Promotion and subsequent inhibition of plasminogen activation after administration of intravenous endotoxin to normal subjects. *N Engl J Med.* 1989;320:1165–1172. (20) Fogoros RN. Runner's trots. *JAMA.* 1980;243:1743–1744. (21) Skidmore R, Gutierrez JA, Guerriero V, Kregel KC. Hsp70 induction during exercise and heat stress in rats: Role of internal temperature. *Am J Physiol*. 1995;268:R92–R97. (22) Knochel JP. Management of heat conditions. *Athletic Ther Today.* 1996;2:30–34. (23) Gisolfi CV, Matthes RD, Kregel KC, Oppliger R. Splanchnic sympathetic nerve activity and circulating catecholamines in the hyperthermic rat. *J Appl Physiol*. 1991;70:1821–1826. (24) Koroxenidis GT, Shepherd JT, Marshall RJ. Cardiovascular response to acute heat stress. *J Appl Physiol*. 1961;16:869–872. (25) Baska RS, Moses FM, Graeber G, Kearney G. Gastrointestinal bleeding during an ultramarathon. *Dig Dis Sci*. 1990;35:276–279. (26) Caridis DT, Reinhold RB, Woodruff WH, Fine J. Endotoxaemia in man. *Lancet*. 1972;1:1381–1386. (27) Rosenthal T, Shapiro Y, Seligsohn U, Ramot B. Disseminated intravascular coagulation in experimental heatstroke. *Thromb Diath Haemorrh.* 1971;26:417–425. (28) Boyd AE, Beller GA. Acid base changes in heat exhaustion during basic training. *Proc Army Science Conf.* 1972;1:114–125. (29) Bouchama A, Parhar RS, Er-Yazigi A, Sheth K, Al-Sedairy S. Endotoxemia and release of tumor necrosis factor and interleukin-1-alpha in acute heatstroke. *J Appl Physiol.* 1991;70:2640–2644. (30) Gathiram P, Wells MT, Brock-Utne JG, Gaffin SL. Portal and systemic arterial plasma lipopolysaccharide concentrations in heat stressed primates. *Circ Shock.* 1988;25:223–230.

than that seen with ice water immersion and should be employed only as an alternative when a bath of cold or ice water is unavailable. For further discussion see B. Yarbrough and R. W. Hubbard's chapter in *Management of Wilderness and Environmental Emergencies*.[249]

If other cooling methods are used initially, any patient whose T_c does not fall to 38.9°C within 30 minutes should be placed in an ice water bath. A rapidly falling T_c may not be accurately reflected by measured T_{re}. Therefore, no matter what technique is used, active cooling should be discontinued when T_{re} falls below 39°C (102.2°F) to prevent inducing hypothermia.

Use of antipyretics is generally inappropriate and potentially harmful in heatstroke victims unless T_c is very high. Aspirin and acetaminophen lower temperature by normalizing the elevated hypothalamic set point caused by pyrogens. Furthermore, acetaminophen could cause hepatic damage, and aspirin could aggravate bleeding tendencies. Alcohol sponge baths are inappropriate under any circumstances because absorption of alcohol may lead to poisoning and coma, particularly in a patient with heatstroke who has residual liver injury from the heat and reduced splanchnic blood flow.

In heatstroke, the set point is usually normal, with the temperature elevation reflecting failure of the normal cooling mechanisms. This failure is indicated by successes of external therapeutic cooling procedures. However, elevated levels of LPS and cytokines such as IL-1 have been reported in the plasma of heatstroke victims and in experimental heatstroke models.[156,250] In those particular cases, the thermoregulatory set point may have been altered, and they may benefit from antipyretics. But this has not been proved. To facilitate future research on the underlying mechanisms of heatstroke and sepsis, and to further elucidate many overlapping areas that share common mechanisms and pathways, we offer the comparative summary in Exhibit 5-3.

SUMMARY

Heat illnesses may vary in intensity from a sense of fatigue to severe symptoms, shock, and death. These disorders are always medical emergencies requiring immediate recognition and rapid initiation of therapy. Heatstroke typically appears in two completely different types of patients:

1. Classic heatstroke: the victim is older; debilitated, often with liver failure; does not exercise; and develops the disease over a few days during a heat wave.
2. Exertional heatstroke: the victim is usually in his teens to thirties, in otherwise good health, and collapsed during a few minutes to a few hours of exercise in a cool-to-warm environmental temperature.

Other heat illnesses, such as heat cramps and heat exhaustion, depend on alterations in circulating volume and electrolytes due to relatively unequal sweat losses or inappropriate ingestion of water and salts.

In most medical schools, heatstroke and other heat illnesses have usually been taught as a straightforward series of well-described pathophysiological conditions characterized by various levels of core temperature, extent of dehydration in various body compartments, reduced CVP, depression of sweat gland function, and alterations of intracellular and plasma ions and pH. These physiological mechanisms, however, do not explain why classic heatstroke victims are different from exertional heatstroke victims, nor do they explain why a number of heatstroke symptoms are similar to those of septic shock, such as DIC, nausea, vomiting, diarrhea, intestinal bleeding, and so forth. Furthermore, classic physiological theory does not explain why concurrent physical activity lowers the threshold core temperature.

Recent advances in basic science provide new information that can help explain the underlying cellular, subcellular, and chemical pathophysiology of heat illnesses, and can lead to new therapy and prophylaxes. Specifically, heatstroke patients usually have altered immune systems with elevated and probably inappropriate levels of circulating inflammatory cytokines, Gram-negative bacterial lipopolysaccharides, and corticotropin-releasing hormone; show a programmed generalized response to stressors; and have depressed stores of intracellular ATP.

In addition to providing support for the classic mechanisms for the pathophysiology and diagnoses of heatstroke and other heat illnesses, and their limitations, we presented new information about these other factors: how they may participate in the pathophysiology of heat illness, with a view to using these newer pathways in designing future therapy and prophylaxis. Also included are current (year 2000) protocols for cooling and rehydrating patients with heatstroke.

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Chapter 6

PREVENTION OF HEAT ILLNESS

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INTRODUCTION

Heat stress is a significant factor in many military activities, and heat casualties have posed a problem throughout the history of warfare. Egyptian failure to implement adequate precautions against heat stress contributed to the quick Israeli victory in the 1967 conflict in the Sinai desert.[1] Heat casualties among US military trainees were a major concern during World War II[2] and continue as a problem to this day.[3,4] Both desert (hot, dry) and tropical (warm, wet) climates pose hazards; the current requirement that US forces stand ready to deploy on short notice anywhere in the world means that all four US military services must be prepared to operate under hot conditions to which personnel are unaccustomed. Relevant research programs are conducted at a number of military laboratories, including the US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts, and the Naval Health Research Center, San Diego, California.

Heat has a broad spectrum of effects. Heatstroke is equivalent to a major wound: each case is a potential fatality, and survivors are lost to service for prolonged periods. In contrast, heat exhaustion and related syndromes are only temporarily incapacitating, are generally treated in the field, and often go unreported; they can nevertheless significantly affect mission accomplishment.[5] There is also a growing body of evidence that heat stress that is physiologically tolerable can impair human ability to accomplish complex tasks of military importance.

The implementation of relatively simple preventive measures can have a dramatic effect on heat illness rates in military settings,[5,6] yet the necessary preparations are readily overlooked during contingency planning. Medical personnel must be prepared to advise commanders on the potential adverse effects of heat and to propose practical options for control of heat stress under difficult circumstances. The development of credible medical guidance requires a thorough understanding of the material in the Hot Environments section of this textbook, including knowledge of normal human responses to heat (Chapter 2, Human Adaptation to Hot Environments, and Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues), its psychological effects (Chapter 4, Psychological Aspects of Military Performance in Hot Environments), the nature of heat illness (Chapter 5, Pathophysiology of Heatstroke), and its treatment (Chapter 7, Clinical Diagnosis, Management, and Surveillance of Exertional Heat Illness). This chapter summarizes this material as it relates to the prevention of heat-induced errors, performance decrements, and casualties.

Complexity of the Threat

Heat casualties occur when the stresses imposed by some combination of environment, work, and clothing combine to exceed individual tolerance limits (Figure 6-1). Personnel may encounter high environmental heat loads while working outdoors on paved surfaces, muddy trails, or desert sand; during road marches; and while carrying litters or servicing aircraft. Enclosed spaces can be extremely hot owing to the heat generated by internal sources as well as the environmental heat load; examples include poorly ventilated vehicles, maintenance facilities, and ship compartments. Hot working conditions are often made more dangerous by military

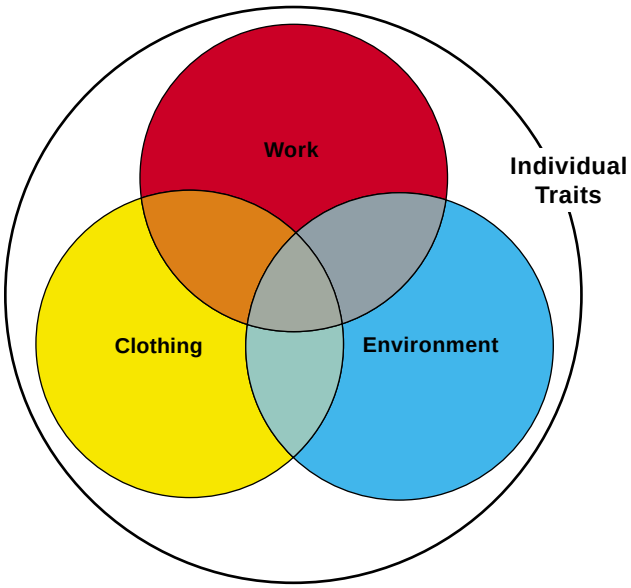

Fig. 6-1. Venn diagram of the three factors that determine heat stress: work, the environment, and clothing. Single factors represent a lesser risk of heat stress, two overlapping factors represent intermediate risks, and the area where all three factors overlap represents a heightened risk of heat stress. The response to a given level of heat stress also depends on a variety of individual traits, which interact with the three factors and may vary from one day to the next. Adapted with permission from Nunneley SA. Design and evaluation of clothing from protection from heat stress. In: Mekjavic I, Banister E, Morrison J, eds. *Environmental Ergonomics.* London, England: Taylor & Francis; 1987: 88.

demands for prolonged exertion. In addition, heavy or impermeable protective clothing may cause heat illness in otherwise temperate conditions. The picture is further complicated by the multiplicity of interacting variables that determine the normal range of individual heat tolerance.[7]

The level of heat stress and consequent risk that are acceptable can vary widely depending on the military situation. The goal for troops on a road march may be simply to go the distance without incurring frank casualties. On the other hand, a fighter pilot requires substantial protection from heat and dehydration to support the stress of aerial combat. Low-level missions in all types of aircraft expose crews to hot ambient conditions that are linked to performance decrements.[8,9] Helicopter crews face special problems in performing highly skilled flying tasks alternating with physical work to prepare the aircraft for the next flight; in addition, they often sleep in tents and thus fail to obtain the level of comfort and recovery afforded to aircrews in fixed-base and carrier operations.

Although most knowledge of heat stress is based on studies of healthy young men, military deployment now involves men and women, regular troops, reservists, and contractors, over a wide range of ages. Some of these personnel may have preexisting medical conditions and take prescription medications. Such a mixed population will produce a broader range of responses to heat stress and may develop medical problems not seen in younger populations.

Heat Stress in Military Settings

Preventive strategies cannot be expected to eliminate heat stress but should minimize its impact on the mission while preserving the health of personnel to the fullest extent possible. Techniques for primary prevention in both civilian and military settings include administrative plans and procedures, engineering control of the environment, appropriate use of equipment, and continuous medical surveillance to screen out vulnerable individuals. Supply officers preparing for deployment should be made aware of the need to stock hats, sunglasses, sunscreen, lip balm, and skin-care items, while individuals should ensure that their own deployment kits contain an initial supply of these items.

Flexibility and practicality are critical in providing medical support to commanders who face difficult decisions. Military working conditions often limit the effectiveness of natural thermoregulatory mechanisms and constrain normal behavioral defenses. Common civilian strategies to ameliorate environmental conditions, reduce workload, or lighten clothing may be unacceptable because they interfere with military objectives. Furthermore, exposure to heat in military settings is not limited to a conventional work shift but may continue without relief for prolonged periods, especially for troops living in tents or structures that are subject to solar load and lack air conditioning. Therefore, programs to control heat stress among deployed troops must often be extended beyond the working environment to cover conditions for feeding, rest, and recreation.

Military medical personnel who must work in hot conditions should recognize that they are themselves subject to deterioration in performance and possible heat illness. Health maintenance is particularly important in this group because healthcare involves a combination of physical effort, skill, and judgment that affects the welfare of their patients. Medical facilities in buildings and tents should be actively cooled in hot climates because high indoor temperatures cause difficulties with equipment and deterioration of supplies and are detrimental to the welfare of patients and staff. In the presence of heat stress, performance of critical tasks should routinely be double-checked for errors of omission and commission.

IMPROVING HEAT TOLERANCE

Human capacity to work in hot conditions can be maximized through preparatory physical conditioning and attention to details on deployment. Careful attention must be paid to physical fitness and heat acclimatization, living conditions, personal hygiene, and replacement of fluid and electrolytes.

Physical Fitness and Acclimatization

Human tolerance for work in heat is substantially affected by an individual's recent history of exposure to such stress. Acclimatization (ie, physiological adaptation to repeated stress, in nature) to heat produces complex physiological changes that improve heat transport from the body's core to its surface, and then dissipation of the heat to the environment.[10] Any exercise program that builds and maintains a high level of aerobic fitness will also confer at least partial adaptation to heat stress.[11,12] In addition, highly fit individuals will achieve complete acclimatization more quickly and with less discomfort than is the case for sedentary persons.[13,14]

An exception to this linkage between aerobic capacity and heat tolerance is fitness that is developed primarily through swimming in water cool enough to limit the usual exercise-induced rise in core temperature.[15] It should also be noted that high levels of fitness and acclimatization offer little improvement to work in heat with protective clothing.[12]

Military units preparing for deployment to a hot climate should intensify their physical training and use it to gradually increase their state of heat acclimatization. Personnel who cannot prepare in advance must be allowed a period for acclimatization on arrival at the deployment site. In either case, acclimatization is most quickly accomplished through daily exercise bouts that last a minimum of 1 to 2 hours per day and are sufficiently strenuous to produce a rise in core temperature and profuse sweating. Although complete acclimatization requires about 10 days, substantial changes take place in the first 2 to 3 days. Exposure to heat stress every second or third day will also produce acclimatization, although such intermittent exposure greatly prolongs the total period required to achieve complete adaptation.[16] If the unit is located in a cool climate, acclimatization can be improved by wearing heavy clothing during exercise, cautious use of a vapor-barrier layer in the clothing, or indoor exercise. Passive exposure to heat (as in a sauna) does little to improve capacity to work in heat.

Both physical fitness and heat acclimatization are subject to decay if conditioning is discontinued for long. Therefore, unless military operations themselves provide a high level of activity, appropriate physical training should be resumed soon after arrival at the deployment site. However, maintaining fitness in the field requires careful planning.[17] Outdoor training may have to be scheduled at unusual hours; local climate and weather conditions should be analyzed to select the best possible times to allow adequate exertional stress without undue risk of heat casualties. Vigorous exercise such as running can produce heat illness at any time of day if the combination of heat and humidity produces a wet bulb globe temperature (WBGT) of 18°C (65°F) or higher,[4] or at lower WBGTs if the participants are loaded with protective clothing and equipment. Furthermore, exercise in hot weather requires that personnel have suitable lightweight clothing, sufficient water and soap to wash themselves and their garments, and space to dry clothing and towels. Laundry requirements can be reduced if exercise equipment is set up in an air-conditioned building or tent, but this limits the stimulus to maintain heat acclimatization, one of the purposes of the exercise.

While acclimatization is critical to maximizing heat tolerance, it does not confer immunity, and fully adapted personnel can still be overwhelmed by a stressful combination of work, environmental heat load, and protective clothing. In addition, the benefits of acclimatization can be nullified by other stresses associated with deployment, including sleep deprivation, illness, dehydration, missed meals, or use of drugs and alcohol. Heat strain also reduces the physiological resources available for defense against other environmental stressors and makes it difficult for individuals to assess their own reserves. Examples include the occurrence of parade-ground syncope and unexpected acceleration-induced loss of consciousness in flight.

Although early studies of women seemed to indicate that they had a relatively low tolerance for work in heat, later experiments showed that finding to be an artifact related to fitness and other factors that differed systematically between the study groups of men and women.[18] Later experimental protocols that used matched subjects or otherwise made allowance for differences in physical characteristics found that men and women responded similarly to heat stress.[19–22] Closely controlled laboratory studies show small, consistent changes in thermoregulation over the menstrual cycle, but this has no practical effect on women's heat tolerance.[21,23]

Living Conditions and Personal Hygiene

Heat stress and sleep loss tend to form a positive feedback loop. It is therefore important to develop and enforce adequate work/rest schedules and sleep discipline among troops, not forgetting those in leadership positions. Special provision must be made for individuals who work at night because they often have difficulty getting adequate sleep during the day, particularly on deployment to hot climates. Every effort must be made to provide such personnel with cool, dark, quiet sleeping accommodations that are located as far as possible from noise sources such as roads, aircraft landing zones, maintenance shops, and recreational areas. Because windows must be covered or tent sides rolled down to shut out daylight, active cooling must be provided in the form of evaporative cooling or refrigerated air conditioning, supplemented by appropriate use of fans. Cooling systems also provide low-level background sound, which helps to mask outside noise. Ear plugs and sleep masks should be available for those who find them helpful. Short-acting hypnotics may also be considered under special circumstances.

Maintaining safe supplies of food and water in

hot weather requires especially strict enforcement of sanitation and hygiene. Use of local supplies and handling of food and water by indigenous personnel introduce potential sources of infection. Seemingly minor lapses in cleanliness and refrigeration can produce immediate, disastrous consequences. Precautions must also be taken to prevent the proliferation of pests and the transmission of endemic diseases caused by bacterial, viral, and parasitic agents. Gastroenteritis constitutes a serious threat to people who work in hot conditions because emesis and diarrhea lead to dehydration and electrolyte disturbances, which can then impair heat tolerance.

Care of the skin and eyes is especially important in hot climates. Whenever possible, lightweight garments should be used to prevent sunburn while allowing free movement of air over the body. In addition, sunblock should be used generously on exposed skin to prevent acute sunburn and decrease the lifetime risk of skin cancer (a serious problem for military members, who may accumulate many years of exposure to solar ultraviolet radiation). Personnel should be encouraged to wear gloves and appropriate hats; hats with brims all around are preferable to baseball-style caps, which fail to protect the neck and ears.[24] Routine use of sunglasses or goggles is advisable, especially in windy conditions and around aircraft. Protective eyewear keeps sand and other foreign material out of the eyes, and prevents acute solar keratoconjunctivitis as well as lowers the incidence of pterygium. Eye protection also reduces the long-term effects of ultraviolet exposure with associated cataract formation.

Fungal and bacterial skin infections are a serious problem in hot weather; in desert environments the threat arises primarily from grit and irritation in crevices and areas of friction, while in humid conditions the problems are related to continuous exposure to sweat and moist clothing. It is therefore important to provide adequate facilities for bathing and laundry and to encourage their regular use by all personnel. Undergarments, socks, and shoe liners should be changed frequently, washed with disinfectant detergents or additives, and kept as dry as possible between uses.

Metal and other materials that have been sitting in the sun can be hot enough to blister skin on contact.[25] Personnel should be forewarned and wear gloves if they must handle materials under such conditions. Because swelling of the hands is a common problem during the first several days of heat exposure, personnel should be advised to remove rings in advance and string them onto their dog-tag chains. Body-piercing ornaments present special hygienic difficulties in hot climates and, like rings, present hazards of physical trauma and burns under wartime conditions.

Fluid and Electrolyte Balance

Unimpaired mental performance and physical work in the heat can be sustained only with adequate intake of water, electrolytes, and energy substrates; of those three, it is water that must be replaced on an hourly basis. Even mild dehydration leads to early fatigue and may also be associated with increased incidence of nonthermal illness and injury.[26] Dehydration is associated with a progressive rise in core temperature, reduced plasma volume, and tachycardia,[27] leading to clinical effects ranging from syncope to heatstroke.[28]

Deployment produces a variety of associated stresses such as frantic schedules, cumulative fatigue, infectious illness, and anxiety, which often cause people to ignore thirst or forgo meals. Individual trouble signs include weight loss and dark (concentrated) urine. In addition, troops in the field (especially women) may deliberately limit drinking to delay the need to urinate; this self-imposed dehydration is dangerous and must be counteracted through education and by providing the best possible latrines, which are both reasonably convenient and sufficiently private. Supervisors may need to institute a system of urine-color checks as a simple means to monitor hydration.

Desert air may be so dry that sweat evaporates instantly; under such conditions, personnel who are sweating profusely may develop severe dehydration without becoming aware of it.[29] During hard work in either hot–dry or warm–humid conditions, sweat output can exceed the rate at which water can be consumed, emptied from the stomach, and absorbed from the gut, a maximum of about 1.5 L/h in men.[30] When water intake and absorption fail to keep pace with loss, progressive dehydration is the inevitable result. The options for avoiding trouble are either

- to work until some physiological limit is reached and then take the substantial time required for complete recovery, or
- to take frequent rest breaks that lower time-averaged metabolic heat production and permit catch-up rehydration.

Attempts to drink more water than the gut can absorb will lead to abdominal distress and possibly to vomiting.[31,32]

Water Intake

Early investigators found that troops in the desert lost 1% to 3% of their body weight before voluntarily beginning to drink and then drank less water than they were losing through sweat; the resulting water deficit was not reversed until the evening meal.[29] Adolph and associates named this condition "voluntary dehydration"[29]; its magnitude depends on a variety of factors, including overall stress level and psychosocial factors as well as physiological control mechanisms.[33] Much of the current knowledge in this area derives from sports medicine. Distance runners on average consume water at the rate of only about 500 mL/h during a race and frequently lose 5% to 6% of their body weight before reaching the finish line.[34] Such dehydration alters the distribution of water through the various body compartments, contributes to cardiovascular drift, and can lead to premature exhaustion.[27,35]

Statements of requirements for water and electrolytes may be found in the chapter by Montain, Hydration, in Volume 2 of *Medical Aspects of Harsh Environments*, and elsewhere.[36–38] Contrary to popular belief, acclimatization does not decrease the need for water but rather *increases* it through earlier onset and higher rates of sweating.[39] Requirements for drinking water range from about 2 L/d for a sedentary person in a temperate climate to 15 L/d or more for someone performing hard work under the hottest desert conditions (Figure 6-2).[29] The same database was also used to estimate the water needs of sailors aboard lifeboats in hot climates.[29]

For troops on deployment, rehydration after a workday (or night) is an important aspect of preparation for the next work bout. Complete rehydration is best accomplished at meals, which should be accompanied by generous supplies of cool water and flavored drinks served in large cups. Personnel should take a generous drink before going to sleep and again on arising because this routine allows the kidneys to produce a normal state of fluid and electrolyte balance before the next exposure to stress. The possible benefits of prehydration or deliberate water loading just before the onset of heat stress are a matter of debate.[35]

Voluntary dehydration can be reduced through training and active encouragement of drinking in amounts equaling at least 80% of water lost as sweat.[40] Frequent intake of moderate volumes is generally better than large drinks at longer intervals.[31,32] Leadership must implement a system for delivering water on a prescribed schedule to both fixed work sites and troops on the move, and should implement a simple system for monitoring individual consumption. For example, soldiers on a road march may be instructed to carry two canteens and to consume their contents during a 1-hour interval, at the end of which there is a rest break and every canteen is refilled under supervision from unit supplies. At air bases and other fixed locations, water must be readily available at each work site, in vehicles, and in sleeping quarters. Drinking cups or free-flowing containers should be provided to facilitate rapid consumption of large volumes. In this spirit, normal US Air Force rules forbidding loose objects on the flight line were waived during the Persian Gulf War to allow aircraft mechanics to keep water bottles in their work areas.[41]

For purposes of rehydration, plain or pleasantly flavored water is preferable to beverages that are carbonated, heavily sugared, or contain caffeine (which is a diuretic).[42] Any drink that decreases thirst may inhibit intake before full rehydration is accomplished. So-called sports drinks and other flavored beverages may enhance consumption by some individuals but should be used with caution (discussed later and in Chapter 5, Pathophysiology of Heatstroke), and plain water must always be available to be drunk alone or mixed with other beverages. Commercial flavorings neutralize chlorine or other water purification agents and should therefore be added just before use, while drinks con-

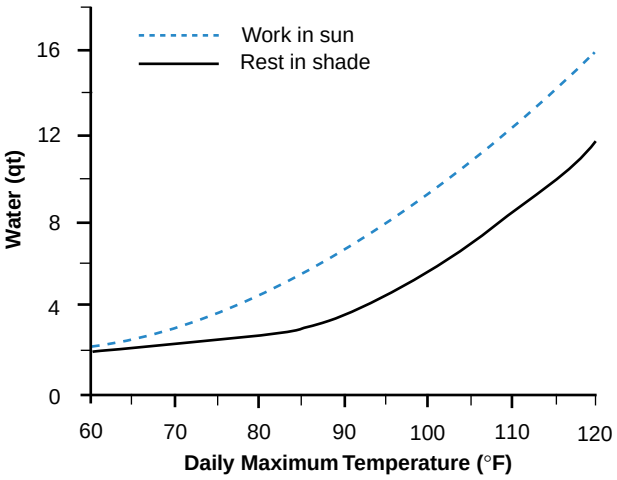

Fig. 6-2. Daily water intake requirements for men in the desert. Adapted with permission from Adolph EF, Associates. *Physiology of Man in the Desert*. New York, NY: Haffner Publishing (1969 facsimile of the 1947 edition); 1947: 121.

taining nutrients must be handled in the same manner as foodstuffs.

Drinking will be maximized if fluids are cool but not ice cold.[42–44] A simple cooling technique in the field is to store water in semiporous containers, where external evaporation lowers the temperature of the contents. This works best in arid environments, where low humidity assures efficient evaporation. Cooling by refrigeration or ice requires access to sophisticated machinery and power supplies. In addition, ice is a common medium for the spread of gastroenteritis, a lesson that has been relearned in every major deployment, including the Persian Gulf War. Ice may be made from an unsafe water supply or be contaminated in handling, and it cannot be disinfected. If there is any doubt of its purity, the ice should be used only for external cooling of drink containers without allowing either the ice or its meltwater to mix with drinks. In addition, nonpotable water and ice can be used to wet skin and clothing in the hope that such external cooling might lower the sweat rate and thus reduce dehydration.

Electrolyte Requirements and Nutrition

Secreted sweat is a markedly hypotonic solution containing sodium chloride, a small amount of potassium, and traces of other minerals and organic compounds.[45] Acclimatization to heat enhances reabsorption of electrolytes from the sweat ducts and thus improves electrolyte conservation; however, total salt loss may increase due to high sweat volume. Under normal conditions, the diet contains sufficient sodium and electrolytes to replace daily losses in the sweat of acclimatized persons.[37] However, it is possible for sodium depletion to develop in persons who are sweating profusely and either not fully acclimatized or not consuming normal amounts of dietary sodium. Adequate sodium is required to support the acclimatization process[46]; unfortunately, both loss of salt in sweat and failure to replace it through eating are likely to be worst early in deployment.

However, overzealous water intake over prolonged periods can increase total body water and produce dilutional hyponatremia (water intoxication).[47] Although the kidneys ordinarily can excrete a free water load, their capacity to do so may be reduced by various physiological and pathophysiological influences, including intense exercise. If the surplus water is not excreted, the fluid volume of the extracellular space increases, thus producing dilutional hyponatremia. Hyponatremia can develop fairly rapidly, and may be accompanied by cerebral edema, a potentially life-threatening complication.

Depressed appetite and gradual weight loss are common occurrences among troops deployed to hot climates.[48] Personnel should be actively encouraged to eat all scheduled rations to replenish calories and nutrients. Personnel must be taught neither to skip meals nor to replace them with candy bars, snack foods, or sugary drinks, items that may be convenient and pleasurable but lack important nutritional components. Military units should make every effort to provide at least one cooked, communal meal per day as the most effective means of encouraging adequate nutrition, complete rehydration, and reduction in stress through the opportunity to interact with others. Those responsible for planning meals should monitor dining areas to see which foods go uneaten and use that information to improve consumption.

Electrolyte and carbohydrate supplementation in drinks is not ordinarily necessary but may become an issue when logistical problems or military contingencies impede delivery of meals, and when illness, anxiety, and heat stress itself interfere with individual ability to eat. If troops are missing meals or are subjected to prolonged hard work in heat, small quantities of table salt may be added to the drinking water to improve intake and retention.[49] Carbohydrates (usually glucose) may also be added to provide energy and reduce fatigue but cannot be expected to alter thermoregulatory capacity.[50] (USARIEM recommends the following as a "homemade" rehydration drink: 6 g salt and 40 g sugar per quart of potable water.[51])

A carbohydrate concentration of 4% to 8% in drinks is recommended for endurance athletes.[40] The lower end of that range might be appropriate for military personnel, for whom heat stress is likely to originate more from environmental sources and less from extreme exertion. Concentrations greater than 8% improve carbohydrate delivery but slow gastric emptying, and therefore impair water replacement.[52] Commercial sports drinks are potentially useful for electrolyte supplementation, although brands that are hypertonic owing to their very high sugar content should be mixed with 1 to 2 times their volume of water to prevent nausea and vomiting. Salt tablets are not necessary and their use should be actively discouraged because they are readily abused. Although it is difficult to drink too much water, excess salt intake is a real hazard, leading to increased water requirements, greater urinary output, nausea, and increased susceptibility to heat illness.

MEDICAL SCREENING FOR RISK

Certain physical characteristics and a number of medical conditions are associated with increased risk of heat illness (Exhibit 6-1). The factors fall generally into two categories, transient and chronic, and include situational, personal, and medical conditions. Medical officers and other military personnel should keep in mind that casualties may be unable to thermoregulate when rashes, sunburn, or occlusive dressings cover large areas of their skin.

Transient Conditions

Recent arrivals at the deployment site are especially susceptible to heat injury and illness. Contributing factors include travel fatigue, jet lag, nutritional deficit, and sleep loss. Other problems may include recent immunizations or exposure to viral illnesses in transit. New personnel coming from cooler regions are unlikely to be fully acclimatized to work in heat and will have to learn local routines for self-care and prevention of heat illness. There is also evidence that heat stress on one day may increase vulnerability to heat illness on the next (see Figure 7-4 in Chapter 7, Clinical Diagnosis, Management, and Surveillance of Exertional Heat Illness).[4]

Heat tolerance is reduced by many common illnesses, including colds and other conditions that cause fever, vomiting, diarrhea, or failure to eat and drink normally. Extensive sunburn, miliaria rubra (prickly heat), and other rashes can seriously impair thermoregulatory capacity by altering cutaneous perfusion and inhibiting secretion of sweat over the injured area. All personnel should understand that those recovering from illness or skin problems require protection from heat stress for several days following apparent recovery to ensure full return of thermoregulatory capacity.

EXHIBIT 6-1

FACTORS THAT REDUCE TOLERANCE FOR WORK IN HEAT

Transient Conditions

- Situational
  - Travel fatigue, jet lag, or both
  - Sleep deprivation
  - Failure to eat or drink
  - Alcohol
  - Lack of acclimatization
- Medical
  - Recent immunization
  - Febrile illness
  - Gastroenteritis (emesis, diarrhea)
  - Skin conditions
  - Self-medication

Chronic or Permanent Conditions

- Personal
  - Small size
  - Low aerobic capacity
  - Age > 40 years
  - Overweight or obesity
- Medical
  - History of heat illness
  - Cardiovascular disease
  - Metabolic abnormalities
  - Prescription medications
  - Pregnancy

Chronic Conditions

Small size (ie, low muscle mass) and lack of physical fitness are risk factors in both men and women who must perform physical work at a fixed pace because the set work load uses a relatively high percentage of their strength and aerobic capacity. Aging is another risk factor because thermoregulatory competence tends to diminish with age,[53] although this trend is attenuated in persons who maintain a high level of fitness and avoid gaining weight.[53,54]

Overweight does not directly interfere with heat-dissipation mechanisms (vasodilation and sweating) but is usually associated with low aerobic capacity and lack of acclimatization, while the excess weight also increases the physical cost of any task involving locomotion. The combination of relatively low fitness and high body mass index (weight ÷ height^2, often used as an index of obesity) in military trainees significantly increases the risk of heat illness.[55]

Persons who have suffered previous heatstroke are at increased risk of recurrence,[56] although the mechanism is a matter of debate.[57,58] In addition, a small percentage of apparently normal individuals prove unable to adapt to heat. Such persons generally abhor

heat stress and have learned to avoid it in their daily lives, a behavioral defense that deployment disrupts. For this reason, medical screening for hot work should include specific questions regarding past experience with heat stress. Disqualification should be considered for anyone with a history of two incidents of heat intolerance or a single occurrence of unexplained heat illness with persistent sequelae or difficulty in readapting to heat. A few individuals may require referral for a heat tolerance test.[57]

Deployment now includes significant numbers of women and therefore requires consideration of gynecological conditions and pregnancy in relation to environmental stress. Pregnancy involves altered hormone levels, changes in fluid balance, and increased circulatory demands. In addition, morning sickness can cause problems with nutrition, electrolyte balance, and hydration. The result may be increased susceptibility to syncope and diminished tolerance for dehydration. Although severe maternal hyperthermia due to febrile illness increases the incidence of fetal malformation, heat stress in the physiological range appears to pose little risk.

Chronic medical conditions that cause difficulty in hot climates include diabetes mellitus, thyroid disorders, renal disease, and cardiovascular disease. Any process that limits cardiac pumping or venous return can cause problems when heat-induced increases in cutaneous blood flow are added to the circulatory demands of working muscle.[59]

EXHIBIT 6-2

PHARMACOLOGICAL AGENTS THAT MAY REDUCE HEAT TOLERANCE

Drugs of abuse (eg, cocaine, ethanol)
Anesthetic agents
Anticholinergics
Antidepressants
Antihistamines
Antihypertensives (sympatholytics)
ß-blockers
Diuretics
Lithium
Monoamine-oxidase inhibitors
Phenothiazines
Salicylates
Stimulants
Sympathomimetics
Thyroid hormone

Medications and Drugs of Abuse

Several classes of prescription medications diminish heat tolerance by increasing metabolic heat production, suppressing body cooling, reducing cardiac reserve, or altering renal capacity to defend fluid and electrolyte balance (Exhibit 6-2). Problems with pharmaceutical agents are especially likely to arise from new prescriptions or changes in dosage. Self-medication with over-the-counter agents can also cause difficulties.

Aspirin and other nonsteroidal antiinflammatory agents should be used with caution because they may reduce renal blood flow and thus predispose heat-stressed users to acute renal dysfunction. Although hyperthermia due to heat stress alone does not respond to antipyretics, use of these medications in febrile illnesses may be justified on the grounds that fever itself significantly increases the risk of heat illness.

Sedatives and narcotic analgesics affect mental status and may thereby suppress beneficial thermoregulatory behaviors. Alcohol is a common and serious problem because it impairs intake of food and water, acts as a diuretic, and disturbs judgment. The adverse effects of alcohol extend many hours beyond the time of intake, especially if the person has vomited.

REDUCING HEAT STRESS

When the thermal load exceeds the coping ability of fully trained, well-led, well-supported troops, the prevention of casualties requires the modification of work conditions. *This is also the only safe and effective means of reducing water requirements when supplies are limited.* Selection of optimal stress-reduction methods requires a dialogue between medical staff members and military leadership to develop a trade-off analysis of possible modifications to the three components of heat stress: work, the environment, and clothing (see Figure 6-1).

Environmental Heat Load

External heat load is a function of air temperature, humidity, wind, and solar load. Whenever possible, advance planning should include study of mean and extreme climatic conditions at the deployment site as described in atlases and in long-term weather records such as those maintained by the US Air Force Meteorological Center at Scott Air Force Base, Illinois. On arrival at the new site, staffs should establish communications links to obtain regular real-time weather data and predictions, because short-term weather phenomena may increase the risk of heat casualties or provide windows of opportunity for critical military operations.

Engineering techniques should be employed wherever possible to improve environmental conditions at work sites. Buildings and other enclosed spaces should be actively cooled by means of evaporative systems or refrigerated air conditioning. Conditions for outdoor work can be improved by using misting to lower air temperature, fans to increase air movement, and portable structures to provide shade. If the worksite cannot be improved, an alternative is to provide an air-conditioned space nearby for use during rest breaks. Arduous work that must be conducted in the open should be performed during the cooler hours and may have to be scheduled at night. Planners should consider the 24-hour pattern of temperature and humidity for the deployment site, as well as the times of sunrise and sunset. Note that effective thermoregulation depends on ambient water vapor pressure rather than relative humidity, as it is the skin-to-air difference in vapor pressure that allows the evaporation of sweat. Conversion among relative humidity, water vapor pressure, wet bulb temperature, and dew point can be accomplished using a psychrometric chart (which is discussed in the chapter by Santee and Matthew, Evaluation of Environmental Factors, in Volume 3 of *Medical Aspects of Harsh Environments*), provided that input values of temperature and humidity are simultaneous measurements and not averages representing differing times of day.

Trucks, tanks, and aircraft that have been parked in the open present thermal hazards because solar heating can produce extreme internal air temperatures and make surfaces hot enough to blister skin.[25] Such machines should be parked in shade whenever possible; the simple act of covering transparencies (eg, windows, portholes, and aircraft canopies) also provides substantial protection. High-performance jet aircraft present a special problem owing to the need to protect their crews from severe heat stress (Exhibit 6-3).[60,61]

Metabolic Heat Production

When military priorities dictate that neither environmental conditions nor protective clothing can be modified, the only remaining option for thermal control is to lower individual work load. In some cases, mechanical aids can be deployed to reduce human effort. For instance, dollies can be used to move equipment and supplies that could otherwise be carried by hand, and troops can be provided with mechanized transport. A second line of defense is to spread out the work over time. When the military situation allows prolongation of a task, the time-averaged level of effort can be reduced by adopting a work/rest schedule either developed on an ad hoc basis or calculated from a predictive model (see the section on predictive models later in this chapter); as much time as possible should be spent in shade. For time-critical tasks, work-load reduction can be accomplished by dividing the task among a greater number of personnel laboring simultaneously or in alternating teams.

A major influence on work/rest planning is the fact that recovery from hyperthermia is an inherently slow process. Although air conditioning or a cool shower provides symptomatic relief by lowering skin temperature, substantial reduction of core temperature requires 30 minutes under the best of conditions and much longer in a hot environment. It is difficult to monitor recovery; humans cannot directly sense their own core temperature, and its measurement in the field is usually impractical. Use of fixed rest intervals is one way to get around this problem. Alternatively, resting (unstressed) heart rate can be used as an indication of return toward baseline core temperature; a common technique is to count the pulse at 10-minute intervals after cessation of physical work.

Use of predetermined work/rest intervals has major disadvantages. Because of the wide variation in individual response to heat stress, fixed schedules waste potential work capacity of more resistant personnel while continuing to pose some risk of casualties among the most vulnerable. Self-pacing may be a practical alternative for workers who have previous experience under similar conditions. Recent advances in electronic instrumentation also make it possible to measure individual temperatures and heart rate for real-time display on a body-mounted unit or telemetry to a central monitoring station. Commercial versions of such systems are now available, but most use an algorithm to set work limits that may be unsuitable for military applications.

EXHIBIT 6-3

PROTECTING PILOTS FROM UNDUE HEAT STRESS

Crews of high-performance aircraft require effective protection from heat and dehydration in order to maintain both physiological resistance to inflight stress and ability to operate a complex weapons system under dynamic conditions. Specifically, aerial combat entails sequences of aerobatic maneuvers with levels of acceleration (G-stress), which challenge human tolerance limits, and heat stress lowers the threshold at which the crew may lose consciousness. Although fighter crews experience only limited physical work loads in the cockpit, flight clothing imposes a significant thermal burden for hot-weather operations. The multilayered, protective clothing includes cotton underwear, fire-retardant coveralls, antigravity suit, parachute harness, boots, gloves, and helmet. A chemical defense layer may be added as underwear or incorporated into the coverall. The process of dressing in this ensemble, walking to the aircraft, and conducting preflight inspection on a hot ramp significantly raises core temperature. Thus, it is an already warm crew that enters the cockpit of a heat-soaked aircraft and goes through the sequences required for engine start. Although modern fighter aircraft can cool the cockpit during ground operations (standby and taxi), the thick clothing and impermeable layers of the antigravity suit mean that the occupants receive only limited benefit. Typically, heat removal occurs so slowly that the aircraft is in combat or returning to base before cooling is complete. In wartime, crews are expected to fly two, three, or more missions in quick succession with little chance to achieve full recovery in terms of body temperature and hydration. The following procedures are designed to minimize heat stress impact under such conditions:

- keep the sun out of transparencies by using rolling roofs or fabric covers;
- precool cockpits by means of air-conditioning the ground carts;
- transport crew members directly to the aircraft;
- assign alternate crew members to perform preflight aircraft inspection;
- encourage crews to drink water before cockpit entry, during standby, and in flight;
- limit the permitted duration of in-cockpit standby;
- in cases of mechanical delay, allow only one change of aircraft before requiring return to Ready Room;
- optimize conditions for cooling and rehydration between flights; and
- support self-assessment and empower crews to stand down when they judge that further flights would be unsafe.

Protective Clothing

Thermal Costs of Protection

Protective clothing amplifies heat stress in many settings.[62,63] The military services face serious problems due to increasing requirements for use of chemical–biological defense garments (ie, mission-oriented protective posture [MOPP] gear, either with or without headgear).[64,65] Other items that contribute to heat stress include helmets, flack jackets, aircrew clothing, and special protective clothing that is worn when handling hazardous material. Such clothing impedes convective and evaporative dissipation of body heat to the environment and may also significantly increase the metabolic cost of movement by adding weight and hobbling movement.[64,66] The only thermal advantage of heavy clothing is its tendency to damp transient pulses of external heat, an important function of firefighter bunkers (ie, firefighter clothing that combines reflective and insulative protection).

Protective helmets and masks create special discomfort because they absorb solar heat and retain sweat. White or reflective helmet surfaces can reduce the radiant heating, but their high visibility makes them unacceptable for military use. Helmets fitted with a suspension harness allow some air movement over the scalp. However, holes in a close-fitting shell have little effect until the openings occupy a significant proportion of the surface, as in bicycle helmets. The use of face masks is often troublesome because the combination of heat stress with respiratory resistance and anxiety often produces hyperventilation.[67,68] Although aircrew members are accustomed to wearing visors and oronasal masks, ground troops must undergo thorough training to develop confidence in their ability to work while wearing respiratory protection. Use of chemical–biological protective facemasks in hot weather creates an additional problem: wearers cannot wipe their faces, so sweat runs into their eyes and causes pain with potentially disabling blepharospasm.

Interaction With Work Load and the Environment

Because heavy clothing isolates the body from the environment, ambient humidity is less important to those wearing heavy clothing, and the level of physical activity becomes a dominant factor in heat stress.[69,70] In addition, clothing diminishes or eliminates the advantages conferred by high levels of physical fitness and heat acclimatization, because it prevents evaporation of sweat from the skin.[12] Furthermore, the high sweat rates associated with acclimatization lead to wet clothing and rapid dehydration.

Continuous wear of chemical–biological protective clothing in moderate to hot weather requires major revision of work/rest schedules.[71] For this reason, the use of WBGT with a fixed "add-on" as a guide to work in heavy clothing[72] is not only simplistic but also can be misleading; more sophisticated models are required to provide valid predictions of tolerance.[12,69] To avoid the need for prescribed rest breaks, the US Army has developed modified clothing configurations for its MOPP levels to lower heat stress while maintaining appropriate levels of protection in the presence of low-to-moderate chemical and biological threats. (For a more complete discussion, see the chapter by Musa, Bandaret, and Cadarette, Protective Uniforms for Chemical and Biological Warfare, in Volume 2 of *Medical Aspects of Harsh Environments.)*

The thermal burden imposed by a particular clothing ensemble depends on its effective thermal insulation and resistance to transfer of water vapor.[73] A small role is played by absorption of solar radiation in the visible and infrared spectra, in which color is a minor factor. The addition of external items such as body armor and backpacks can significantly alter the thermal situation by adding weight, inhibiting air movement within the clothing, and obstructing the evaporation of sweat.

The thermal insulation of clothing is proportional to its thickness and depends primarily on the volume of air trapped within and between layers rather than the fiber from which the item is made.[73] Membranes that prevent liquid penetration impose very little insulation but drastically curtail air movement and the evaporation of sweat. Only limited improvement is offered by use of specialized semipermeable membranes. Although such high-technology materials increase water vapor transfer compared with impermeable materials such as polyvinyl chloride, semipermeable membranes still form a barrier to evaporation on the scale required for work in the heat.[74]

Heavy clothing has profound effects on the prescription of work/rest schedules.[70,75] For instance, performing very hard work while wearing chemical–biological protective clothing can induce such a rapid rise in core temperature that a safe schedule dictates very short bouts of work alternating with prolonged rest; because few tasks can be accomplished on such a schedule, it may be more practical to use a single work session of 30 to 40

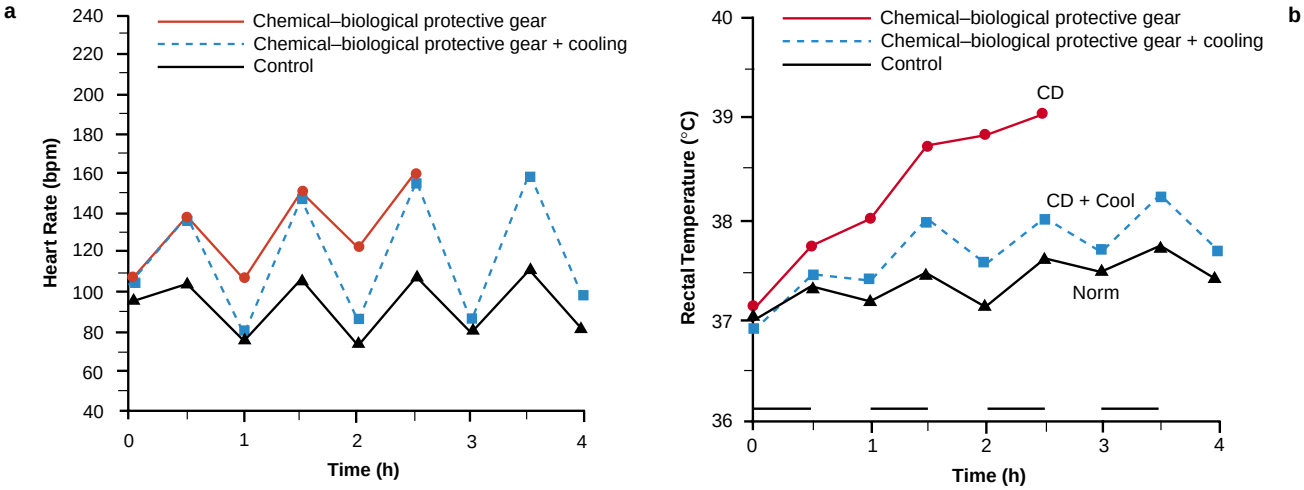

Fig. 6-3. An example of the effects of intermittent cooling in extending tolerance time for work in heat. Panels show (**a**) heart rate and (**b**) core temperature of subjects (n = 8) performing simulated work/rest cycles of ground crews. Bars indicate work intervals. Ambient conditions were T_{db} = 38°C, T_{wb} = 26°C, and T_{bg} = 43°C (db: dry bulb; wb: wet bulb; bg: black globe). Adapted with permission from Constable SH, Bishop PA, Nunneley SA, Chen A. Intermittent microclimate cooling during rest increases work capacity and reduces heat stress. *Ergonomics*. 1994;37(2):280.

minutes, followed by immediate retreat to collective protection for recovery. When personnel must continue to wear chemical–biological protective clothing after hard work, their rectal temperatures continue to rise for the first several minutes of rest, and subsequent cooling may be extremely slow. Under these conditions, personnel are acutely aware of their discomfort and may hyperventilate or become syncopal while resting. This phenomenon is a potential source of casualties among troops waiting to process into collective chemical–biological defense shelters at the end of work.

Gaining experience with heavy clothing in hot environments presents a special problem, because troops must achieve and maintain proficiency in their work while at the same time trying to avoid becoming heat casualties. Because the primary impediment to performance derives from the limitations to vision, communication, and manual dexterity, one response is to train wearing the headgear and gloves while substituting lightweight clothing for the heavy layers, thus eliminating the primary source of risk.

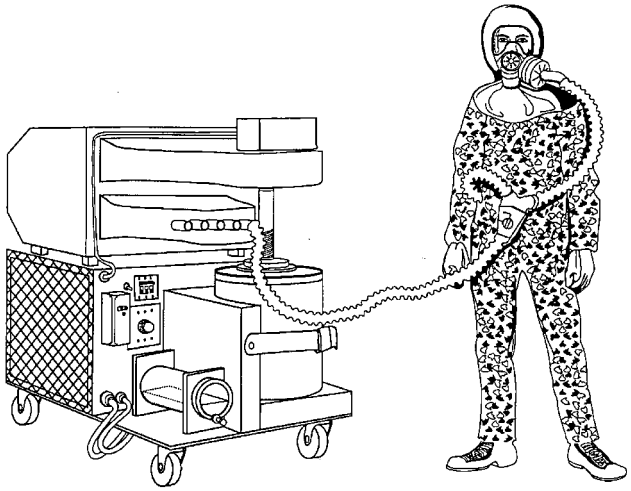

Fig. 6-4. The US Air Force developed a deployable system for air-cooling groundcrews during rest breaks with continuous wear of chemical defense clothing (otherwise known as chemical–biological protective gear or mission-oriented protective posture [MOPP] gear). Personnel wore an air vest under their garments and attached themselves to the cooling system while awaiting the arrival of the next aircraft. Cool, filtered air was provided by a device attached to a standard aircraft air conditioning cart.

Personal Cooling Systems

In an ideal world, personal cooling could be used to eliminate heat stress as a risk factor in military operations. Both air- and liquid-cooled garments have been studied for military applications.[76–78] In practice, technological limitations and the logistical costs of system support have limited their applicability. Individuals who can move freely with body-mounted cooling systems receive limited benefit because the added weight of the equipment increases the work load enough to offset most of the cooling. Practical applications are therefore limited to personnel who work in and around machinery, or at fixed locations where they can either be tethered to a mechanical heat sink or have frequent access to ice cartridges. To date, specific military applications of personal cooling systems include ice vests for personnel in ship engine rooms[79]; liquid-cooled garments for helicopter crews[80]; air vests for tank crews[81,82]; and air- and liquid-cooled garments for fighter pilots.[77] Certain tasks may require freedom during work but allow tethered cooling during rest breaks (eg, crews who rearm and refuel aircraft during surge operations). Laboratory experiments confirmed that intermittent cooling can substantially extend work capacity for subjects wearing chemical–biological protective ensembles (Figure 6-3),[83] and the US Air Force deployed an air-vest cooling system during the Persian Gulf War (Figure 6-4).

TOOLS FOR PREVENTION

Advance planning is critical to prevention of heat casualties. Fortunately, several tools are available to aid in this endeavor, among them training, predictive models, and surveillance.

Training

Medical staff members play key roles in providing education and supporting the realistic training exercises that underlie effective control of heat stress and prevention of heat casualties in hot climates.[5,84] Whenever possible, all deployable personnel should attend lectures and receive appropriate written materials well in advance of departure. Personnel arriving on site without such preparation should be briefed at once on issues related to heat stress. Commanders and unit leaders must be made aware of possible heat stress effects on performance and general health, as well as the potential tactical impact of heat casualties. They should also clearly understand that casualties are most likely when troops first arrive at their deployment site and during time-

EXHIBIT 6-4

MYTHS ABOUT HEAT STRESS

- Real men don't drink water.
- Don't drink unless you're thirsty.
- You can get a lot of cooling from a damp cloth on forehead, neck, or wrists.
- Training decreases the need for water.
- Sports drinks are better than water.
- Salt tablets counteract dehydration.
- Women are more vulnerable to heat than men.
- Baseball caps are good protection against the sun.

critical military contingencies.

Successful programs of prevention require universal education on sources and control of heat stress, normal responses, practical measures to maximize heat tolerance, signs of impending trouble in oneself and others, and appropriate corrective actions. Key points should be reinforced with relevant case reports or afteraction summaries to enhance individual and group resourcefulness and efficacy in dealing with heat stress. Education must also aim to stamp out common myths and erroneous assumptions that imperil health, because they induce inappropriate behaviors in the presence of heat stress (Exhibit 6-4). A dangerous example is the persistent belief that troops can somehow be trained or toughened to get along on reduced water rations, a concept that is incompatible with human physiology; military leaders *must* understand that water requirements can be reduced only by decreasing the need to sweat.

Predictive Models

It is often desirable to predict the effects of heat stress or recommend safe work/rest schedules under hot conditions. Various tables, equations, and computer programs have been developed to aid these processes. All of these can be called "models" in the sense that they use data and inference to develop a numerical description of human response to a given set of conditions (see also the chapter by Reardan and Pandolf, Modeling the Effects of Exposure, in Volume 3 of *Medical Aspects of Harsh Environments*). A computer program developed at USARIEM has been widely applied to US Army scenarios,[85] the US Navy uses an index developed in-house for shipboard operations,[86] and the US Air Force has its own specific guidance for operation of fighter aircraft in hot weather.[87]

A flag system is used to prevent heat casualties at US Department of Defense training installations (Table 6-1). Its development was prompted by the occurrence of heat casualties and deaths in US military training camps during World War II. Researchers developed the WBGT, a simple but effective index of environmental heat stress, to serve as a basis for modifying training activities in accordance with prevailing weather conditions.[2] A colored flag (green, yellow, red, or black) is displayed to indicate the current level of risk, and associated directives prescribe appropriate reductions in outdoor activity, differentiating between new trainees and fully acclimatized troops. An immediate and dramatic reduction in the incidence of heatstroke followed the implementation of the WBGT system.[6] WBGT has also been adopted as the basis for industrial work/rest schedules, where different boundaries may be used.[63,88]

More recently, computer-based mathematical models of heat stress have been developed in a number of laboratories. Their purposes range from the theoretical study of human thermoregulation[73,89,90] to empirical prediction of temperature rise and water requirements.[85,91,92] All such models require the input of multiple variables to describe work rate and clothing characteristics as well as heat, humidity, and other aspects of the environment. Simplified computer programs and derivative tables necessarily involve a number of assumptions, the details of which are often lost in the process of disseminating the information. Those who use such materials should carefully review the underlying assumptions and limitations to confirm that the model is applicable to their situation. Because small changes to input variables can have a major effect on output, users may wish to examine a variety of related scenarios to understand the effect of seemingly small variations (eg, an unexpected change in the weather, or the rescheduling of a task to a different time of day).

In any model designed to set safe schedules, the prescribed duration of work is set according to one of two criteria:

1. a single work bout, which is expected to produce the maximum safe core temperature, and following which the worker must return to base for recovery; and
2. a shorter work interval with a limited temperature rise, which can be repeated after a suitable rest interval.

TABLE 6-1

ACTIVITY RESTRICTIONS FOR OUTDOOR TRAINING OR PHYSICAL CONDITIONING IN HOT WEATHER

WBGT* °C (°F)	Flag Color	Guidance† for nonacclimatized personnel in boldface *Guidance for fully acclimatized personnel in italics*
25°C–26.9°C (78°F–81.9°F)	No flag	**Extreme exertion may precipitate heat illness** *Normal activity*
27°C–28.9°C (82°F–84.9°F)	**Green**	**Use discretion in planning intense physical activity** *Normal activity*
29°C–30.9°C (85°F–87.9°F)	**Yellow**	**Cancel intense physical activity; curtail other outside work** *Use discretion in planning intense physical activity*
31°C–31.9°C (88°F–89.9°F)	**Red**	**Stop work details and physical conditioning** *Curtail strenuous exertion, limit outdoor work to 6 hours*
= 32°C (= 90°F)	**Black**	**Cancel all outdoor work requiring physical exertion** *Cancel all outdoor work involving physical exertion*

*WBGT: wet bulb globe temperature
Calculation of WBGT: 0.7 T_{wb} + 0.2 T_{bg} + 0.1 T_{db}, where T_{wb}: wet bulb temperature; T_{bg}: black globe temperature; T_{db}: dry bulb temperature
†Guidelines assume that personnel are wearing summer-weight clothing; all activities require constant supervision to assure early detection of problems.
Adapted from HQ AETC/SGPB. *Prevention of Heat Stress Disorders*. San Antonio, Tex: Air Education and Training Command, Randolf Air Force Base; 17 Oct 1994. AETCI 48-101.

It should be noted, however, that neither body temperature nor hydration are likely to return to baseline during the prescribed rest breaks because complete recovery requires 30 minutes or more under cool conditions.

Heat-stress models generally express work and rest as subdivisions of 60 minutes. Although the work period cannot be safely lengthened beyond the prescription, the schedule can be divided into shorter intervals as long as the ratio between work and rest is preserved. For example, when a chart suggests 40 minutes of work alternating with 20 minutes of rest, a 20:10 schedule could also be used if better suited to a particular task.

Models are largely based on data from experiments on fit, young men and make no allowance for individual variation in physiological response. Although the more complex models allow adjustment for body size and fat content,[89] simpler empirical models do not.[85] Furthermore, models can safely predict the effect of conditions only within their envelope of experimental validation, when a valid model should produce satisfactory predictions in the hands of someone other than its developer.[70] Unfortunately, extrapolation from predicted core temperature to incidence of heat casualties remains largely speculative because it has not been possible to collect adequate experimental data on this relationship.

Surveillance

Surveillance and detection of signal events are important aspects of prevention requiring careful implementation (Exhibit 6-5). Leaders must be aware that heat casualties can occur suddenly and in large numbers. Therefore, seemingly minor complaints or signs of impaired performance among troops call for immediate corrective action to minimize deterioration in psychomotor performance as well as to prevent epidemic heat illness; stragglers or staggerers on a road march may indicate that the entire group is on the edge of serious trouble (Exhibit 6-6). Readers should keep in mind, however, that as currently defined, surveillance counts only casualties who have entered the medical treatment system and does not include those whose injury is too minor to require medical intervention.

In addition, the signs and symptoms of heat illness are not unique. Because desert and tropical climates also involve exposure to unfamiliar agents of disease as well as heat, medical personnel must always be aware of the need to exclude other potential diagnoses before settling on heat stress as the sole cause of a problem.

EXHIBIT 6-5

CURRENT APPROACH TO HEAT INJURY SURVEILLANCE

In recent years, the US Department of Defense (DOD) has significantly increased the emphasis on preventing disease and injury associated with military service. Under the general heading of Force Health Protection, a variety of preventive measures have been mandated, including disease and nonbattle injury (DNBI) surveillance on deployments. DNBI surveillance focuses on monitoring and controlling problems that could have a significant impact on a military force, including heat injuries.

The key elements of surveillance are systematic collection of health data, rapid analysis to identify problems, and corrective action based on the data. DNBI surveillance is essentially unit-based, and is conducted at every facility or unit that provides care for a deployed population. The diagnosis of every patient seen is recorded in a logbook. At the end of each week, each diagnosis is placed in a defined category, and the total number is counted. A weekly rate (expressed as percent per week [%/wk]) is then calculated for each category, based on the total population being cared for by the medical facility. Analysis consists of comparing this weekly rate to a standard reference rate. If the rate is higher than expected, further investigation and analysis is done to identify potential problems, and corrective action is then initiated.

The DOD DNBI surveillance category of heat injury includes the specific diagnoses of heatstroke, heat exhaustion, heat cramps, and dehydration. The heat injury category is designed to capture even the relatively mild cases that require treatment, because such cases may indicate breakdowns in the command's preventive efforts. A reference rate of 0.1%/wk is provided for comparison. If a unit's rate is appreciably higher than 0.1%/wk, then causative factors should be sought and corrected. Such factors might include lack of acclimatization, inadequate access to water, or failure to follow established guidelines for the ambient conditions. Immediate feedback should be given to unit commanders on the elevated rates and the probable causative factors. Commanders (rather than medical personnel) are usually in the best position to correct the problems that are causing heat casualties. In many cases, action can be taken immediately after recognizing that an abnormal number of heat injuries has occurred, without waiting to calculate weekly rates.

Although DNBI surveillance is currently mandated only for deployments, it is a useful tool for military units in any environment, including in garrison. This is especially true for heat injuries, which are a significant threat to units doing routine training during the warmer months. The heat injury rate is a very useful outcome measure for how well a unit is protecting its personnel.

Exhibit prepared for this textbook by Kevin Hanson, MD, MPH, Captain, Medical Corps, US Navy; Director, General Preventive Medicine Residency, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799

SUMMARY

Heat casualties can impose significant penalties on military operations in hot climates, including both desert and tropical areas of the world. The spectrum of effects runs from subtle psychomotor impairment to discomfort, disability, and death. Medical officers should be prepared to assist commanders with planning and trade-off analyses of practical alternatives, to minimize the impact of heat stress on the mission while preserving the health of personnel to the fullest extent possible.

Heat stress results from the combined effects of three factors: (1) environmental heat load, (2) metabolic heat production, and (3) protective clothing. Individual response to a given stress varies with age, physical conditioning, and the presence of additional factors such as sleep deprivation or intercurrent illness. While most studies of thermal stress have been conducted on healthy young men, deployed personnel may include both genders, a range of ages, and some persons using prescription medications that affect thermoregulation.

Human heat tolerance can be optimized through medical screening to disqualify unusually vulnerable individuals, systematic physical conditioning, and gradual acclimatization to work in heat. Special attention must be paid to living arrangements at the deployment site, including arrangements for personal hygiene, laundering of clothes, and appropriate sleeping arrangements. Sanitary handling and storage of food and water assume critical importance under hot conditions, where gastroenteri-

EXHIBIT 6-6

MARINE CAPTAIN COURT-MARTIALED AFTER RECRUIT'S HEATSTROKE DEATH

On 8 April 2000, a Marine captain was found guilty of dereliction of duty in the heatstroke death of a reservist under his command. The captain was court-martialed after the Marine collapsed and died following a conditioning hike. The 180-man Marine company, all carrying weapons and packs, made an 8-mile night march at Camp Lejeune, North Carolina, on a July night with an ambient temperature of 80°F. The Marine who died was a 21-year-old college student. He was seen vomiting at the first rest stop and later was heard telling a noncommissioned officer that he "couldn't make it," but was nevertheless pressed back into the formation. After the hike, he was seen to be lethargic with slurred speech; he wandered off and his body was found 2 hours later.

Testimony at the court-martial indicated that the march was conducted at a fast pace with few breaks, and that the captain appeared to be in a hurry to complete the march and go home. It was reported that many Marines became overheated or ill and straggled out of the captain's sight. Three Marines who were checked for hyperthermia had body temperatures exceeding 103°F.

The prosecution charged that the captain did not "follow established procedures for training marches, normally conducted at a slightly slower pace and with more rest stops than hikes for seasoned marines"[1(pA8)]; and that the captain "violated standing operating procedures for conditioning hikes and that he showed a careless disregard for his men."[2(pA4)]

This case exemplifies the risk of heat exhaustion and heatstroke during sustained exertion—even at night—if both temperature and humidity are high. In addition, the commander failed to make due allowance for green troops or to heed the signal events when numbers of trainees fell out during the march.

(1)Associated Press. Marine captain goes on trial for reservist's death. *The Washington Post.* 4 April 2000: A8. (2) Associated Press. Captain is convicted in death of Marine: Judge finds neglect of duty on fatal hike. *The Washington Post.* 9 April 2000: A4.

tis—a constant threat—can turn tolerable heat stress into life-threatening dehydration.

Frequent water intake is required to replace secreted sweat and prevent development of a significant water deficit. Under some conditions, sweat rate may exceed the maximum rate at which the human gut can absorb water (1.5 L/h); under such conditions, work load must be reduced or breaks provided to avoid progressive dehydration. The limited quantity of electrolytes lost in sweat can generally be replaced in meals, so that electrolyte drinks are required only for troops who are unable to eat regularly or for seriously dehydrated individuals.

Techniques for reduction of heat stress include (1) scheduling work for cooler times of day or night; (2) moving activities to cooled or shaded sites; and (3) reducing metabolic heat load through use of mechanical devices, spreading the work among more personnel, or instituting planned rest intervals.

Military contingencies may require wearing protective clothing such as MOPP ensembles, which interfere with convective and evaporative cooling of the skin; such clothing nullifies the thermoregulatory advantages of physical fitness and heat acclimatization. Clothing made with semipermeable membranes offers negligible relief under these extreme conditions. However, pilots and others working with machinery may be able to use air- or liquid-based personal cooling garments to improve comfort and performance of complex tasks.

Effective advance planning is the key to prevention of heat casualties on deployment. Useful tools include training of all personnel, use of models to predict and control heat stress levels, and surveillance and detection of signal events before casualties reach elevated levels.

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Chapter 7

CLINICAL DIAGNOSIS, MANAGEMENT, AND SURVEILLANCE OF EXERTIONAL HEAT ILLNESS

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INTRODUCTION TO THE PATHOPHYSIOLOGY OF EXERTIONAL HEAT ILLNESS

Exertional heat illness (EHI) encompasses a spectrum of disorders deriving from the combined stresses of exertion and thermoregulation (Figure 7-1). These include exertional dehydration, heat cramps, heat exhaustion, heat injury, heatstroke, rhabdomyolysis, acute renal failure, and hyponatremia.[1–4] Early in the course of EHI it may be difficult or impossible to distinguish these entities and, in fact, they often overlap and are differentiated as the clinical manifestations evolve. They represent primarily a continuum of multisystem illnesses related to elevation of body core temperature and the metabolic and circulatory processes (including changes in fluid and electrolyte balance) that are brought about by exercise and the body's thermoregulatory response.[5–9]

Heat dissipation occurs primarily at the skin. The blood carries body thermal energy to the skin, where the heat is dissipated through conduction, convection, radiation, and evaporation. A large temperature differential between the skin and its surroundings provides efficient heat dissipation under cool conditions.[10,11] However, under hot conditions there is inefficient heat dissipation, and blood flow to the skin increases dramatically, even at rest, thereby requiring a large increase in cardiac output.[12–14] With strenuous exercise there is a dramatic (3- to 6-fold) increase in cardiac output owing to increased blood flow to exercising muscle (Figure 7-2). Exercise elevates body temperature because at least three fourths of energy released during exercise is converted to heat. As body temperature rises, more blood flows to the skin for heat dissipation. These circulatory demands of sustained exercise and heat stress may also encroach on visceral blood flow to the extent of producing organ dysfunction or cellular injury (eg, watery/bloody diarrhea often seen in marathon runners, and perhaps acute renal failure and encephalopathy often seen in EHI).[15,16]

Heat exposure and regular strenuous exercise produce *heat acclimatization*, which improves the body's response to heat stress within a few days. Most of the physiological improvement in heat tolerance occurs within 10 days of combined heat exposure and regular exercise.[17–22] In acclimatized individuals blood volume increases, stroke volume increases, the heart rate is lower, metabolic generation of heat decreases slightly, sweating begins ear-

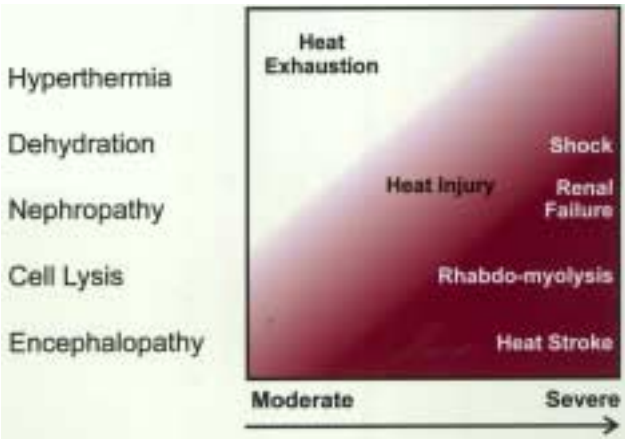

Fig. 7-1. A diagrammatic depiction of the spectrum of exertional heat illness, encompassing the continuum of mild (heat exhaustion), moderate (heat injury), and severe (heatstroke and other syndromes) in terms of severity in each area of physiological dysfunction: hyperthermia, dehydration/cardiovascular dysfunction, nephropathy, cell lysis (muscle or liver tissue damage), and encephalopathy. The horizontal arrow depicts severity of specific symptoms and signs corresponding to the categories of dysfunction on the left. The degree of shading depicts the severity of overall illness.

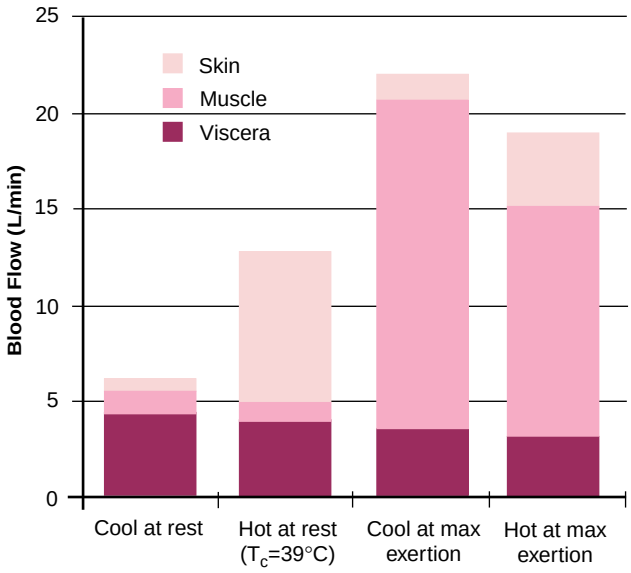

Fig. 7-2. Estimated distribution of blood flow to the skin, muscle, and viscera as fractions of cardiac output, under cool and hot conditions, at rest, and at maximum exertion. Adapted with permission from Gardner JW, Kark JA. Heat-associated illness. In: Strickland GT, ed. *Hunter's Tropical Medicine*. 8th ed. Philadelphia, Pa: WB Saunders; 2000: 141.

lier with a higher sweat rate and lower sodium content, and the threshold for cutaneous vasodilation is reduced.[11,23–32] These changes improve transfer of body heat from the core to the skin and enhance heat dissipation at the skin. Although sodium is conserved with heat acclimatization, water losses are not reduced because sweat volume increases.

Wind, solar radiation, and humidity play important roles in the efficiency of heat dissipation through convection, radiation, and evaporation. When the surrounding temperatures are at or above that of the skin (about 36°C [96°F]), then evaporation is the only mechanism for cooling. As more body heat must be dissipated, these fluid losses increase due to heavier *sweating*, which may exceed 2 L/h (up to a maximum of 12–15 L/d).[9,32] In hot and humid environments there is inefficient heat loss through convection and evaporation.[33] The resultant increased body core temperature induces heavier sweating, but dripping sweat provides minimal heat exchange, resulting in large fluid losses with minimal impact on core temperature reduction.[34]

As strenuous exercise produces more body heat than can be efficiently dissipated, core temperature continues to rise, sweating continues at maximal rates, and dehydration (ie, reduction of total body water, with some resultant reduction in plasma volume) rapidly develops, compounding the cardiovascular and metabolic stresses of exercise and thermoregulation.[35–37] These stresses may combine to produce orthostatic hypotension,[38] electrolyte imbalance,[8,39] liver and muscle cellular damage,[40] acute renal failure,[39,41] disseminated intravascular coagulation (DIC),[42–45] and neurological disturbances.[1,46] The spectrum of illness is thus quite broad, portraying the effect of these stresses on a variety of body functions.

Hyponatremia is an unusual consequence of excessive hydration in an overly aggressive attempt to prevent or treat EHI. Hyponatremia in this circumstance seems to be due to failure of the kidneys to excrete excess water. The problems of hyperhydration and hyponatremia are discussed later in this chapter and also in the chapter by Montain, Hydration, in Volume 3 of *Medical Aspects of Harsh Environments.*

CLINICAL FEATURES OF EXERTIONAL HEAT ILLNESS

We include all of the exertion-related heat illness syndromes within the term EHI. These syndromes form a continuum of multisystem illnesses, which may be divided into three main levels:

1. *mild* EHI, which includes heat exhaustion, mild dehydration, and heat cramps;
2. *intermediate* EHI, which includes exertional heat injury and mild rhabdomyolysis, renal insufficiency, orthostatic hypotension, heat-related syncope, and reversible electrolyte and metabolic disturbances; and
3. *severe* EHI, which includes heatstroke, severe rhabdomyolysis, liver necrosis, acute renal failure, cardiovascular collapse, and marked electrolyte or metabolic disturbances.

In addition, there are some minor heat-related syndromes, not part of the EHI continuum, which include heat rash (prickly heat), heat edema, parade syncope, exertion-associated collapse, and sunburn. There has been no agreement on criteria for separating EHI into levels of severity, so precise definitions are a matter of personal choice. Differentiation of heat syndromes by body core temperature has been popular, but this is unreliable because the severity of organ dysfunction correlates poorly with the maximum body temperature.

Exhibit 7-1 lists the wide variety of symptoms and signs associated with EHI. Early in the course of illness, slow mentation with impaired judgment, weakness, fatigue, headache, thirst, hyperventilation, muscle symptoms, or gastrointestinal symptoms may predominate. As the illness becomes worse, orthostatic symptoms develop such as faintness, ataxic gait, visual disturbances (eg, blurred vision, narrowed or tunnel vision, scotomata), and collapse.[1,47–50] Collapse may occur with or without loss of consciousness. Most severe symptoms are also accompanied by extreme mental status changes.[8,51] The major effects of EHI on clinical status are related to

- water and electrolyte loss,
- encephalopathy and other neurological problems,
- impaired renal function,
- muscle cell damage (rhabdomyolysis) with potential progression to the cell lysis syndrome,
- unusual complications, and
- consequences of life-threatening complications.

Rather than emphasizing distinct syndromes it is best to anticipate these manifestations, assess the appropriate level of monitoring and treatment needed for each, and derive a management plan

EXHIBIT 7-1

CLINICAL FEATURES OF EXERTIONAL HEAT ILLNESS

Nonspecific Symptoms

Thirst	Myalgia	Poor concentration	Hysteria	Vomiting
Weakness	Cramps	Impaired judgment	Headache	Diarrhea
Fatigue	Hyperventilation	Anxiety	Nausea	

Progressive Orthostatic Signs and Symptoms

Mild Symptoms

- Faintness
- Dizziness (not vertigo)
- Wobbly legs
- Stumbling gait
- Visual: blurred vision, tunnel vision, scotomata, blackout
- Collapse (without loss of consciousness)

Exertional (Heat) Syncope

- Collapse with brief loss of consciousness (< 3 min)

Orthostatic or Sustained Hypotension

- Evaluated via tilt tests

Shock or Cardiovascular Collapse

Common Presentations

Mild Exertional Heat Illness

- Nonspecific symptoms, mild orthostatic signs and symptoms
- Brief nonfocal encephalopathy, cooperative with medical care
- Hyperthermia clears immediately with cooling and rehydration
- Mild elevated creatinine without oliguria
- Mild elevated muscle enzymes

Intermediate Exertional Heat Illness

- Exertional syncope with electrolyte disturbance
- Nonfocal encephalopathy, uncooperative with medical care, amnesia
- Hyperthermia > 104°F
- Elevated creatinine with oliguria that responds to rehydration
- Elevated muscle and liver enzymes, mild acidosis

Severe Exertional Heat Illness

- Persistent hypovolemia or shock, metabolic acidosis
- Encephalopathy with delirium, obtundation, or coma
- Hyperthermia > 106°F
- Acute renal failure
- Rhabdomyolysis with life-threatening complications

Uncommon Clinical Problems

- Complications including ischemia, hypoxemia, and infection
- Hyponatremia
- Midline cerebellar speech or gait ataxia
- Seizures
- Hypoglycemia
- Gastrointestinal bleeding
- Liver failure or necrosis
- Pulmonary edema
- Cardiac arrhythmia
- Disseminated intravascular coagulation

based on the potential development of these problems.

The differential diagnosis of symptoms associated with EHI is broad and varies with locality and time. Most of the clinical findings associated with EHI are also found in other diseases. These diseases may provoke or accompany EHI, thus increasing the severity of the illness and the risk of serious complications. Infectious diseases are likely to provoke EHI by contributing to dehydration and hyperthermia. It is particularly important to consider meningitis, sepsis, pneumonia, myocarditis, viral infections, asthma, drugs and toxins, sickle cell disease, and cardiovascular or cerebrovascular disease.[1,4,50] The differential diagnosis list for patients with high body temperature also includes malaria, Rocky Mountain spotted fever, other infections, anticholinergic poisoning, neuroleptic malignant syndrome, and thyroid storm.[39]

Hyperthermia

In EHI, the elevated body temperature is not referred to as fever but as hyperthermia.[2] *Fever* regulates the body core temperature to a higher value (100°F–106°F [38°C–41°C]) as a result of change in the thermoregulatory set point (usually in response to inflammation). Heat production increases only temporarily to achieve the new set point temperature. Attempts to cool the patient (except by administering antipyretic drugs, which return the set point toward normal) are resisted by the patient's thermoregulatory responses (cutaneous vasoconstriction, shivering); and if attempts to cool the patient are successful, the body core temperature will rise again once cooling is stopped.[11]

On the other hand, *hyperthermia* refers to any significant elevation of core temperature above its normal level, and fever is only one of three ways in which hyperthermia may be produced. Hyperthermia is also a normal accompaniment of sustained exercise, and during exercise the elevation of core temperature is the chief stimulus that elicits the thermoregulatory, heat-dissipating responses that are necessary to dissipate the heat produced by the exercise. Thus, during exercise an equilibrium is achieved in a hyperthermic state. (The differences between fever and the hyperthermia of exercise are discussed in greater detail in Chapter 2, Human Adaptation to Hot Environments.) Finally, hyperthermia results if the sum of heat production and heat gain from the environment exceeds the body's level of heat dissipation to the environment. Such a situation may result from physiological factors (eg, impaired ability to sweat or to increase skin blood flow) or environmental factors (eg, extreme environments or the wearing of heavy impermeable clothing), and is a common circumstance in which EHI develops.

In EHI, hyperthermia is not the result of an elevated set point, even though there is evidence that inflammatory processes may be involved (see Chapter 5, Pathophysiology of Heatstroke). Patients with EHI usually do not shiver during cooling (unless they are overcooled), and once the heat source is eliminated and cooling is completed, a rise in body temperature is uncommon. Recurrence of hyperthermia without an added heat source implies that inflammatory processes have progressed.

While severe hyperthermia (≥ 106°F [41°C]) is sometimes considered a distinctive or defining feature of exertional heatstroke, it is more appropriate to consider it to be a common feature of the more severe forms of EHI. Survival has been reported in a patient with maximum rectal temperature of 116°F [46.5°C].[52] However, severe cases of EHI also occur at lower body core temperatures,[53–55] and many patients with high rectal temperature do not manifest the severe neurological symptoms or organ damage that define heatstroke. We have seen considerable dissociation between hyperthermia and signs of nonfocal encephalopathy that characterize exertional heatstroke. In one series of 468 cases of EHI in Marine Corps recruits, only half of the casualties with neurological symptoms consistent with heatstroke (delirium, obtundation, or coma) had maximum rectal temperatures of 106°F (41°C) or higher.[56] And half of the casualties with maximum rectal temperature of 106°F (41°C) or higher had milder forms of EHI inconsistent with heatstroke.

Dehydration and Electrolyte Imbalance

Volume depletion, with loss of water and salt, is one of the fundamental features of EHI. Both the physical signs and the laboratory abnormalities that are characteristic of volume depletion are relatively insensitive and largely nonspecific. The clinician often assumes the presence of mild-to-moderate volume depletion based on the clinical situation without confirmatory findings, and observes the patient's response to a trial of fluid and salt replacement to make the diagnosis retrospectively. Interstitial volume depletion may result in dry oral mucous membranes, loss of skin turgor (tenting), and dry tongue with longitudinal furrows. Signs of plasma volume depletion are orthostatic hypotension, syncope, recumbent hypotension, and shock; as well as lethargy, fatigue, loss of mental concentration, confusion, and obtundation. Moderate or severe

volume depletion produces oliguria. Important laboratory signs of water depletion include hypernatremia, hyperosmolality, elevated blood urea nitrogen (BUN) to creatinine ratio, elevated hemoglobin concentration and hematocrit, and high urine specific gravity.

The most common situation seen in EHI is a patient who is mildly dehydrated from prior heat stress and who then undertakes heavy exercise without adequate replacement of water losses. This usually results in hypernatremic hyperosmolar dehydration, which in its earlier stages is adequately treated with water and rest in a cool area. More severe cases (particularly those who have suffered exertional syncope, exhibit orthostatic hypotension, or have exertional heat cramps) benefit from intravenous infusion of 1 to 3 liters of half-normal saline. In our studies of EHI in Marine Corps recruits at Parris Island, South Carolina, about one third of episodes presented as exertional syncope during or immediately after strenuous exercise.[56–58]

Syncope must be distinguished from sustained obtundation, coma, or, rarely, a seizure. Guidelines for making this assessment are given in Exhibit 7-2. *Syncope* is characterized by its brief duration; frequent recall of onset; absence of abnormal tone, posture, or movement during unconsciousness; and abrupt return to consciousness. Antegrade amnesia lasting beyond 3 minutes after the collapse suggests a neurological injury other than uncomplicated syncope, while amnesia retrograde to collapse does not differentiate the cause of collapse. *Seizure* is often characterized by a stereotypic aura or prodrome, with incontinence, abnormal eye position, clonic–tonic movements, focal deficits, and a postictal stage with amnesia lasting beyond unconsciousness. *Coma* is recognized by unresponsiveness for more than 3 minutes following collapse, without the characteristic signs of seizure.

Other electrolyte problems include hypokalemia or hyperkalemia, low bicarbonate levels, and hyponatremia. Hypokalemia of uncertain etiology has been found in about one third of large case series of exertional heat stroke, and is a factor in the development of rhabdomyolysis.[43,50,59–62] Hyperkalemia results from acute renal failure, tissue necrosis, and metabolic acidosis. Lactic acidosis produces low bicarbonate. And hyponatremia usually results from extreme overhydration (discussed below).

Dehydration is most often associated with both

EXHIBIT 7-2

EVALUATION OF LOSS OF CONSCIOUSNESS

Syncope:	Nearly always occurs when upright
	Skin becomes pallid or ashen gray
	Brief prodrome (giddiness, swaying, loss of vision, nausea)
	Victim recalls onset as start of visual black out or fall
	Recovery within seconds to 3 minutes
	Abrupt return to full consciousness
	Amnesia only from time of collapse to awakening
Seizure:	Not dependent on erect posture
	Often starts with stereotypic aura or prodrome
	Often stereotypic posture or movement
	Bilateral stereotypic movements always accompanied by coma
	Incontinence (urinary more often than fecal)
	Rolling up of eyes, biting of tongue, foaming at mouth
	Often unconscious for > 5 minutes, with gradual recovery
	Postictal state with lethargy and confusion for minutes to hours
	Amnesia from prodrome through much of postictal stage
Coma:	Prolonged unresponsiveness lasting > 3 minutes
	Slow recovery without treatment (hours to days)
	If due to exertional heat illness, may reverse in minutes with cooling and hydration

water and salt depletion, so that most patients have nearly normal serum sodium levels.[63] Dehydration may be due to isotonic or hypotonic fluid loss.[1,8,30,64] Water losses are universal, but salt loss may be excessive in unacclimatized individuals, who excrete sweat with high salt concentration. This occurs primarily during the first 2 weeks of training in a warm environment. Acclimatized individuals excrete large volumes of sweat with low salt content, and thus hypotonic fluid losses predominate.[2] These patients need free water replacement as well as isotonic solutions. In predominant water depletion (usually from limited water consumption), there is characteristically intense thirst, hyperventilation with paresthesias, tetany, hypernatremia, and renal insufficiency. In predominant salt depletion (usually from limited salt intake), there are characteristically generalized muscle cramps, vomiting, and hyponatremia.[1,5,8]

Neurological Manifestations

Neurological symptoms are characteristic in EHI, particularly in exertional heatstroke.[3,65] These symptoms are not focal neurological deficits relating to specific cranial or peripheral nerves but represent a *generalized encephalopathy* affecting mental status. A precise description of this encephalopathy is not available in the literature. The symptoms range from lethargy and drowsiness, through confusion and disorientation, to delirium, obtundation, or coma. No generally accepted methodology for quantifying heat encephalopathy exists. However, we have developed and used an acute encephalopathy scale in our collection of research data on cases of EHI at the Parris Island Marine Corps Recruit Depot (Exhibit 7-3).[57,58] This scale was adapted from the Los Rancheros Scale,[66] the Reaction Level Scale,[67,68] and the Glasgow Coma Scale.[69]

Other neurological manifestations include frequent amnesia and, rarely, seizures or persistent ataxia. A *thorough neurological examination* is important. This consists of the following steps: assess pupillary size and response to light; accommodation; conjugate movement to six positions; opening and closing of eyes; drooping of lids; and response to pain in each hand, foot, the trunk, and both sides of the face. Assess ability to respond to voice and to move each limb, the neck, and the face. Ask the patient to perform simple and complex commands. Assess ability to respond with questions defining orientation to person, place, and time and, if needed, proceed to performance testing such as the Mini-Mental State Examination.[70] Assess higher function and speed of response in the history. Question the patient for amnesia, confusion, poor concentration, and judgment during the periods before and after the initial signs of EHI. Exhibit 7-4 suggests some questions for assessment of mental status.

Amnesia is present when loss of memory extends outside the period of loss of consciousness. With *syncope*, however, the patient usually recalls a prodrome (starting to black out or fall) and recovers abruptly and completely; loss of memory will exist for the period during the syncopal episode (< 3 min). If the patient does not remember passing out but others witnessed it, then he has amnesia. The duration of amnesia should be established (eg, patients may black out for less than a minute but be unable to recall the 10 minutes spent in getting into the ambulance and traveling to the clinic).[65] In our studies of Marine Corps recruits at Parris Island, about one third of EHI cases had a period of amnesia lasting at least 10 minutes.[56–58]

The *history of witnesses* is crucial to neurological assessment. An observer needs to report whether the person fell to the ground, fainted, suffered loss of consciousness (and its duration), was slow, lethargic, dazed, difficult to arouse, confused, disoriented, combative, obtunded, comatose, or had a seizure. The patient generally cannot reliably describe these manifestations. Many patients with EHI have mild deficits, especially slow mentation with a dazed affect, short periods of amnesia, or confusion and disorientation, which may only be documented by an appropriate history from witnesses.[65] It is very important to describe the chronological sequence of symptoms and signs and to explain the precise medical events for "falling out" of an activity (eg, stopping, collapsing to the ground, having a seizure, or fainting).

Seizures are usually related to hypoxia or late manifestations of advanced exertional heatstroke. Occasionally, seizures result from hyponatremia, hypoglycemia, or other disturbances.[71]

Impaired Renal Function

Acute renal insufficiency is a frequent component of EHI that is often relatively easily reversed. It is believed to be due to prerenal volume depletion, reduced perfusion of the kidney due to redirection of blood flow to the muscle and skin, and acute renal tubular injury from myoglobin and myoglobin casts. Clearly, the pathogenesis is more complex but the contributions of other factors, such as cytokines, have not been fully characterized. Acute renal failure is hard to detect in its early stage because there are

EXHIBIT 7-3

PROPOSED PARRIS ISLAND SCALE FOR NONFOCAL ENCEPHALOPATHY

The simplicity of the scores for the Parris Island Scale (Exhibit Table 1) allows rapid assessment of the global level of central nervous system function for patients with heat illness. The Glasgow Coma Scale[1] scores specific responses but does not assess global function. The Parris Island Scale is analogous to the Los Rancheros Scale,[2] which describes central nervous system function following closed head injury. The observations required for severe dysfunction are identical to those used in the Glasgow Coma Scale, but these are categorized as *functional* levels. To more precisely describe obtundation (ie, minimal conscious function), we adapted the validated definition of the Reaction Level Scale,[3] which also utilizes the observations required for the Glasgow Coma Scale. At higher levels of function we retain those features of the Los Rancheros Scale that apply to acute illness. During acute exertional heat illness, the time course may be so rapid that detailed evaluation of each level of encephalopathy may be difficult.

EXHIBIT TABLE 1: PARRIS ISLAND SCALE

Parris Island Scale	Level of Function	(For Comparison) Glasgow Coma Scale	Los Rancheros Scale
8	Normal: alert, oriented, cooperative	15	8
7	Drowsy or Lethargic, or Dazed Affect: slow mentation, poor concentration, fully arousable	13–15	7
6	Confused and Appropriate: incomplete orientation, cooperates with medical care	12–14	6
5	Confused and Inappropriate: disoriented, uncooperative, purposeful, directed	11–13	5
4	Delirious: disoriented, agitated, combative, out of touch, fragmented behavior, poorly directed	10–12	4
3	Obtunded: minimal mental response; can obey a command, ward off pain, utter a word, or make eye contact	9–12	3
2	Light Coma: unconscious; reflex response to pain	4–8	2
1	Deep Coma: unconscious; no response to pain	3	1

Patients with mild dysfunction present with slow mentation, poor concentration, and dazed affect (Level 7). Drowsiness is identified by droopy or intermittently closed eyelids.[3] Patients between Levels 6 and 4 may have a normal, reduced, or increased level of arousal. Assessment can be made quickly by determining orientation to person, place, and time; observing response to simple and complex commands; and noting spontaneous actions and cooperation with medical care. At Level 6 a patient has incomplete orientation but is cooperative and appropriate even for painful procedures. At Level 5 a patient is disoriented and uncooperative, but behavior is directed, purposeful, and appears related to real events. At Level 4 (delirium) a patient is disoriented and behavior is fragmented, poorly directed, and often appears unrelated to external stimuli. Combative behavior is common among young military personnel in delirium. Obtundation (Level 3) is defined as somnolence with minimal conscious function. The best performance an obtunded patient can accomplish is at least one of the following: (1) obeys a one-step command, (2) wards off pain, (3) utters a single word, or (4) makes sustained eye contact during movement. Patients in coma are unable to demonstrate any of these basic signs of mentation. Patients in light coma (Level 2) respond to pain, whereas those in deep coma (Level 1) do not.

We hope and expect that clinical investigations will show the predictive power of the Parris Island Scale in terms of patient outcome and the need for treatment.

(1) Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974;2:81–84. (2) Hagen C. Language-cognitive disorganization following closed head injury: A conceptualization. In: Trexler LE, ed. *Cognitive Rehabilitation: Conceptualization and Intervention.* New York, NY: Plenum Press; 1982: 131–151. (3) Starmark JE, Stalhammar D, Holmgren E. The Reaction Level Scale (RLS85): Manual and guidelines. *Acta Neurochir (Wien)*. 1988;91:12–20.

EXHIBIT 7-4

QUESTIONS FOR ASSESSMENT OF MENTAL STATUS

- **Orientation:** (Normal: oriented to person, place, and time [3/3])
  1. Your name? Who are you? What are you doing here?
  2. What is the year? Season? Month? Day of week? Date?
  3. Where are you? What country? State? City? Building?
- Your last address and phone number before coming to this site?
- Could you concentrate normally and fully at the time of illness?
- Were you confused about anything while ill?
- **Amnesia:** Describe events from the start of activity to arrival here (especially just before and after any fainting episode). Include exertional activities and name three persons with you at the time of onset and describe one person traveling here with you.
- **Optional tests:**
  1. Calculate serial 7s (subtract 7s from 100: 93, 86, 79, 72, 65, …).
  2. Recall of three objects after 5 minutes.
  3. Recall of own Social Security number.

Examples of Patient Responses Indicating Confusion[1]:

"I just wasn't all there, thinking was fuzzy."
"I was getting dumb."
"I don't know where I was, I couldn't answer questions."
"I was having trouble doing simple math."
"I was disoriented, couldn't remember where I was."
"I couldn't think clearly, couldn't concentrate."
"I couldn't keep a thought in my head, forgot instructions."
"I don't remember if I went out or not, things became confused."

Source for patient response examples: (1) Carter BJ, Cammermeyer M. A phenomenology of heat injury: The predominance of confusion. *Mil Med*. 1988;153:118–126.

no symptoms or signs until sufficient time has passed for marked alteration in serum chemical levels regulated by the kidney. The most important early indicators of acute renal failure are inadequate urinary output, evidence of myoglobinuria, and elevated creatinine and BUN. After adequate rehydration, urinary output should exceed 50 mL/h and be approximately equal to fluid intake. While serum creatinine levels often increase during exercise, owing to release of creatine from injured muscle, elevations above 1.7 mg/dL are of concern.

Urinalysis is useful to determine whether there are signs of myoglobinuria (occult blood positive with pigmented casts but without red blood cells) or other problems contributing to impaired renal function. Mild proteinuria and modest abnormalities of the urinary sediment are frequently found in patients with heatstroke.[50] The presence of abnormal numbers of cells or cell casts in the urine is important. If urinary output remains inadequate after hydration, then urinary sodium and creatinine concentrations, fractional excretion of sodium, urine osmolality, and the urine-to-plasma creatinine concentration ratio are useful to distinguish between prerenal azotemia, which may be rapidly reversible, and acute renal failure, which is a standard indication for intensive hospital care.[72]

Although we generally think of *renal failure* as

part of rhabdomyolysis with myoglobinemia or heatstroke, acute renal failure can occur as the primary form of EHI without rhabdomyolysis, encephalopathy, or hyperthermia.[41,73–81] This was more common in the 1960s among military trainees who received salt tablets as prophylaxis or treatment for heat cramps.[41,73,74] Salt supplements cannot substitute for water replacement, and excessive salt intake increases water requirements. For this reason prophylactic use of salt tablets is inadvisable, and it is preferable to replace salt through regular meals.[8,50,82–84]

Muscle Symptoms and Rhabdomyolysis

Exertional heat cramps are painful, migratory skeletal muscle spasms. These appear most often in well-conditioned persons, at the end of the training day, or in the shower as muscles cool.[8,21,50] They are often attributed to salt depletion with resultant cellular hyponatremia and are rapidly reversed with water and salt replacement, usually by intravenous infusion of normal or half-normal saline. Oral salt solutions (0.5%) are also effective if the parenteral route is unavailable. Prevention is through adequate water replacement, a high-salt diet (salt tablets should not be used, particularly at the time of exercise), and slow progression of conditioning exercise.

Exertional rhabdomyolysis is the syndrome caused by skeletal muscle damage with release of cellular contents into the circulation, including myoglobin, potassium, phosphate, creatine kinase (CK), lactic acid, and uric acid.[85–87] Manifestations of rhabdomyolysis can vary from asymptomatic elevation of serum skeletal muscle enzymes to muscle weakness, pain, tenderness, and stiffness with associated myoglobinuria with or without acute renal failure.[77,88–91] In its most severe form obvious muscle necrosis can be demonstrated, but marked laboratory abnormalities can occur without extensive cell necrosis. Severe rhabdomyolysis may present without early muscle pain or tenderness, and muscle numbness may be the only symptom in the first few hours (documented in approximately one third of severe cases).[92,93]

Physical assessment of strength of major muscle groups and search for areas of numbness are important. Mild rhabdomyolysis with asymptomatic elevation of serum skeletal muscle enzymes is the most common laboratory abnormality in EHI. Serum levels of muscle enzymes usually peak at 24 to 48 hours after onset.[90,94] Early enzyme levels may not be helpful in making a diagnosis in the emergency department, whereas casualties with more severe rhabdomyolysis usually exhibit muscle numbness, weakness, pain, tenderness, and stiffness.

Exercising military trainees frequently develop CK values ranging from 500 to 1,000 U/L without other clinical abnormalities, but seldom above 1,200 U/L in the absence of EHI or acute muscle injury.[95] Other laboratory tests for cell damage include serum levels of uric acid, lactate dehydrogenase (LDH), aspartate aminotransferase (AST, formerly called SGOT), and alanine aminotransferase (ALT, formerly called SGPT).[8,90,96–98] LDH can also be released from erythrocytes, lung, and liver, while elevation of serum aminotransferases often reflects liver damage.

If skeletal muscle or liver necrosis is extensive, it produces the *cell lysis syndrome,* which consists of acute renal failure, hyperkalemia, hyperuricemia, a low ratio of BUN to urate, hyperphosphatemia, hypocalcemia, lactic acidosis, DIC, and hypotension.[99–102] These may progress to advanced renal failure requiring dialysis, infectious complications, pulmonary complications (eg, pneumonia, atelectasis, adult respiratory distress syndrome, pulmonary edema), shock with possible myocardial ischemia, bleeding disorders and organ damage from DIC, and death.[1,40,73,103–105] The chemical features and the management of the full-blown syndrome of cell lysis resemble the tumor lysis syndrome resulting from aggressive chemotherapy for highly responsive tumors.[106]

Exertional rhabdomyolysis may occur without elevation of body core temperature[55,107,108] or encephalopathy.[1,109] On the other hand, rhabdomyolysis occurs frequently as part of the clinical syndromes of exertional heatstroke and heat injury.[1,109] It is extremely important to closely monitor hydration, electrolytes, acid–base status, and fluid intake and output, because early aggressive parenteral correction of dehydration and metabolic disturbances is the basis of treatment. The early clinical picture may be deceptive because the patient may manifest minimal clinical symptoms in the presence of profound renal failure or metabolic abnormalities.[55]

Other Complications of Exertional Heat Illness

Hyponatremia

Several recent reports have described severe hyponatremia (Na < 130 mEq/L) associated with exercise in the heat.[110–116] This illness occurs primarily in the setting of forced water drinking (> 15 L/d) for prevention or treatment of early symptoms of EHI.[71,100,112,115–119] Casualties may present with repeated emesis or seizures due to brain swelling. These patients need urgent intensive care with management by appropriate specialists.

When hyponatremia develops slowly through sodium depletion, the brain mobilizes interstitial fluid and expels intracellular solute to avoid a dangerous degree of swelling; but if the osmolality of the extracellular fluid falls too rapidly, these protective mechanisms are not enough to protect the brain from dangerous degrees of swelling and increased intracranial pressure. Thus, the level of plasma sodium is, by itself, not a good indicator of the severity of the problem. If hyponatremia has developed slowly and there are no neurological symptoms due to increased intracranial pressure and brain swelling, the medical officer should correct the hyponatremia slowly. This is because too-rapid correction of hyponatremia can cause a dangerous demyelinating syndrome called pontine myelinosis.[120,121]

In exercise, however, hyponatremia is more commonly produced rapidly through excessive water drinking and water retention in the heat. In this circumstance the risk of demyelination is much less, and more rapid treatment is indicated in order to forestall or relieve pressure on the brain.[102,111,120,121] Owing to recent increases in military casualties with hyponatremia associated with heightened emphasis on fluid replacement during hot weather training, the US Army Research Institute of Environmental Medicine (USARIEM) has issued new fluid replacement guidelines that limit maximal water intake to 1 to 1.5 qt/h, not to exceed 12 qt/d.[122] These new guidelines appear to have reduced the rate of casualties with hyponatremia among army trainees.[123] These guidelines address only the maintenance of fluid equilibrium and body temperature (ie, prevention of EHI) and not treatment for EHI. Because early symptoms of hyponatremia may resemble mild dehydration, the duration of field treatment of casualties with even mild EHI should never exceed 1 hour, with a maximum of 2 quarts of fluid.[119] Otherwise, there is serious potential for significant exacerbation of hyponatremia through excessive fluid ingestion. Persistent, severe, or unusual symptoms or signs warrant immediate medical evaluation (including laboratory assessment).

Ataxia

While focal lesions of the central nervous system (CNS) are not generally seen in exertional heatstroke, the one exception is the rare development of midline lesions of the cerebellum, causing residual gait or speech ataxia. These unusual signs are characteristic of heatstroke when they occur (2 of 137 cases in our series at Parris Island).[56–58] These lesions often persist after complete recovery from the encephalopathy.[124,125] Presence of such ataxia following an episode of exertional rhabdomyolysis or heat injury is a strong indication that heatstroke was part of the acute heat illness syndrome, whether or not it was recognized at the time.

Seizures

Seizures usually occur late in advanced exertional heatstroke or after cardiovascular collapse, indicating a higher risk of permanent brain injury or death. This complication is common among cases of exertional heatstroke receiving delayed cooling and hydration. Several well-known early case series of exertional heatstroke have described an incidence of seizures at 50% to 70%.[39,43,53,126] In recent experience where treatment of EHI (cooling and rehydration) was initiated within minutes of collapse, early seizures have been uncommon except when associated with severe hyponatremia due to excessive water ingestion. Thus, the appearance of early seizures should engender suspicion of hyponatremia, hypoglycemia, hypoxia, cerebral hemorrhage, or other cause, in addition to heatstroke. These casualties need urgent intensive care with management by appropriate specialists.

Disseminated Intravascular Coagulation

DIC is an advanced complication of severe EHI in which there has been considerable tissue damage.[45,61,127–130] DIC usually develops between 18 and 36 hours after the onset of exertional heatstroke.[42] Rarely, it can develop in less than 1 hour.[127] DIC is an ominous sign associated with severe morbidity and high fatality rates. This complication is common among casualties of heatstroke who receive delayed cooling and hydration. However, it has not been observed in our experience with more than 200 cases of exertional heatstroke who received early cooling and rehydration.[58] Hemorrhages can occur in many organs, including the myocardium, lungs, brain, liver, muscle, and gastrointestinal tract.[42,53,127] Severe gastrointestinal bleeding most often follows shock, hypoxemia, and DIC.[15,131]

Hypoglycemia

Exertional hypoglycemia occurs only rarely but is important because immediate treatment prevents brain injury. Medical officers should consider the known causes, which include an abnormal postprandial response (seldom an issue during heavy exercise), endogenous insulin excess, severe hepatic insufficiency, alcohol intoxication, adrenocortical

insufficiency, hypothyroidism, or growth hormone deficiency. Usually the results of subsequent special studies, including tests for fasting hypoglycemia in the absence of exercise, are normal. The single documented case we have seen among more than 2,000 episodes of EHI in Marine Corps recruits and cadre presented as syncope and coma, which developed while standing in a tepid shower after correction of hyperthermia and clearing of mental confusion. The patient responded immediately to a 50-mL ampule of 50% dextrose in water, which was repeated. He then was given an infusion of 5% dextrose in half-normal saline for 24 hours. Subsequent tests for fasting hypoglycemia at rest were negative.

Liver Failure

Rhabdomyolysis is sometimes accompanied by signs of liver necrosis, especially in casualties who have life-threatening complications.[132–137] Occasionally there is little evidence of rhabdomyolysis, but severe liver necrosis produces a similar syndrome of acute cell lysis and renal failure, as discussed above. These cases are likely to be accompanied by DIC, aggravated by failure of the liver to synthesize the vitamin K–dependent factors and fibrinogen, and reduced liver clearance of fibrin degradation products.

Sudden Death, Cardiovascular Collapse, and Arrhythmia

Cardiovascular collapse (shock or a fall in blood pressure) is a relatively common complication in the early presentation of exertional heatstroke.[1,38,43,53,62] The role of cardiac arrhythmia is unclear, although spontaneous resolution of arrhythmia with cooling has been documented in several cases.[7,62,138] In nonfatal cases the blood pressure usually responds rapidly to cooling and rehydration, while failure to respond reflects a poor prognosis. Descriptions of myocardial infarction in exertional heatstroke are largely confined to cases following a period of prolonged shock or ischemia, or associated with severe hepatic or renal damage, as a late complication of heatstroke.[61,62] Several case reports have documented myocardial hemorrhage or necrosis in the absence of any coronary artery disease.[53,61,62]

There have been several published case reports of hyperthermia in exercise-related sudden death among young adults with no silent heart disease detected at autopsy or from medical history.[139] Most authors have assumed that these sudden deaths were from heatstroke, but there is controversy as to whether the mechanism of death should be considered primary cardiovascular collapse from distributive shock[38] or cardiac arrhythmia. Extreme hyperkalemia from rhabdomyolysis may also play a role.

We reviewed all EHI cases, serious cardiovascular events, and deaths among 269,000 recruits in Marine Corps basic training at Parris Island during a 12-year period. There were 7 life-threatening cardiovascular events (5%) among 137 patients during exertional heatstroke (2 sudden deaths; all 7 events were unexplained by preexisting disease from prior history or detailed autopsy) versus 4 (0.0015%) among 267,500 recruits without EHI (4 sudden deaths, with 3 explained by silent preexisting heart disease).[140] These data suggest that the risk for life-threatening cardiovascular events during exertional heatstroke is at least 3,000-fold higher than in exercise without EHI. Exertional heatstroke accounted for at least 7 of 11 fatal or serious sudden cardiovascular events in this population. This contrasts with autopsy studies of exercise-related sudden death among civilian athletes, which report that fewer than 1% of cases were due to EHI.[141,142] These preliminary data suggest that EHI should be considered in the management of exercise-related cardiac resuscitation and in the diagnosis of exercise-related sudden cardiac death.

Military recruits with sickle cell trait (hemoglobin AS) have a 10- to 30-fold greater risk for exercise-related death (unexplained by preexisting disease) than those without the trait.[139,142,143] Half of these excess deaths were due to EHI (heatstroke and rhabdomyolysis) and the other half were unexplained sudden deaths. Most exercise-related, sudden, unexplained deaths of recruits without hemoglobin S occurred in relation to heat stress. Preliminary analysis of a 10-year program to prevent EHI suggests that nearly all excess exercise-related deaths among recruits with sickle cell trait can be prevented, and that large reductions can also be obtained for exercise-related death in recruits without hemoglobin S.[144–146]

EXERTIONAL HEAT ILLNESS SYNDROMES

Exertional heatstroke is fairly common in healthy young adults undergoing strenuous physical training in warm and humid weather, but sedentary or classic heatstroke (included here, briefly, for completeness) is quite rare. *Exertional heatstroke* arises from sustained or heavy exertion, usually in hot environments.[1,147] Typically, onset is abrupt, occurring during or shortly after exertion, with orthostatic manifestations (faintness,

staggering, or visual disturbance) leading to collapse (with or without syncope), followed by confusion, combativeness, delirium, obtundation, or coma. This syndrome frequently evolves in minutes. If it is treated immediately with aggressive cooling and parenteral rehydration, then severe organ damage and mortality are almost always prevented.

Classic heatstroke, on the other hand, is generally associated with extended exposure to a hot environment in the absence of strenuous exercise and is a somewhat different illness.[1,148–150] It is seen primarily in older people (aged ≥ 45) who have diminished cardiovascular reserve and usually develops over several days due to exposure or confinement in a hot environment.[1,151–158] Classic heatstroke is minimally associated with exercise, and dehydration and obtundation are gradual.[159] Patients present with high body core temperature; hot, dry skin; and coma. This type of heatstroke has a high mortality because victims often have chronic illness and tend to present at an already advanced stage. It has been emphasized in medical textbooks because it is the predominant form seen at academic medical centers.[160–163] Classic heatstroke is frequently associated with anhydrosis (absence of sweating), but this is not a common finding in exertional heatstroke.[126,164,165]

Exertional Heatstroke

Exertional heatstroke (the most common form among military personnel) is characterized by early, severe, nonfocal encephalopathy with hyperthermia. There has been no consistent definition of this syndrome. Most case series[39,43,53,126] have described patients with exertional heatstroke who were first evaluated more than an hour after onset of obtundation or coma. This delay in medical care, largely due to medical inaccessibility at training sites, resulted in advanced life-threatening disease on presentation to the medical team. Such patients usually exhibited progressive multisystem disease with severe morbidity and high mortality, and often suffered cardiovascular collapse, rhabdomyolysis, acute renal failure, DIC, and seizures. Metabolic complications often included hyperosmolar states, hyperkalemia, hyperuricemia, hyperphosphatemia, lactic acidosis, and hypocalcemia. Mortality was high and liver necrosis, myocardial infarction, acute respiratory distress syndrome, and diffuse brain damage were often seen in survivors.

The importance of rapid access to emergency medical care is being increasingly recognized in military training. The usual clinical problem is to identify exertional heatstroke prior to onset of nonneurological complications. In this situation a reasonable definition of exertional heatstroke is EHI that causes early delirium, obtundation, or coma (see Exhibit 7-3).[2,3,151] Such cases usually occur without anhydrosis (a common characteristic in classic heatstroke) and often occur with relatively low rectal temperatures (< 106°F [< 41°C]). Many cases of exertional heatstroke with severe encephalopathy but modest hyperthermia have been reported. We do not consider hyperthermia, anhydrosis, or other complications essential to the diagnosis of exertional heatstroke.[1,4,5,50,166,167] Water and electrolyte depletion, acute renal failure, and muscle necrosis (rhabdomyolysis) are common components of heatstroke. Casualties must be closely monitored and aggressively treated to minimize morbidity and mortality.

When cooling and hydration are instituted early on the basis of the rectal temperature measurement, the encephalopathy of exertional heatstroke generally peaks within 10 to 30 minutes. Among Marine Corps recruits treated early with ice water and intravenous hydration, recovery of orientation and cooperative behavior generally occurs within 20 to 40 minutes from arrival at the emergency department, and correlates with rectal temperature lower than 102°F (39°C). Most patients can give a coherent clinical history (limited by amnesia) within 40 to 60 minutes.

Early treatment of exertional heatstroke also appears to prevent the development of life-threatening acute renal failure and cell lysis characteristic of severe rhabdomyolysis and hepatic necrosis. Among more than 200 cases of exertional heatstroke at Parris Island, from 1979 to 1994, none exhibited life-threatening complications of rhabdomyolysis involving hyperkalemia, hyperuricemia, hyperphosphatemia, altered calcium levels, lactic acidosis, progressive renal failure requiring dialysis, or DIC.[58] Minor complications in the early stages of muscle or liver injury responded quickly to cooling and hydration. The only life-threatening complications noted in this series were episodes of early cardiovascular collapse or sudden cardiac arrest.[138,140] Sudden cardiovascular collapse within the first hour was the syndrome that preceded all deaths. Exertional heatstroke was not observed in women recruits, although 18 hospitalizations for EHI were predicted from hospitalization rates for men.[58]

Exertional Heat Injury

Exertional heat injury is a progressive multisystem disorder, with hyperthermia accompanied by organ damage or severe dysfunction (usually metabolic acidosis, acute renal failure, muscle necrosis, or liver

necrosis).[49,163] We classify it as involving more-severe manifestations than exertional heat exhaustion, with less-severe complications than those of heatstroke, severe rhabdomyolysis, or acute renal failure. Casualties with exertional heat injury often have mild neurological symptoms, but periods of combativeness, delirium, obtundation, or coma define exertional heatstroke rather than heat injury.[8] Organ damage or dysfunction is frequently not manifested in early heat injury, so during the first hours of illness it may not be possible to distinguish exertional heat injury from heat exhaustion. Therefore, it is essential that all casualties with EHI be thoroughly evaluated for organ damage or severe dysfunction before release from the medical treatment facility, with reevaluation often necessary on the following day.

Exertional Heat Exhaustion

Exertional heat exhaustion is a reversible, non–life-threatening multisystem disorder reflecting the inability of the circulatory system to meet the demands of thermoregulatory, muscular, cutaneous, and visceral blood flow. It represents primarily a syndrome of dehydration without serious metabolic complications.[1,3,163] Heat exhaustion is typically thought to produce minor elevations of core temperature (< 104°F [< 40°C]), but it can be associated with the full range of rectal temperatures, from subnormal to very high. We define heat exhaustion as reversible system dysfunction, without evidence of organ damage or severe metabolic consequences.

The symptoms of heat exhaustion can be quite varied (see the mild and nonspecific symptoms listed in Exhibit 7-1) and are rapidly improved by water and salt replacement, a cool environment, and rest.[50,163] Rapid cooling to a body temperature of less than 102°F (39°C), rest, and rehydration are essential to prevent progression to more-serious levels of EHI.[2,11] Because heat exhaustion represents system dysfunction, without organ or tissue damage, rapid improvement is the rule.

Minor Heat-Related Syndromes

In addition to the disorders discussed above, which represent the primary spectrum of EHI, there are minor syndromes also associated with heat exposure. We do not recommend that the following be included as a part of an EHI reporting system, however, as they represent either very minor or unrelated syndromes.

Heat Rash

Heat rash (also known as prickly heat or miliaria rubra) is a pruritic red papular rash, located particularly in areas of restrictive clothing and heavy sweating.[49,168,169] It is caused by inflammation of the sweat glands. It may interfere with sweating and can therefore be a risk factor for more-serious heat illness. Sleeplessness due to itching and secondary infection may further aggravate thermoregulation. Treatment consists of cooling and drying the affected skin, controlling infection, and managing pruritus; the rash usually resolves over 7 to 10 days. Rare, severe cases with generalized and prolonged rash (miliaria profunda) may require evacuation to a cooler environment for several weeks to restore normal sweat gland function.

Heat Edema

Heat edema is dependent edema of the legs ("deck legs"), which may occur during acclimatization to a hot environment; it resolves spontaneously and is of no clinical significance.[21,49] Edema is due to expansion of blood volume during acclimation and does not indicate excessive water intake or cardiac, renal, or hepatic disease.[2] Management consists of loosening the clothing and elevating the legs. It is unwise to offer diuretics, as they may interfere with acclimatization and induce potassium loss.[50]

Parade Syncope

Parade syncope is fainting during prolonged standing due to inadequate venous blood return to the heart and brain.[49] It is more common in a hot environment but is unrelated to exercise. Syncope occurring during or after work in the heat should raise the suspicion of heat exhaustion or heat injury. Management of parade syncope is to restore normal blood circulation and minimize peripheral pooling through a brief period of recumbency in a cool place. Allowing individuals to move about will help prevent parade syncope, and provision of chairs and railings will decrease the risk of injury.

Exertion-Associated Collapse

Collapse *following* strenuous exercise may sometimes have a physiological etiology. During exercise, repetitive muscle pumping action assists the return of venous blood to the heart. On cessation of exercise, this muscle assistance to blood flow

ceases, and venous blood return may diminish rapidly enough to produce nausea, collapse, or syncope. Prolongation of mild exercise to allow time for adequate vasoconstriction is the rationale for a "cool-down lap" at the end of a strenuous run. Although this form of syncope may occur in the absence of EHI, clinical evaluation is warranted because these symptoms may be due to hypovolemia and other features of EHI. In contrast, collapse *during* exercise must be intensively investigated because of the greater potential for significant EHI or heart disease.

Sunburn

Sunburn reduces the thermoregulatory capacity of the skin and should be prevented through adequate sun protection. It should be managed like any other burn, and heat stress should be avoided until the burn has healed.[49]

CLINICAL MANAGEMENT OF EXERTIONAL HEAT ILLNESS

The severity of illness is often not apparent on initial presentation of EHI in the field. Severe EHI is a catastrophic event and should be treated as a medical emergency. Since milder forms of EHI may progress rapidly to more-serious illness, immediate and thorough evaluation is necessary to assess severity. Rectal temperature should be obtained immediately and rapid cooling should be initiated in the field.[170,171] Mental status assessment must always accompany measurement of rectal temperature and vital signs. Rehydration should always accompany cooling.[172] Delay in cooling and rehydration probably represents the single most important factor leading to death or residual, serious disability in those who survive.[50] In addition, there must be close monitoring of vital signs, aggressive replacement of fluid and electrolytes, and appropriate laboratory workup.[55,148,173]

In monitoring core temperature, it is important to avoid relying on measurements made at superficial sites such as the mouth, axilla, tympanum, or ear canal. Oral temperature is lowered by mouth breathing, especially in the presence of hyperventilation, a frequent response to hyperthermia. Tympanic and ear canal temperatures must be measured under carefully controlled conditions to avoid their being biased by the environment. It is not feasible to control those conditions adequately in most clinical settings, and in collapsed hyperthermic athletes, ear temperature readings have been 6 to 10 Fahrenheit degrees below rectal temperatures.[174]

Diagnostic Evaluation

Close monitoring of vital signs and serum chemistries is essential since during the first few hours, clinical symptoms may not reflect profound underlying metabolic abnormalities.[55,151,173,175] The initial laboratory evaluation for casualties, at different levels of clinical concern (mild-to-moderate and severe illness), is shown in Exhibit 7-5. The panel for mild or moderate illness screens for infection, anemia, and thrombocytopenia, and assesses dehydration. Evaluations of serum electrolytes screen for hypernatremia, hyperkalemia, and metabolic acidosis, although occasionally hyponatremia or hypokalemia may be found. The evaluation for glucose level detects exertional hypoglycemia and diabetic hyperglycemia, both of which are treatable causes of encephalopathy. The BUN and creatinine are tests for renal insufficiency, dehydration, or renal failure. Urinalysis should be performed, particularly to identify pigmented casts and a positive heme test out of proportion to red cells in the sediment (findings that are typical of rhabdomyolysis with myoglobinuria and renal tubular injury). Often the patient is unable to urinate until considerable hydration has occurred, so the urine is relatively dilute and reflects the posttreatment, rather than the pretreatment, status of the patient.

A serum enzyme panel is used to identify rhabdomyolysis and liver damage. These assays may be insensitive in early illness because of delayed release of the enzymes into the circulation. Maximum serum muscle or liver enzyme levels often occur 24 to 48 hours after the onset of illness, necessitating follow-up laboratory assessment the next day for all but the mildest cases.[50,62,154,176]

Selected cases of intermediate severity may require additional screening for the early signs of significant rhabdomyolysis and acute renal failure (see Exhibit 7-5). These tests are performed to distinguish between renal insufficiency due to poor perfusion of the kidney and that due to acute renal failure. Elevations of creatinine up to 1.7 mg/dL are often seen initially in rapidly reversible EHI, probably due more to reduced glomerular filtration rates and muscle-cell release of creatine than to tubular dysfunction. It is important to determine whether the creatinine level is improving after a few hours of

EXHIBIT 7-5

RECOMMENDED LABORATORY EVALUATION FOR EXERTIONAL HEAT ILLNESS

- **Mild-to-Moderate Illness**

  Complete Blood Count: Hemoglobin, hematocrit, white blood count and differential, platelet count

  Urinalysis: Specific gravity, pH, dipstick, and microscopic examination of sediment

  Serum Chemistries (Heat Panel): Sodium (Na), potassium (K), chloride (Cl), bicarbonate (HCO_3), glucose, blood urea nitrogen (BUN), creatinine (Cr), osmolality, creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), uric acid, myoglobin

- **Severe Illness, Add:**

  Examine Peripheral Blood Smear

  Serum Chemistries: Calcium, phosphorus, albumin, lactic acid, myoglobin, cardiac enzymes

  Arterial Blood Gases

  Coagulation Studies: Prothrombin time (PT), partial thromboplastin time (PTT), fibrin degradation products or D-dimer, fibrinogen level

  Consider Tests for Renal Failure (**Exhibit Table 1**)

EXHIBIT TABLE 1

LABORATORY EXAMINATION IN OLIGURIC ACUTE RENAL FAILURE

Diagnosis	U/P Cr	U_{Na}	FE_{Na} (%)	U Osmolality
Prerenal Azotemia	> 40	< 20	< 1	> 500
Oliguric ATN	< 20	> 40	> 1	< 350

U: urine; P: plasma; FE_{Na}: fractional excretion of sodium; ATN: acute tubular necrosis
Table: Reprinted with permission from Coyne DW. Acute renal failure. In: Carey CF, Lee HH, Woeltje KF, eds. *The Washington Manual of Medical Therapeutics*. Philadelphia, Pa: Lippincott-Raven; 1998: 230.

hydration. If not, then a casualty may require hospitalization or prolonged observation in the emergency treatment facility. It is also important to ensure that initial oliguria has been corrected.

If some of these screening tests are positive, casualties with severe illness may require further screening for early phases of the cell lysis syndrome, during which symptoms may be mild. Cell lysis can result in hyperkalemia, hyperuricemia, hyperphosphatemia, lactic acidosis, and hypocalcemia. Arterial blood gases should be obtained, with particular attention to metabolic acidosis. If the bicarbonate level is low, serum lactic acid levels will help determine the cause.[50,177] An evaluation of cardiac enzymes should be included to identify myocardial injury. Serum chemistries should be followed serially, with a determination of serum albumin to provide correct interpretation of calcium. Screening for DIC should include examination of the peripheral blood smear to identify fragmented erythrocytes and to confirm true thrombocytopenia rather than artifactual platelet clumping. If available, the D-dimer test is more specific and preferable to the assay for fibrin degradation products.

Stratification and Immediate Care

Immediate physical assessment must include frequent monitoring of mental status and vital signs, including rectal temperature and orthostatic changes in pulse and blood pressure (which are seen when a patient is subjected to tilt tests [see Exhibit 7-1]). For casualties with marked hyperthermia it is convenient to use a rectal probe, which provides continuous visualization of rectal temperature. Frequent assessments of mental status and muscle

symptoms are essential. Laboratory and clinical findings need to be integrated and level of care for diagnosis and management determined. There are no obvious, well-defined boundaries between the disease categories of life-threatening EHI (heatstroke, severe rhabdomyolysis, acute renal failure, shock), heat injury, and heat exhaustion. Each clinician chooses definitions that suit triaging of patients within his or her clinical system (ie, selecting patients for intensive or routine hospital care, extended emergency or clinic care, routine clinic care with follow-up, or clinic care without follow-up). Thus, each syndrome categorization along this continuum of EHI is somewhat arbitrary and has implications for treatment and prognosis in a specific healthcare setting.

A practical approach to the management of EHI is to examine each of the major areas of clinical involvement on a severity scale that reflects the usual management for each component problem. It then becomes easier to develop a sensible plan for management of the entire set of clinical problems. This is illustrated in Table 7-1 for management of a military recruit being seen at the nearest emergency care facility within 5 to 20 minutes after onset of EHI. These casualties are medically screened young adults without known chronic illness, taking few or no medications, and who were able to participate in conditioning exercise just prior to the onset of illness. The specific areas of physiological dysfunction considered are

- dehydration and electrolyte disturbances;
- nonfocal encephalopathy and other neurological signs;
- evidence of renal insufficiency;
- evidence of rhabdomyolysis (cell lysis); and
- other manifestations, including hyperthermia, muscle symptoms, hypoglycemia, and coagulopathy.

Categories 1 and 2 generally indicate clinical findings consistent with exertional heat exhaustion. A laboratory panel will be obtained for some casualties with mild illness (see Exhibit 7-5). As a rule, patients are treated with rest and rehydration and are removed from exercise overnight and perhaps throughout the following day. Laboratory evaluation is optional for Category 1. Category 2 implies that the laboratory panel for mild illness will be obtained, but follow-up is optional. Exertional heat cramps are usually treated by infusion of normal or half-normal saline (1–2 L). Oral salt solutions (0.5%) are also effective if the parenteral route is unavailable. Patients in Category 2 will often receive parenteral hydration for signs of fluid deficit or muscle cramps and will be kept at rest throughout the next day.

Patients in Category 3 and some in Category 4 would generally be considered to have exertional heat injury. The standard of care for patients in Category 3 includes the appropriate laboratory panel, rapid cooling with ice water for those whose rectal temperature is above 102°F (39°C), parenteral rehydration, and clinical and laboratory follow-up at 12 to 24 hours. The recruit will often be kept at rest for 2 to 3 days.

Patients in Categories 4 and 5 have severe EHI, and the standard of care includes a more extensive laboratory panel and dictates more vigorous intervention. Patients in Category 4 receive (*a*) rapid cooling with ice water for rectal temperature above 102°F (39°C), (*b*) rapid parenteral hydration with 2 to 3 liters of isotonic fluid, and (*c*) reevaluation of laboratory studies before discharge from emergency care (see Exhibit 7-5). At Category 4, some patients may be hospitalized, and all will be followed up at 12 to 24 hours. Indications for hospitalization are found in Exhibit 7-6. At Category 5, hospitalization is the standard of care. Most of these patients are managed in an intensive care unit. With aggressive management, most will not develop progressive rhabdomyolysis and are discharged after 2 or 3 days of hospitalization.

A frequent clinical problem is a patient with EHI who appears normal after routine management except for elevated serum CK or evidence of myoglobinuria. In this circumstance, the medical officer should maintain alkaline urine, then rule out the cell lysis syndrome and acute renal failure by laboratory evaluation (see Exhibit 7-5). If these tests are normal, the patient can be followed as an outpatient with continued hydration, observation, monitoring of urinary output, and daily laboratory follow-up until CK is returning to normal. The same applies to a patient with localized muscle breakdown due to overuse, which produces elevated CK with or without myoglobinuria and without other systemic symptoms or signs.

Usually, patients who are severely ill will declare themselves within the first few hours. Typical severe syndromes are (1) heat injury involving several systems, (2) exertional heatstroke with severe CNS dysfunction (often accompanied by rhabdomyolysis), and (3) severe rhabdomyolysis (occasionally without hyperthermia or encephalopathy). However, acute renal failure is often not apparent during the first hours of illness. Occasionally, the onset of severe rhabdomyolysis has been obscured, particularly in individuals who are highly motivated to conceal

TABLE 7-1

CLASSIFICATION, STRATIFICATION, AND CLINICAL MANAGEMENT OF EXERTIONAL HEAT ILLNESS

Category	Dehydration	Encephalopathy*	Renal Function† (mg/dL)	Cell Lysis† (U/L)	Other	Clinical Management
1	Nonspecific symptoms	Slow, drowsy, clears rapidly	Cr: 2 1.4 No oliguria	CK: < 700	Nonspecific symptoms Max temp < 100°F	Rest and rehydration. Follow-up is not usually needed.
2	Collapse or orthostatic Sx in field, normal v.s. in ER	Confused/cooperative, clears rapidly	Cr: 1.5–1.7 No oliguria	CK: 700–1,200	Cramps, myalgia resolve in ER Max temp 3 100°F	Rest, cooling, and IV rehydration. Laboratory evaluation as indicated. Clinic follow-up, laboratory follow-up, or both may be indicated.
3	Exertional syncope or positive tilt test, or abnormal electrolytes, corrected in ER	Confused/uncooperative, clears rapidly, amnesia	Cr: 1.8–1.9 High urinary SG decreases Oliguria responds rapidly	CK: 1,200–4,000 ALT/AST: 50–150 Mild rhabdomyolysis	Minor muscle Sx persist Max temp 3 104°F	Rest, rapid cooling, IV rehydration, and laboratory evaluation. Clinic and laboratory follow-up are the usual standard of care.
4	Patient requires > 3 L IV fluid, or hypotension, or electrolyte abnormality persists	Delirium, obtundation, clears rapidly, amnesia	Cr: 3 2.0 Oliguria responds slowly; mild urine abnormalities	CK: 4,000–10,000 ALT/AST: > 150 Metabolic acidosis Negative labs for cell lysis syndrome	Muscle Sx persist Max temp 3 106°F	Rest, rapid cooling, IV rehydration, and extensive laboratory evaluation. Hospitalization may be indicated.
5	Mean BP < 70, systolic < 90, tachycardia persists Hypokalemia Hyponatremia	Coma, or CNS signs clear slowly, seizure	Cr: 3 2.0 Oliguria persists Other signs of renal failure	CK: 3 10,000 Signs of cell lysis syndrome Severe metabolic acidosis	Major muscle Sx persist Hyperthermia > 40 min Hypoglycemia Thrombocytopenia Coagulopathy	Rest, rapid cooling, IV rehydration, and extensive laboratory evaluation. Close observation and life support/intensive care may be indicated. Hospitalization is the usual standard of care.

ALT: alanine aminotransferase, AST: aspartate aminotransferase, BP: blood pressure, CK: creatine kinase, Cr: creatinine, CNS: central nervous system, ER: emergency department or emergency care facility, IV: intravenous, labs: laboratory evaluations, SG: specific gravity, Sx: symptoms, U/L: units per liter, v.s.: vital signs

*See Exhibit 7-3 for explanation of terms describing encephalopathy.

†Early levels of CK and Cr have limited meaning because these values are based on maximums seen at 12–36 h. Continued muscle symptoms, other laboratory evidence of the cell lysis syndrome, and renal failure are critical determinants. The direction of change in sequential studies is essential to the early recognition of serious rhabdomyolysis. Rarely, the ALT and AST are elevated beyond the muscle enzymes (CK), implying liver necrosis as the dominant source of the cell lysis syndrome.

EXHIBIT 7-6

INDICATIONS FOR HOSPITALIZATION IN EXERTIONAL HEAT ILLNESS

- Difficulty correcting hypotension
- Persistent electrolyte abnormalities
- Seizure
- Severe encephalopathy
- Moderate encephalopathy not clearing rapidly
- Persistent creatinine above 2 mg/dL
- Persistent oliguria
- Laboratory evidence of acute renal failure (see Exhibit 7-5) or myoglobinuria
- Laboratory evidence of the cell lysis syndrome
- Persistent muscle symptoms suggesting evolving rhabdomyolysis
- Marked hyperthermia (rectal temperature 3 105°F [3 41°C]) not improving (or with slow response to aggressive cooling)
- Evidence of DIC
- Exertional hypoglycemia
- Persistent substantial diarrhea
- Significant gastrointestinal bleeding
- Hypokalemia

muscle pain, and rarely, the onset of hyperthermia is delayed in patients with exertional heatstroke.

Principles of Clinical Management

Exertional heat illness requires urgent diagnosis and treatment. Although Category 4 and Category 5 syndromes, which include heatstroke, severe muscle or liver injury, and acute renal failure, are clearly medical emergencies, patients with EHI at milder levels of severity require urgent and aggressive management to avoid progression. Rectal temperature should be obtained immediately, and if it is above 102°F (39°C), rapid cooling should be initiated in the field. Such patients should be kept at rest in a cool environment, and vital signs and mental status must be closely monitored. Rapid cooling of hyperthermic patients should proceed until the body core temperature remains below 102°F (39°C).[178,179] The degree of organ damage in EHI with hyperthermia appears to be directly related to the magnitude and duration of elevated core temperature.[53,180,181] Patients who require cooling will also require rehydration at the same time to restore adequate circulation for effective thermoregulation.[172] Frequent serial evaluation of vital signs, mental status, and clinical assessment should start in the field and continue throughout initial emergency care. After cooling and correction of fluid and electrolyte problems, it then becomes possible to assess for acute renal failure, rhabdomyolysis, hypoglycemia, and other complications. The initial clinical assessment will also permit identification and management of coexistent problems such as infection.

In controlled settings, emergency medical care for EHI should be arranged in advance. Management of EHI is always urgent, to avoid the potential for rapid deterioration. Patients need immediate access to medical support in the field, to include, at a minimum, measurement of rectal temperature, brief clinical assessment of vital signs and mental status, and effective cooling.[149,182] The field team should also be prepared to provide basic life support (BLS) and cardiopulmonary resuscitation (CPR), advanced cardiac life support (ACLS, including early automated defibrillation), and first aid for injuries. If transportation to an emergency department will require more than 5 to 10 minutes, provision should be made for administering intravenous fluids en route. The field medical team needs to provide an accurate clinical description of the immediate events, symptoms, signs, vital signs, and mental status of the patient, along with training activities, environmental conditions, clothing, and treatment given prior to arrival at the medical facility. To avoid substantial delay in treatment in settings where EHI is common, strenuous physical training should not be conducted without on-site medical capability.[183] For example, a recruit training center should require that at least one medical corpsman or medic (with equipment, ice, and a transport vehicle) be on site while strenuous training is conducted. When the vehicle leaves the training site, strenuous exercise should be stopped until medical support and transport are again available.

There is a known risk for sudden fatal cardiac arrest with exercise, which has been an important component of unexpected exercise-related mortality of recruits.[142] Furthermore, we believe that risk for sudden fatal cardiovascular events increases dramatically during the hyperthermic phase of exertional heatstroke. Although not routine in the past, we believe that cardiac monitoring should be instituted early in the treatment of heatstroke, and that ACLS must be immediately available for patients who develop exercise-related shock or arrhythmia while hyperthermic with EHI.

In this treatise we provide guidelines for field, outpatient, and emergency care of military personnel with EHI, but we leave intensive care of these patients to other specialized texts. Patients with heatstroke potentially have encephalopathy; hyperthermia; acute renal failure; rhabdomyolysis; electrolyte and acid–base imbalances, including lactic acidosis; and may develop additional complications such as DIC. Such patients obviously require intensive care following their initial presentation, but important initial treatment is given in the field and the emergency care setting before arrival in the intensive care unit.[151]

Treatment of Hyperthermia

Most important in the treatment of EHI is the early initiation of cooling and rehydration, starting in the field with an immediate rectal temperature reading. First the patient needs to lie down. An obtunded or comatose patient should be placed on one side, with the airway closely monitored to avoid aspiration of vomitus. As much clothing should be removed as is practical, and those who are hyperthermic should be doused with cold water followed by ice water sheet massage or other effective means of cooling. If lengthy transport is required, immediate parenteral access and rapid initial infusion of normal or half-normal saline are recommended.

There is debate over the best method for body cooling.[162,184–190] The American College of Sports Medicine states that "cool or cold (ice) water immersion is the most effective means of cooling a collapsed hyperthermic runner."[191] Concerns have been expressed that ice water may induce peripheral vasoconstriction or shivering and reduce skin temperature without reducing core temperature. In actual practice in Marine Corps recruit training at Parris Island, cooling with ice water was found to reduce rectal temperature by about 0.15°C (0.25°F) per minute, with no mortality from heatstroke.[184] Studies on thousands of religious pilgrims who succumbed to heat illness during their Hajj in Saudi Arabia show that a cooling rate of 0.06°C (0.1°F) per minute is obtained when a device is used that sprays tepid water and cools with fans.[192] The mortality rate for heatstroke in this setting (which included a large number of older patients) was about 20%. Because coronary arteries with atherosclerosis often undergo spasm in response to ice water exposure, patients with atherosclerosis may be at less risk from tepid water cooling.[193,194] Therefore, we recommend that ice water cooling be used for patients younger than age 35 to 45 years, but that tepid water and fans be used instead (at least initially) to cool older patients.

Body cooling should be accomplished as quickly as possible, using the most practical means available.[166] Methods for cooling include removing outer layers of clothing, soaking the skin with water, using wet sheets or spray bottles, massaging the skin, and resoaking. Cold water applications may be alternated with massage to encourage local blood flow and heat dissipation.[184] If these techniques do not rapidly reduce the rectal temperature (best monitored with a rectal probe), the patient can then be partially immersed, using a stretcher over a tub filled with ice water. The stretcher is lowered so that only the patient's buttocks and lower back are in the ice water, leaving the anterior trunk and limbs accessible for clinical evaluation, parenteral lines, and other treatments. Alternatively, ice bags or a cooling blanket—especially if placed over major arteries—can provide cooling while the patient lies on a bed board. In this situation, CPR and ACLS can safely be implemented. The cooler the water the more rapid the cooling progresses. The room should be air conditioned to maintain a low humidity and air temperature. Fanning can be accomplished manually, by electric fans, or by placing the patient under rotating helicopter blades (a well-tested military field expedient). Rapid cooling of hyperthermic patients should continue until the rectal temperature remains below 102°F (39°C), after which cooling can proceed without cold water (such as a tepid shower) until the rectal temperature remains below 100°F (38°C). The patient must be physically supported and observed during this process, and monitoring of body temperature should be continued for at least 2 to 4 hours. Regardless of the method of cooling, it appears that the degree of organ damage is directly related to the magnitude and duration of elevated body temperature.[147,172,195]

It is important to avoid using antipyretics in the treatment of hyperthermia due to EHI. This is because antipyretics work by lowering the body temperature set point in the hypothalamus. As discussed above, this is not altered during heatstroke. Disadvantages of antipyretics include risk of severe liver injury with acetaminophen, reduced potassium excretion with nonsteroidal antiinflammatory drugs, depressed platelet function and risk of subsequent DIC with aspirin, and risk of gastric irritation or bleeding. Occasionally, cooling leads to inappropriate shivering while the body temperature is still elevated; this may increase the generation of body heat and aggravate rhabdomyolysis. Chlorpromazine should not be used because of a relatively high incidence

of hypotension in patients with EHI.[4,151] Lorazepam, which is also a sedative, is probably the safest drug for this purpose, in part because of its low risk for hepatotoxicity and its rapid metabolism.

Treatment of Fluid and Electrolyte Depletion

Oral rehydration is adequate for the treatment of mild heat exhaustion and mild heat cramps. Heat cramps respond to oral salt solutions (0.5% salt, or 1 teaspoon per quart of water); otherwise patients are usually given water. Patients with more severe EHI, including those with orthostatic or supine hypotension, history of collapse or syncope, severe heat cramps, hyperthermia, or clinical findings that place them in Category 3 or higher levels of EHI (see Table 7-1) are better treated with parenteral rehydration. Young military personnel with these problems are usually first given 1 to 2 liters of normal or half-normal saline, starting at a rate of 250 to 500 mL/h, and their response is carefully monitored. Normal saline is used for hypotensive patients and those whose evaluation suggests hyponatremia or severe deficits of both salt and water.[8,166] The need for more than 2 liters of fluid is an indication for laboratory monitoring both to assess the severity of illness and to choose the best infusion to correct electrolyte abnormalities—especially alterations in sodium, potassium, and bicarbonate levels.[50,179] Most often, it is necessary to replace some free water as well as sodium. However, patients who had been drinking large volumes of water might have become hyponatremic.[115] Fluid intake and output should be measured, with frequent monitoring of vital signs, especially for those patients with Category 4 or higher levels of EHI (see Table 7-1). When supine blood pressure is adequate, tilt tests should be used to identify patients whose blood pressure control is still abnormal.[99]

A practical approach to the urgent correction of hypernatremia, hyperkalemia, hyponatremia, hypokalemia, and acidosis has been published elsewhere[196] in more complete form. Potassium depletion is best treated orally, but patients with severe EHI may not be able to take oral medications because of obtundation or nausea and vomiting. Intravenous potassium replacement should be administered in half-normal or normal saline; dextrose (which tends to move potassium into cells) should be avoided. Rates of infusion are usually limited to 20 mmol/h unless paralysis or malignant ventricular arrhythmias are present, in which case higher rates are recommended. Close electrocardiographic (ECG) and neurological monitoring are required.

The earliest ECG sign of hyperkalemia is peaked T waves. Hyperkalemia should always be suspected when young exercising individuals collapse with arrhythmias. When hyperkalemia is found by chemical analysis, the medical officer should exclude pseudohyperkalemia, primarily due to needle hemolysis (with an elevated LDH level), fist clenching during blood drawing, marked leukocytosis or megakaryocytosis, or erroneous assay. An ECG should be performed while awaiting results of the repeat assay. The acute treatment of severe hyperkalemia is a medical emergency, the management of which has been well described.[196] Treatment of either hypokalemia or hyperkalemia requires careful monitoring of response because of shifts between intracellular and extracellular compartments and changes in rate of renal loss.

Severe metabolic acidosis is often present in severe dehydration. Arterial blood gas measurements are essential to establish arterial pH.[50,177] Aggressive replacement of fluids and conservative replacement of base deficits are required until blood volume and pH are adequate.[197] This requires intensive monitoring of cardiopulmonary status and urinary output (via Foley catheter), with adjustment of intravenous fluid therapy based on the clinical response.[55,72] Severely ill patients will require invasive monitoring and management in an intensive care unit.

Treatment of Impaired Renal Function

Early management of acute renal insufficiency in EHI should focus on correction of the prerenal component due to volume depletion and hypotension. If response to initial rapid isotonic fluid infusion confirms a substantial fluid deficit, then infusion of parenteral fluids should continue, guided by monitoring of urinary output and assessment of blood volume. Persistent or severe hypotension may require the use of dopamine hydrochloride, a pressor favoring renal perfusion. Diuretics are used only after correction of hypovolemia. Probably the most common error made in emergency care is to administer diuretics too early, while the volume deficit is still substantial. Rhabdomyolysis produces myoglobinemia and lactic acidosis, which increase the risk of continued renal tubular damage from myoglobin. Forced alkaline diuresis is believed to be protective. However, we cannot administer bicarbonate safely, or increase diuresis with diuretics, unless blood volume has already been restored and urinary output is adequate (urinary output should exceed 50 mL/h).

Allopurinol should be used to treat severe levels of hyperuricemia by decreasing production of uric acid. Treatment is usually not necessary unless uric acid exceeds 15 mg/dL. The detailed management of acute renal failure requires close monitoring of renal, cardiovascular, and pulmonary function; and monitoring and correction of a number of potential electrolyte and chemical problems. This is best accomplished in an intensive care setting, with institution of dialysis if renal failure persists. An additional feature of rhabdomyolysis is the cell lysis syndrome, which causes early hyperkalemia, hyperuricemia, hyperphosphatemia, hypocalcemia, and lactic acidosis due to release of these materials from injured tissues, particularly muscle and liver. Management of this syndrome is discussed in standard texts on intensive care.

Treatment of Neurological and Other Signs and Symptoms

The encephalopathy of EHI is treated by rapid cooling of patients with hyperthermia and by rehydration. It is important to be aware of the possibility of a reversible metabolic encephalopathy from other complications of EHI, such as hypoglycemia or severe water and electrolyte disturbances involving sodium, potassium, calcium, magnesium, and acid–base abnormalities. Unrelated metabolic problems that might mimic EHI include thiamine deficiency and certain endocrinopathies (thyroid storm and adrenal crisis). Mass lesions, meningitis, brain abscess, thrombosis, and hemorrhage usually produce other signs not seen in uncomplicated EHI and fail to improve with rehydration and reduction in body temperature. Most often the duration of delirium is not sufficient to require use of sedatives, which are not recommended for EHI. Sometimes the need to protect the airway during obtundation or the development of abnormal or ineffective breathing mandates intubation and administration of oxygen via endotracheal tube. This may be followed by maintenance on a ventilator until the patient's respiratory drive and airway improve.

If the patient has a seizure, then management will include assessment for reversible metabolic causes, including hyponatremia, hypoglycemia, magnesium deficiency, infection, mass lesions, and brain injury from thrombosis or bleeding. When the seizure is due to heatstroke, then rapid cooling and rehydration are important elements of treatment. In recent years, many cases of early exertion-related seizures among military trainees have been due to hyponatremia. Regulated, but rapid, correction of hyponatremia is then urgent. Treatment may include administering antiepileptic drugs, particularly short-acting benzodiazepines followed by loading doses of phenytoin, and maintaining protection for the airway.

Management of *exertional hypoglycemia* consists of infusion of 50% dextrose in water as indicated by the level of glucose and clinical signs, followed by infusion of 5% dextrose in water or saline over the next 24 hours, with frequent reassessment.

Gastrointestinal bleeding in EHI can be due to other gastrointestinal disease aggravated by exercise-induced diarrhea. It is important to exclude DIC, to replace significant blood loss, and to stabilize the patient for early endoscopy if the bleeding is extensive.

DIC typically becomes a problem between 18 and 36 hours after onset of exertional heatstroke.[42] The most important therapy for DIC is to correct the underlying problem causing the hypercoagulable state. In EHI this is often tissue necrosis, typically of muscle and liver, but later in the course it may be due to infection. If bleeding becomes the major problem from DIC, the medical officer can replace fibrinogen and clotting factors by infusion of cryoprecipitate and fresh frozen plasma, and use transfusion of leukofiltered platelets to sustain an adequate platelet count. If thrombosis is the main problem, then the judicious use of heparin can be considered.

Disposition of Casualties With Exertional Heat Illness

The duration of increased risk for recurrent episodes of EHI is unknown, although about 10% of cases among Marine Corps recruits at Parris Island (discussed below) had more than one episode of EHI during their 12 weeks of basic training. Recurrent risk has been discussed most often with exertional heatstroke. There are numerous anecdotal reports of recurrence of EHI even years later, raising the possibility of long-term host susceptibility. Intensive physiological studies on the effects of repeated exposures to exertional heat load suggest that there may be increased susceptibility for some months following exertional heatstroke.[198] Furthermore, there are individuals who have repeated high levels of CK or symptomatic mild rhabdomyolysis with heavy exercise, some of whom have had repeated severe episodes of rhabdomyolysis. However, the data required to properly address this issue would need to be obtained from a large, controlled, follow-up study of individuals who did and did not suffer severe EHI and were reexposed at multiple, subse-

quent times to heavy exercise in hot environments. While such data are potentially available from military populations, such a study has not yet been reported.

The conventional view is that the potential for increased susceptibility is substantial and that severe EHI is grounds for permanent medical discharge from the military. In our experience, a substantial percentage of Marine Corps recruits with EHI present at a medical treatment facility with high body temperature but only mild symptoms and signs. Laboratory screening for electrolyte disturbances, renal failure, and rhabdomyolysis show only mild problems. Early aggressive cooling and hydration results in rapid reversal of hyperthermia, normal urinary output, and improvement of symptoms. We believe that these patients have not suffered severe EHI and therefore should not be considered for the military career restrictions that are placed on those who have been hospitalized for heatstroke. Preliminary data on long-term follow-up suggest that subsequent hospitalization for EHI is rare.

Many patients appear to have mental (and perhaps physical) impairment for several days following even a mild episode of EHI, including poor judgment and difficulty concentrating.[43,51,199] We recommend that patients with mild EHI be provided rest in a cool area at least overnight. Those with moderate illness should be followed up with clinical evaluation (including careful examination for slow mentation and poor concentration) and repeat laboratory studies at 24 hours, then given perhaps another 24 to 96 hours before resuming strenuous activities. In our experience, patients who suffered mild EHI with marked hyperthermia during physical fitness training have occasionally exhibited 3 to 4 days of diminished mental function, affecting their performance of tasks that require concentration, calculations, and judgment. Those with severe EHI may require several weeks or months before resuming strenuous activities.

EPIDEMIOLOGY AND PREVENTION OF EXERTIONAL HEAT ILLNESS IN THE MILITARY

EHI has been a substantial problem in military operations and training; it occurs primarily with sustained exertion, especially in a warm, humid environment.[200,201] When adequate hydration and emergency medical management are unavailable, each case is a potential death.[1,55,83] EHI has been recognized since biblical times and is mentioned in accounts of several ancient military campaigns.[202,203] Recognition of its relationship with warm environmental temperatures was solidified by 1900. During World War I there were particularly high rates of EHI in British troops in Mesopotamia, with at least 600 deaths.[204] By 1917 heatstroke stations were authorized in the British military; ice was provided daily, abundant drinking water was made available, and, as far as possible, troops were relieved of duty between 10 AM and 4 PM.[205,206]

During World War II the lessons of World War I had to be relearned[207] (not unusual in the history of warfare). British operations in the Middle East had particularly high rates of heat illness.[208,209] The biggest problem for American troops during World War II was in stateside training. During the war there were about 250 reported deaths from heatstroke, mostly at training centers in the southern United States.[53,210–215] After the war, EHI associated with US military training in hot weather continued to result in significant morbidity.[216–218] During the 1967 Six-Day War between Egypt and Israel, intelligence reports indicated that the Egyptians suffered about 20,000 deaths with no visible wounds (apparently caused by dehydration or heatstroke), while Israeli casualties were minimal.[83]

Although many other reports of EHI morbidity in military training and operations have been published,[180,215,219–221] the largest group of military trainees who have been thoroughly studied is recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina (MCRD-PI). Marine Corps recruit training was formalized at Parris Island in 1915 and consisted of 2 months of drill, physical exercise, personal combat, and intensive marksmanship qualification. Since then, recruit training has continued, varying from 4 to 12 weeks in length depending on specific wartime needs. Currently, all female and about half of all male Marine Corps recruits undergo their 12-week basic training course at MCRD-PI. The area has a semitropical climate with high temperatures and humidity, especially during the summer months. Extensive studies on EHI have been conducted in this population since the 1950s,[7,38,90,130,189,222] including those that developed the wet bulb globe temperature (WBGT) index as a guide for regulating the amount of strenuous exercise during training.[217,223–229]

Recent studies at MCRD-PI[56–58,138,144–146,230] have characterized the time patterns for occurrence of EHI during basic training. From 1982 through 1991, the average rate of occurrence of EHI in recruits was 0.7% during their 12 weeks of basic training, with

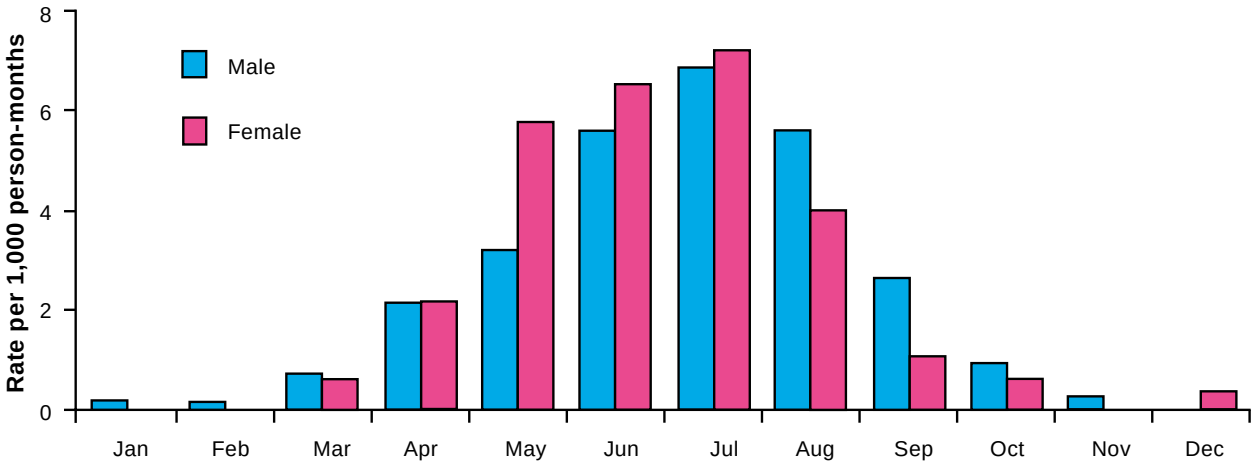

Fig. 7-3. Rates of exertional heat illness in Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by gender and month for the years 1982 through 1991. Rates are cases per 1,000 recruit person-months. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):357.

rates up to 2% during the summer months; 11% of men and none of the women were hospitalized.[58] Figure 7-3 shows the number of recruit cases per 1,000 person-months by month of year, and Figure 7-4 shows the distribution of recruit cases by hour of day. These illustrations show that case rates were highest during the summer months, and that most of the cases occurred during the 7-to-9 AM exercise period. When these early morning cases were categorized by WBGT at the hour of occurrence, we found that the case rates increased dramatically as the WBGT rose above 65°F (18°C) (Figure 7-5). When the same cases were categorized by the maximum WBGT of the previous day, we found the same pat-

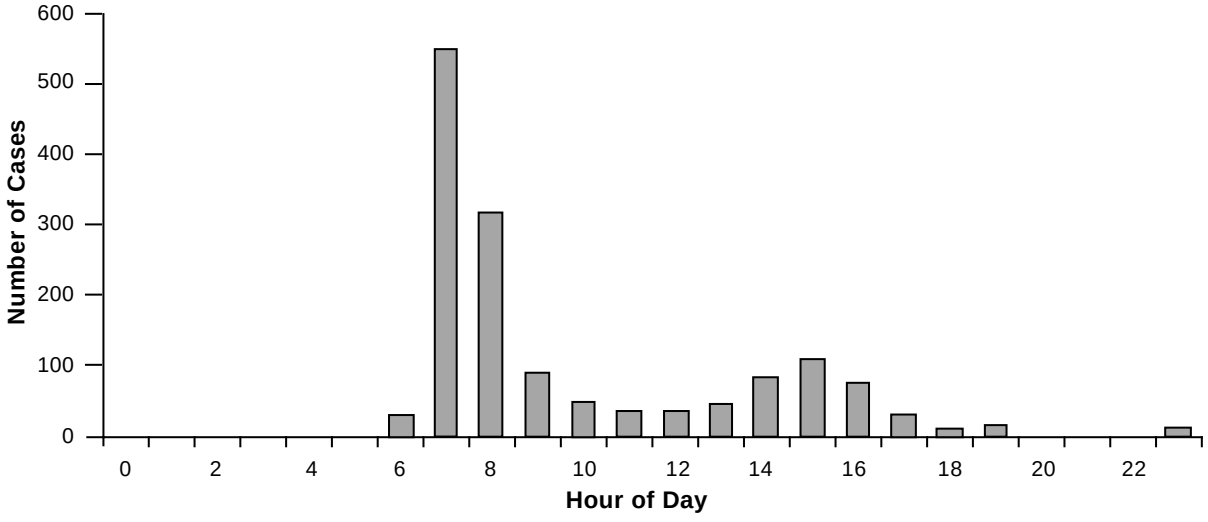

Fig. 7-4. Numbers of cases of exertional heat illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by hour of the day of illness for the years 1982 through 1991. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):357.

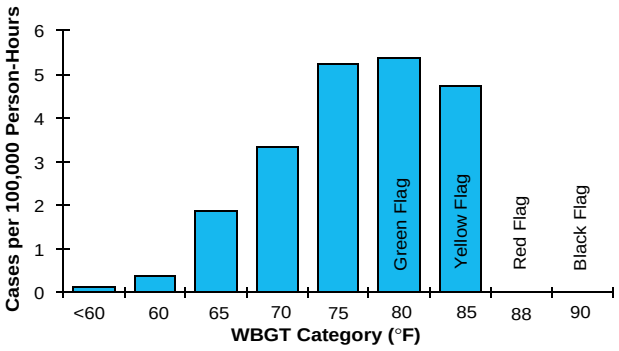

Fig. 7-5. Rates of exertional heat illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by wet bulb globe temperature (WBGT) between 0700 and 0900 hours for the years 1982 through 1991. There were very few person-hours of exposure at WBGT of 88°F or above. Rates are cases per 100,000 recruit person-hours of physical activity. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):358.

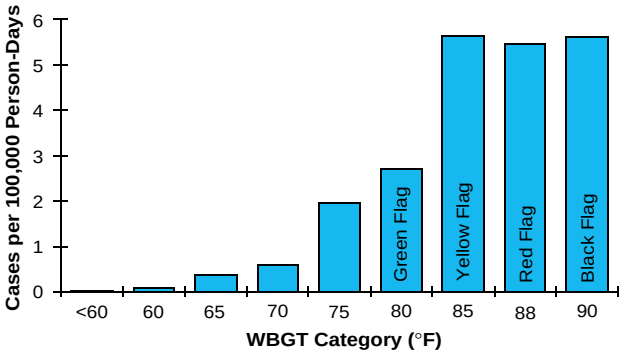

Fig. 7-6. Rates of exertional heat illness by maximum wet bulb globe temperature (WBGT) category on the day *prior* to the day of illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina. The cases are only those that occurred between 0700 and 0900 hours, for the years 1982 through 1991. Rates are cases per 100,000 recruit person-days. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):358.

tern, with case rates rising rapidly as the prior day's maximum WBGT rose above 65°F (18°C) (Figure 7-6). The mechanism for this carryover risk may involve persistent dehydration, fatigue, or undescribed metabolic factors, in addition to the influence of the prior day's heat on the early morning temperature. When the prior day's peak temperatures were analyzed in strata that held the early morning temperatures constant, a 4-fold gradient in EHI susceptibility persisted. These figures illustrate the effect of weather conditions on EHI rates and suggest that the effects of heat exposure may also carry over to the next day.[58]

Risk Factors for Exertional Heat Illness

Numerous risk factors have been identified for EHI, which relate primarily to (*a*) weather, discussed above, (*b*) acclimatization, (*c*) physical fitness, and (*d*) training circumstances.[200,201,215,222,226,230–235]

Acclimatization

Acclimatization reduces the risk for EHI by increasing the sweat rate, decreasing the sodium content of sweat, and initiating sweating at a lower body core temperature.[32,40,236] In addition, acclimatization increases cardiovascular capacity and blood volume.[31] Acclimatization increases the efficiency of heat dissipation, but its benefits can be decreased or nullified by sleep loss, infection, dehydration, salt depletion, or the use of drugs or alcohol.[21,237] A sensible acclimatization program is important during the first 2 to 4 weeks in a warm environment. This should include daily exercise in the heat of gradually increasing intensity and duration (< 2 h), accompanied by copious quantities of water and additional salt in the diet (in the form of high-salt foods, not salt tablets).[20,238–240]

Physical Fitness and Body Type

Poor physical fitness is an important risk factor for EHI.[73,235,240–243] For example, at MCRD-PI we documented at least a 3-fold higher risk for EHI during basic training in recruits with poor fitness (for men, 1.5-mile run-time ≥ 12 min), compared with those who were more fit (1.5-mile run-time < 10 min).[230] This experience is consistent with the above discussion of cardiovascular stresses in EHI. In addition, body type (depicted through the body mass index [BMI], calculated as weight/height^2) appears to relate to risk for EHI.[233,244–246] At MCRD-PI there was about a 3-fold higher risk for EHI during basic training in men recruits who were heavyset (BMI ≥ 26 kg/m^2), compared with those who were thin (BMI < 22 kg/m^2).[230] This relationship with BMI does not appear to hold for women recruits. The combined risk for EHI in men who were heavyset and had slow run-times was about 8-fold higher than in those who were thin and had fast run-times (Table 7-2). The

TABLE 7-2

ODDS RATIOS (95% CONFIDENCE INTERVALS) FOR DEVELOPING EXERTIONAL HEAT ILLNESS DURING MARINE CORPS BASIC TRAINING,* 1988–1992

Body Mass Index	1.5-Mile Run-Time (min)		
(kg/m^2)	< 10	10 – < 12	3 12
< 22	1.0 (referent)	1.5 (0.7–3.2)	3.5 (1.4–8.8)
22– < 26	1.6 (0.6–3.8)	2.0 (0.9–4.2)	8.5 (3.8–19)
3 26	3.7 (0.9–15)	3.3 (1.5–7.1)	8.8 (4.1–19)

Low risk (OR 2 2): BMI < 26 kg/m^2 and 1.5-mile run-time < 12 min
Medium risk (2 < OR < 4): BMI 3 26 kg/m^2 and 1.5-mile run-time < 12 min, or BMI < 22 kg/m^2 and 1.5-mile run-time 3 12 min
High risk (OR 3 4): BMI 3 22 kg/m^2 and 1.5-mile run-time 3 12 min
* Male recruits, Marine Corps Recruit Depot, Parris Island, South Carolina
BMI: body mass index (weight/height^2)
OR: odds ratios
Adapted with permission from Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc.* 1996;28:941.

18% of men recruits with these high-risk characteristics (BMI ≥ 22 kg/m^2 and 1.5-mile run-time ≥ 12 min) accounted for 47% of the cases of EHI that occurred during basic training at MCRD-PI in 1988 through 1992 (Figure 7-7).[230]

Circumstances of Training

Training circumstances provide critical risk factors for EHI.[247] The early recruit studies discussed above established that most heat illness cases at Parris Island occurred with strenuous exercise when the WBGT was 80°F (27°C) or higher.[58,225] In the 1950s, more than half of heat illness cases occurred during daytime infantry drill and training marches.[227] Subsequent studies demonstrated that as the WBGT rose above 80°F (27°C), heat illness rates were reduced by progressively limiting the level of exercise, increasing rest periods, increasing hydration, and reducing heat-retaining clothing.[223] These modifications were implemented in a standard manner for four categories of high WBGT (green flag = 82°F–84.9°F, yellow flag = 85°F–87.9°F, red flag = 88°F–89.9°F, and black flag = 90.0°F or higher). As a rough guide, exercise continued with caution during green flag, strenuous exercise (eg, "marching at standard cadence") was suspended for first-phase recruits during yellow flag, strenuous exercise was curtailed for all recruits during red flag, and all outdoor physical training was suspended during black flag conditions.[20,248,249] An important consequence of these regulations has been the scheduling of strenuous exercise in the cooler, early morning hours (as seen in Figure 7-4).

The flag conditions regulating authorized training activities by WBGT were designed for marching, rather than running, yet the focus of physical conditioning in the past 2 decades has been more on middle-distance running (1–3 miles) than marching.[49] Marching with a heavy load produces about 500 W of heat, while running at 8 to 10 mph produces 1,000 to 1,200 W.[16,149,223] This focus on higher-metabolic-rate training activities leads us to expect EHI casualties at lower WBGT levels than generally occur with marching. This has been demonstrated in studies showing increased risk for EHI at WBGT levels as low as 65°F (18°C) (see Figure 7-5).[58]

Prevention of Exertional Heat Illness

Provision of adequate rest periods during training is particularly important in preventing EHI, with

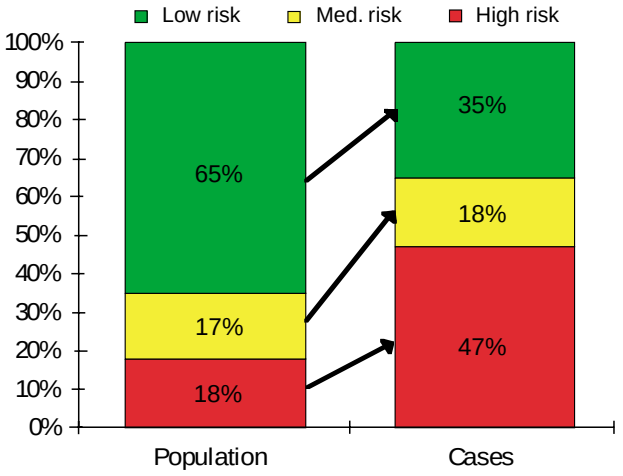

Fig. 7-7. Frequency distribution of cases and population by risk status among male Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, for the years 1988 through 1992. High risk: body mass index (BMI) ≥ 22 kg/m^2 and 1.5-mile run-time ≥ 12 min; medium risk: BMI ≥ 26 kg/m^2 and 1.5-mile run-time < 12 min, or BMI < 22 kg/m^2 and 1.5-mile run-time ≥ 12 min; low risk: BMI < 26 kg/m^2 and 1.5-mile run-time < 12 min.
Adapted with permission from Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc.* 1996;28(8):942.

restrictions on work activities determined by physical fitness, acclimatization, medical status, and weather conditions.[225,250,251] Strenuous activities (particularly running) should be scheduled for the coolest part of the day.[249] Work/rest cycles have been developed by USARIEM for routine military activities, and are reflected in updated regulations regarding flag conditions.[49,122,252] Exhibit 7-7 provides the current recommendations. This chart does not address exercise that exceeds hard work (more than 600 W), such as running at an 8- to 10-mph pace (1,000-1,200 W). Burr[49(Appendix D)] has provided estimates of the maximum minutes of running at a 6-mph pace, which will produce 5% EHI casualties at different levels of WBGT. For example, he estimates that at a WBGT of 90°F, 22 minutes of running will produce 5% casualties; at 82°F, 34 minutes; and at levels lower than 60°F, 50 to 60 minutes. Most trainers would consider a 5% casualty rate much too high, and Exhibit 7-7 provides guidelines (for work at < 600 W), which should be safe for at least 4 hours, given the stated assumptions. These guidelines were designed to safely maintain continuous operations for at least 4 hours, not for an intense activity that leaves one exhausted at the end and requires an extended period of rest and recuperation in com-

EXHIBIT 7-7

FLUID REPLACEMENT GUIDELINES* FOR HOT WEATHER TRAINING (AVERAGE ACCLIMATIZED SOLDIER WEARING THE BATTLE DRESS UNIFORM IN HOT WEATHER)

		Easy Work		Moderate Work		Hard Work	
WBGT Index (F°)	Heat Category (Flag Color)	Work/Rest Cycle (min)	Water Intake (qt/h)	Work/Rest Cycle (min)	Water Intake (qt/h)	Work/Rest Cycle (min)	Water Intake (qt/h)
78–81.9	1 (green)	NL	0.5	NL	0.75	40/20	0.75
82–84.9	2 (green)	NL	0.5	50/10	0.75	30/30	1.0
85–87.9	3 (yellow)	NL	0.75	40/20	0.75	30/30	1.0
88–89.9	4 (red)	NL	0.75	30/30	0.75	20/40	1.0
> 90	5 (black)	50/10	1.0	20/40	1.0	10/50	1.0

* The work/rest cycle times and fluid replacement volumes will sustain performance and hydration for at least 4 hours of work in the specified heat category. Individual water needs will vary ± 0.25 qt/h.
NL: no limit to work time per hour. Rest: minimal physical activity (sitting or standing) and should be accomplished in the shade if possible.
CAUTION: Hourly fluid intake should not exceed 1.5 qt. Daily fluid intake should not exceed 12 qt.
Mission-oriented protective posture (MOPP) gear adds 10°F to WBGT index.

Examples of work intensities:

Easy Work	Moderate Work	Hard Work
Weapon maintenance	Walking on loose sand at 2.5 mph, no load	Walking on hard surface at 3.5 mph, 3 40-lb load
Walking on hard surface at 2.5 mph, 2 30-lb load	Walking on a hard surface at 3.5 mph, 2 40-lb load	Walking on loose sand at 2.5 mph with load
Manual of arms	Calisthenics	
Marksmanship training	Patrolling	
Drill and ceremony	Individual movement techniques (ie, low crawl, high crawl)	
	Defensive position construction	
	Field assaults	

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fortable surroundings. It is clear that weather conditions determine the safety of physical activity and must be considered in scheduling the timing, intensity, and duration of training and operations. It also appears that the effects of heat and exercise stress exposure accumulate, and, as discussed above, the prior day's weather and amount of exercise should be considered in scheduling the day's activities.[58,207,253]

It is important to limit the total amount of strenuous exercise to the minimum necessary for mission accomplishment. Trainees are at particularly high risk for EHI because of their need to succeed in a competitive environment.[39,241] Trainees often work at rates higher than the highest work rates used to determine traditional work/rest guidance. In addition, these individuals are often at low levels of acclimatization and aerobic fitness. They may also have febrile responses to immunization or infection and be experiencing sleep loss and psychological stress. The motto "Train, Not Pain" needs to replace the popular phrase, "No Pain, No Gain." These principles apply to protection against musculoskeletal injury as well as EHI.[254]

Prevention of EHI requires minimizing the use of heavy or retentive clothing, particularly helmets, jackets, and vapor-impermeable materials.[255,256] Lightweight, loose-fitting clothing should be worn to protect from sunburn and allow air circulation to enhance sweat evaporation.[82,257–260] Outdoors, provision of shade and air movement at the training areas is important; indoors, provision of air conditioning, ventilation, low vapor pressure, heat insulation, and reflective shielding will minimize the effects of heat stress. Individuals should spend as much of the day as possible in cool, low-humidity surroundings with good air movement (only 2 h/d of exercise in the heat are needed for heat acclimatization).[238,261,262]

Certain medical conditions and medications can put an individual at high risk for developing heat illness.[50,139,162,170,179,242,263–269] These can make adjustment to hot environments more difficult and include acute illness (especially infections), febrile conditions, chronic illness, prior history of heatstroke, pregnancy, obesity, skin disorders, certain genetic conditions, sunburn, and poor hygiene (ie, dirt that clogs pores, impairs sweating, and insulates skin, as well as predisposes to infection). Medications, "ergogenic" nutritional supplements (with ephedra alkaloids), alcohol, caffeine, loss of sleep, and missed meals can also predispose to EHI.[50,270,271] Categories of pharmacological agents that interfere with thermoregulatory mechanisms are listed in Exhibit 6-2 of Chapter 6, Prevention of Heat Illness.

Prevention of EHI during strenuous exercise requires proper provision for water and salt requirements.[8,33,64,272–274] Leaders must force and verify hydration.[21,83,275] Palatable water must be available nearby, and water intake must be monitored.[50,276,277] Water requirements are not reduced by any form of training or acclimatization; in fact, they are increased with acclimatization owing to increased sweating. Tolerance to dehydration cannot be developed, and water supplies are as important for survival in a military environment as are food and ammunition.[83] Exhibit 7-8 illustrates the extreme hazards of strenuous exercise when overzealously conducted in a warm or humid environment. It also reminds us that in the 1950s, "water discipline" referred to restricting water intake under the false theory that one could be acclimatized to water deprivation. During the 1960s it became clear that acclimatization actually increases the need for water (due to increased sweating), and that those theories of the 1950s were fatally flawed.

Thirst is not an adequate guide to water needs because it lags behind water deficits by at least 1 pint.[18,50,273,278] Urinary volume and color, dryness of the mouth, changes in body weight, and orthostatic blood pressure (tilt tests) can be used as guides to adequacy of hydration.[277] An advantage to carbohydrate or electrolyte beverages beyond their palatability has not been established, and high-sugar solutions may impede water absorption in the gut.[279–283] Salt losses should be made up with the usual military high-salt diet, not with salt tablets.[49,82,84] In EHI, water losses usually exceed salt losses, and salt replacement should follow (not precede) water replacement. Replacement of salt without water will exacerbate cellular dehydration, increase water requirements, and may induce profound hypernatremia.[73] Thus, we strongly recommend against the use of salt tablets. The following is a good rule of thumb: *Replace water losses hour by hour, and salt losses day by day*.

Personal education is the mainstay of prevention of EHI. Each individual should be fully aware of EHI, its signs, and its prevention.[149,171,234,284,285] Personal awareness and common sense will avoid most problems.[166,191] Individuals should understand the early symptoms and signs of EHI, and increased susceptibility with use of certain medications and during and after recovery from acute (even minor) illness. It is important to avoid excessive fatigue, get adequate sleep, maintain calories and adequate salt in the diet, and avoid alcohol and caffeine. Each individual must be responsible to maintain adequate water intake, which requires drinking when not thirsty and monitoring urinary volume and color, weight changes, and so forth.[262] Individuals

EXHIBIT 7-8

HEAT DEATHS DURING OFFICER CANDIDATE SCHOOL

July 1953

The Korean War was still a threat to world peace, and young American men were being drafted into a war that was inevitable but had not been accurately anticipated by either our intelligence agency or our armed forces. Among the best fighters of this fledgling United Nations force were the US Marines, whose members were still for the most part volunteers, and whose officer corps was the product of the Naval Academy, Naval Reserve Officer Training Corps graduates, a few promoted from the enlisted ranks, or recent volunteers who were commissioned after joining the Platoon Leaders Class while in college or from the Officer's Candidate Course immediately after graduation. This last group entered the fray with no knowledge of the military, were inducted with no physical requirements other than a routine and cursory physical examination, and were given a date to report to the US Marine Corps Base at Quantico, Virginia. It was into this group that I fell.

I clearly remember one prospective fellow candidate at the induction center who seemed overweight and exhibited a notable lack of muscular attributes. I had been a college football player, varsity wrestler, and tennis player and had some misgivings about my own capacity for enduring the ordeal that lay ahead. I queried this fellow traveler about his ambitions and selection of the Marines as his choice to serve. I remember his reply, words to the effect of "Well, I had a good time at college and I thought this might be a good way to get back into shape."

The day we reported in was no small awakening to what was in store. We ran everywhere, were insulted and humiliated by the noncommissioned officers, learned what fear was, and finally learned how to get along in a totally unmanageable situation. We all struggled, but soon it became obvious that some of our contingent had chosen their branch of service unwisely and were doomed to fail from the start. The physical demands grew rapidly, and I found myself at the point of exhaustion many times. Some struggled along on pride alone, and I wondered how they had lasted as long as they did.

We were in our third week, as I remember it. We fell out in the dark of morning, did 20 or so minutes of calisthenics, swinging our 9-plus-pound rifles over our heads (the "muzzles and butts" routine), then off to the mess hall running in cadence. I lost more than the usual amount of sweat that morning—it seemed unusually warm that day. Upon returning to the barracks we underwent the usual policing of the area; "squared away" our gear; and went out into the warm, sunlit day with full packs, ammo belts, helmets, and M-1 Garand rifles. After a brief "All present and accounted for" response to the sergeant, we were off on our first "conditioning hike" along the fire line—a trail carved out in the hillsides, partially as a firebreak but also to keep the power lines free of tree limbs.

We each had one canteen of water and were expected to take salt tablets, although whether or not we took them is vague in my memory. We had been told about water discipline—not to squander in answer to thirst—and that part of our training was to acclimate to water need by disciplining ourselves and not satiating our thirsts. As we moved along at a quickstep, Company C was in the lead; we young laden-down candidates struggled to keep pace with our unladen sergeants, which kept us gasping for air. Across the plain and then the hills. Initially none faltered, but our line began to "string out" and I first became acquainted with the word "straggler." The air was unusually still and seemed heavy. As the sun climbed the heat became intense, and off we went up another hill. Finally we halted, gasping, soaked with sweat, and crumpled to the ground for a very short rest. We were ordered to take out our canteens and pour some of our water on the ground. Believe me, we did so sparingly. We were to learn that survival in the rugged clime of Korea would depend on how much more ammunition we could carry than water. Then we were off again.

We stopped about every hour and were allowed to drink, but we were all overly cautious and were never satiated. Our water had become our most precious item. By noon the heat had become oppressive and I realized how wobbly my legs were; others were stumbling, swearing silently between gasps, and longing for the next break, the next sip. Rumors were rampant: everything from "put a stone in your mouth and suck on it" (supposedly to quench thirst) to "there is a water buffalo (a mobile water tank) a few hills hence."

(**Exhibit 7-8** *continues*)

Exhibit 7-8 *continued*

In recollection the details become murky. We must have had hot chow in the field at noon and had our fill of water. The rumor came down that the temperature was over 95°F. Some had dropped out, but none in my platoon; they were apparently on their way back in trucks. Then we were off again. The rests were unrewarding; each successive time it became more difficult to get started again. The sergeant would cry out "Saddle up." Finally we were on our way back, and I remember it being a little easier; however, we could not possibly have acclimat**iz**ed so quickly. Finally, the last hill, and the plain loomed into sight, which seemed to invigorate those of us in the lead.

When standing in formation at the end of the ordeal, I found myself having to consciously maintain my balance; then finally came the cry, "Fall out." The rush for the sink faucets and water coolers was insane—no longer were we gentlemen—some were finally shoved aside to keep the line moving. Others headed to the showers to drink but realized that all had to share; some showered with their fatigues on. Within half an hour we were cleansed, replenished with water, dressed in our khakis, and back in formation.

The rumor was passed that a candidate in the opposite company had collapsed near the end of the maneuver, had been dragged across the plain by his comrades, and was no longer breathing when he was dropped in front of the company area. We were marched to the front of the mess hall where we stood in formation in the hot afternoon sun; the temperature had fallen a few degrees from 98°F. A candidate from the company in front of ours suddenly collapsed and was taken to the Naval Hospital at Quantico, where he expired in the throes of hyperthermia. A third candidate became delirious that evening and was rushed to the Naval Hospital at Bethesda, where he succumbed of "heatstroke."

Within a week some of the training command were relieved of their positions, a congressional investigation was ongoing, and the "conditioning hikes," as our maneuver had been listed on the schedule, were now entitled "terrain appreciation" and were without full gear. We all carried folding camp stools; rested every 50 minutes; and although the theory of water discipline and acclimation to water deprivation was not disavowed, we were no longer asked to pour some of our precious fluid on the ground. The water buffaloes were always in place along the trails.

I later learned that the first to die that day had been the young officer candidate I had met at the induction center who had joined to get himself into shape. The other two were unknown to me. Four years later I resigned my regular commission in the Marine Corps. By coincidence I received my discharge papers in San Francisco from the same lieutenant colonel who had commanded that ill-fated battalion. He was now a major.

Exhibit prepared for this textbook by Brigadier General John Hutton, Medical Corps, US Army (Ret), Department of Surgery, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799

must be aware of the need to hydrate ahead of thirst and before and during exercise. Trainees should wear appropriate clothing, seek shade, utilize rest periods, and adjust the intensity and duration of exercise according to environmental conditions and their own sense of well-being.[20,248,249,258,275,286] Buddies can recognize the symptoms and signs in each other, assisting in early recognition and management.[49,274] Each individual must remember the motto "Train, Not Pain" to prevent serious complications of EHI.

SURVEILLANCE AND REPORTING OF EXERTIONAL HEAT ILLNESS

Surveillance for EHI provides data that are essential for maintaining a healthy population and evaluating prevention and management policies.[287] Only through data-based policy decision-making can prevention of EHI and its serious complications be minimized.[200,201] Management and prevention of EHI requires meticulous record keeping that includes both the circumstances under which the illness occurred and the time course of clinical symptoms and signs. An effective heat illness surveillance and prevention program requires active monitoring not only of EHI cases, outcomes, and all exercise-related deaths, but also of training activities, personal risk factors in the training population, and weather conditions. One must be constantly aware of the amount and timing of exercise, adherence to rest cycle and intensity guide-

lines, and training circumstances. Effective prevention requires knowledge not only of the availability of water and shade, for example, but also of the planned and actual consumption of water and use of shade. Attention must also be given to the clothing and gear involved and to the availability and capability of on-site medical personnel, equipment (including thermometers, ice, and intravenous fluids), and transportation to emergency care facilities.

Weather monitoring involves both hourly determination of the WBGT at the training sites and communication of those measurements to the trainers so they can appropriately adjust their vigilance and training activities. In the military, this is often done utilizing a flag system, which corresponds to heat categories described in the regulations (see Exhibit 7-7), with corresponding activity limitations.[20,49,248,249,275]

Monitoring of personal risk factors in the training population is more difficult, as the physical fitness, acclimatization, and medical status of each individual must be continuously assessed. Keeping track of minor illnesses, medications, hydration, diet, sleep patterns, and use of alcohol and caffeine is somewhat challenging. However, these factors may be critical in susceptibility to serious EHI. Activities need to be adapted to the age, physical capacity, and current fitness levels and medical status of each individual.

Monitoring of cases and medical outcomes involves assessment of triage and immediate care by an assigned acute care or surveillance officer. Each case must be reviewed and classified as to type and severity, with attention given to risk factors and training circumstances. Patterns of illness and relation to training activities can then be evaluated and trends analyzed. Clusters of cases can be explored to determine the specific circumstances that have produced these casualties. The discussion that follows pertains to military surveillance methodology and presupposes that the casualty has entered the military medical system.

A set of standard clinical forms is attached to the end of this chapter, which can be used both as the clinical record and as a means to collect data that are necessary for appropriate EHI surveillance. (The attached forms can be duplicated by the reader for clinical use.) The record should begin in the field (the EHI Field Form is usually completed by a corpsman or medic), with observations by the field medical team of the presenting clinical symptoms, vital signs, neurological status, and early time course of the illness. The field medical team should also provide information regarding the circumstances of EHI occurrence (eg, training events, weather conditions, clothing, and a narrative description of the clinical presentation).

In the medical clinic or emergency department (the two EHI Clinic Forms are usually completed by a nurse), assessment of vital signs and neurological status should continue every 5 minutes until the patient is stable and cooled to a temperature that remains below 102°F (39°C). The listing of symptoms and signs during the first hour of illness and of risk factors (eg, height, weight, physical fitness, amount of fluids consumed, sleep loss, coexistent illness) are important, in addition to notation of the treatments given, diagnostic tests obtained, and a medical review of systems.

The medical workup can be recorded in an efficient manner that facilitates standardization of laboratory workup, diagnoses, and dispositions (the EHI Medical Form is usually completed by the primary medical provider). This form contains blocks for recording fluid intake and output and for results of diagnostic tests, and blocks for diagnosis and disposition of the patient, as well as an open-ended section for medical history (*s*ubjective), physical examination (*o*bjective), *a*ssessment, and *p*lan (also called the SOAP notes). Summary surveillance reports can be generated from the clinical forms, and should include counts of EHI cases by severity, training event, and weather conditions.

SUMMARY

EHI represents a broad spectrum of disease associated primarily with exercise in a warm environment. Symptoms and signs of exertional heat exhaustion, heat injury, heatstroke, and rhabdomyolysis result from detrimental consequences of elevated temperature and circulatory and metabolic products of exercise and the body's thermoregulatory response. Hyperthermia, dehydration and electrolyte imbalance, neurological manifestations, impaired renal function, muscle symptoms, and rhabdomyolysis comprise the predominant clinical features of EHI. These require diligent assessment and aggressive management to prevent progression to more-severe and often life-threatening illness. Progression to severe EHI usually involves muscle- and liver-cell breakdown (cell lysis syndrome), metabolic acidosis, acute renal failure, cardiovascular collapse, DIC, coma, and death. It generally requires management in an intensive care unit.

Early clinical management of EHI requires immediate assessment of vital signs and mental status in the field, with rapid cooling and rehydration.

Moderate and severe cases need frequent reassessment and laboratory evaluation. Aggressive cooling and correction of fluid, electrolyte, and acid–base problems constitute the mainstay of treatment. With rapid and aggressive treatment, guided by meticulous clinical and laboratory reassessment, serious complications are rare. It must be remembered, however, that subtle deficits and increased susceptibility to heat stress persist for several days following even moderate EHI, so casualties should not be immediately returned to regular strenuous activities or mentally demanding tasks.

EHI has been prominent in military training and operations, representing an important cause of morbidity, as well as significant mortality. Acclimatization to hot environments provides some mitigation of EHI morbidity. However, given extreme environmental circumstances and excessive workload, as well as various individual medical and other risk factors, it is always a threat during training and operations.

The key to prevention of EHI is education both of unit leaders and individuals to maintain activities in accordance with the environmental conditions, their physical fitness and acclimatization status, and their individual medical and other circumstances that determine susceptibility. They must remember the mottoes "Train, Not Pain" and "Replace water hour by hour, and salt day by day." Personal awareness and common sense will avoid most problems. Buddies can recognize the symptoms and signs in each other, assisting in early recognition and management of EHI.

Surveillance for EHI encompasses not just tabulating EHI cases but also assessing severity and monitoring environmental conditions; training circumstances; adherence to work/rest cycle guidelines; and individual physical fitness, hydration, and medical status.

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Chapter 7: ATTACHMENT; Clinical and Surveillance Forms

Exertional Heat Illness FIELD FORM

Patient ID: _____

Date/Time: _____

Training Day _____

Time of onset of illness: _____ AM/PM FIELD WBGT: _____ °F

CIRCUMSTANCES before and at onset of symptoms:

Activity: ☐ Running* ☐ Marching* *distance miles _____

Training Event: _____ ☐ Table PT ☐ Other _____

In your own words, DESCRIBE THE ONSET OF ILLNESS:

CLOTHING: ☐ **PT Gear:** ☐ w/sweatshirt ☐ w/sweatpants **Shoes:** ☐ athletic ☐ boots

Utilities: ☐ w/blouse ☐ wo/blouse **Accessories:** ☐ hat ☐ helmet ☐ vest ☐ rifle

☐ pack _____ lbs ☐ MOPP _____ ☐ Other: _____

SIGNS AND SYMPTOMS:

HYPERVENTILATION: ☐ NO ☐ YES

WOBBLY/STUMBLING: ☐ NO ☐ YES VOMITING: ☐ NO ☐ YES

COLLAPSE (No LOC): ☐ NO ☐ YES SEIZURE: ☐ NO ☐ YES

LOSS OF CONSCIOUSNESS: ☐ NO ☐ YES — DURATION _____ (minutes)

PAIN/CRAMPS: ☐ NO ☐ YES — LOCATION _____

OTHER: _____

1—VS Time: _____ **T:** _____ °F **P:** _____ **R:** _____ **BP:** ___/___ **Orientation:** ☐ person ☐ place ☐ time

CNS Scale: ☐ 8 (Normal) ☐ 7 ☐ 6 ☐ 5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 (Deep Coma)

2—VS Time: _____ **T:** _____ °F **P:** _____ **R:** _____ **BP:** ___/___ **Orientation:** ☐ person ☐ place ☐ time

CNS Scale: ☐ 8 (Normal) ☐ 7 ☐ 6 ☐ 5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 (Deep Coma)

TREATMENT IN FIELD: Time cooling began _____

COOLING METHOD: ☐ Remove clothing ☐ Wet down ☐ Sheets/Ice water

☐ Oral fluids: Amt: _____ (quarts) ☐ Tub ☐ Shower ☐ Ice packs

☐ MEDICATIONS/IV FLUID/OTHER:

TIME OF ARRIVAL AT CLINIC/ER: Field Watch _____ Clinic Clock _____

FIELD RECORDER (print) _____

Exertional Heat Illness CLINIC FORM 1

CONDITION ON ARRIVAL:

Patient ID: _____
Date: _____
Arrival Time: _____

EXAM (every 5–10 minutes) **Time:**						
***Rectal Temperature** (°F)**:**						
Pulse:						
Respiratory Rate:						
Blood Pressure:	/	/	/	/	/	/
Orientation (0–3/3)**:**	/3	/3	/3	/3	/3	/3
CNS Scale						
8 Normal (alert, oriented, cooperative)	☐	☐	☐	☐	☐	☐
7 Drowsy/Lethargic/Dazed (fully oriented and arousable)	☐	☐	☐	☐	☐	☐
6 Confused—appropriate (cooperates, partial or varying orientation)	☐	☐	☐	☐	☐	☐
5 Confused—inappropriate (disoriented, uncooperative, but purposeful)	☐	☐	☐	☐	☐	☐
4 Delirious (agitated, combative, fragmented behavior, out-of-touch)	☐	☐	☐	☐	☐	☐
3 Obtunded—barely responsive (wards off pain, obeys a command, utters a word, or makes eye contact)	☐	☐	☐	☐	☐	☐
2 Light Coma—w/response to pain (reflex response to pain)	☐	☐	☐	☐	☐	☐
1 Deep Coma—no response to pain	☐	☐	☐	☐	☐	☐

***When changing thermometers, record the last reading and time with the old thermometer, then the first reading and time with the new thermometer, and note the change.

NOTES:

Race _____
Height _____
Weight _____
Age _____
Sex _____

CLINIC RECORDER (print) _____

Exertional Heat Illness CLINIC FORM 2

Patient ID: _____

Date/Time: _____

Describe circumstances, onset of illness, and initial symptoms:

SYMPTOMS: (ENTIRE FIRST HOUR) Reevaluate when patient has recovered

GENERAL:
- ☐N ☐Y THIRSTY
- ☐N ☐Y SCARED/NERVOUS
- ☐N ☐Y WEAK
- ☐N ☐Y HEADACHE

EXTREMITY MUSCLE:
- ☐ N ☐ Y MYALGIA
- ☐ N ☐ Y CRAMPS
  loc: _____

ORTHOSTATIC:
- ☐ N ☐ Y FAINT/DIZZY
- ☐ N ☐ Y BLURRED VISION
- ☐ N ☐ Y TUNNEL/FADING VISION
- ☐ N ☐ Y VISUAL LIGHTS/SPOTS
- ☐ N ☐ Y WOBBLY/STUMBLING
- ☐ N ☐ Y COLLAPSE—No LOC
- ☐ N ☐ Y SYNCOPE/Brief LOC
  duration _____ (min)

PULMONARY:
- ☐ N ☐ Y HYPERVENTILATION
- ☐ N ☐ Y SHORT OF BREATH
- ☐ N ☐ Y NUMBNESS/TINGLING
  loc: _____

GASTROINTESTINAL:
- ☐ N ☐ Y NAUSEA
- ☐ N ☐ Y VOMITING
- ☐ N ☐ Y ABDOMINAL CRAMPS
- ☐ N ☐ Y DIARRHEA

SWEATING: ☐ PRESENT ☐ NONE

SLEEP last 24 hours: _____ hours

AMNESIA: ☐ N ☐ Y (List events not remembered before and after onset, and estimate duration of memory loss):

☐ **OTHER:**

WATER: (qt)	**CAFFEINE:**	**LAST MEAL:**	**SMOKING:**
Last 12 h: _____	Last 12 h (#cups/cans)	☐ Light ☐ Mod. ☐ Heavy	☐ None Last cigarette (time):
PUNCH/SODA (qt)	**Coffee** _____	Time _____ AM/PM	☐ < 1 ppd _____ AM/PM
Last 12 h: _____	**Tea** _____	**Alcohol** (# drinks)**:**	☐ 1–2 ppd
	Cola _____	Last 24 h: _____	☐ ≥ 2 ppd

REVIEW OF SYSTEMS FOR PAST 2 WEEKS:

			Date of onset:				Date of onset:
"Cold" (URI)	☐ N	☐ Y	_____	Syncope	☐ N	☐ Y	_____
Sore throat	☐ N	☐ Y	_____	Palpitations	☐ N	☐ Y	_____
Fever/Chills	☐ N	☐ Y	_____	Nausea	☐ N	☐ Y	_____
Sunburn > 20%	☐ N	☐ Y	_____	Vomiting	☐ N	☐ Y	_____
Cellulitis	☐ N	☐ Y	_____	Diarrhea	☐ N	☐ Y	_____
Vaginitis	☐ N	☐ Y	_____	Constipation	☐ N	☐ Y	_____
Dysuria	☐ N	☐ Y	_____	Bleeding	☐ N	☐ Y	_____
Hematuria	☐ N	☐ Y	_____	Fracture	☐ N	☐ Y	_____
☐ OTHER: Describe/date of onset:				Strain/Sprain	☐ N	☐ Y	_____
				(Females) LMP date _____			

Immunizations in past 2 weeks? ☐ N ☐ Y — Date and type: _____

PRIOR HEAT ILLNESS: ☐ N ☐ Y — Date and Dx: _____

TREATMENT IN CLINIC:

COOLING METHOD: ☐ Water and Fan ☐ Ice Sheets ☐ Immersion in Tub ☐ Shower ☐ None ☐ Other _____

MEDICATIONS/OTHER:

Exertional Heat Illness MEDICAL FORM

Patient ID: _____

Date/Time: _____

LAB TESTS ORDERED: ☐ TC ☐ CBC ☐ HEAT PANEL ☐ TESTS FOR ARF ☐ OTHER:

☐ U/A → ☐ before ☐ after hydration ☐ repeat in 12–24 hours U/A, CBC, & Heat Panel

IV FLUIDS				
Time start	Solution	Amt	Time finish	Amt infused

PO INTAKE		
Time	Type	Amt

OUTPUT		
Time	Type	Amt

LAB RESULTS (First set) **TIME DRAWN:** _____ **AM/PM**

Na		Glucose		CK		Hgb		Urine Specific Gravity	1.0_ _
K		Creatinine		AST		Hct		Urine pH	
Cl		Osmolality		ALT		WBC		Dipstick + 's	
HCO_3		Uric Acid		LDH		Plts		Occult Blood	
								Casts*	
BUN						Diff		WBC/hpf	
								RBC/hpf	

*especially pigmented granular casts

DIAGNOSIS:
☐ HEAT EXHAUSTION ☐ HEAT CRAMPS
☐ HEAT INJURY ☐ DEHYDRATION
☐ HEATSTROKE ☐ Parade Syncope/External Collapse
☐ RHABDOMYOLYSIS ☐ Other _____

SEVERITY (In each category, score severity level 1–5, or comment.)

Dehydration: _____

Encephalopathy: _____

Renal Function: _____

Cell Lysis: _____

Other: _____

DISPOSITION: ☐ HOSPITAL ☐ QUARTERS____days ☐ LIGHT DUTY____days ☐ Regular Duty (RTD)

EVALUATION (H/P): LIST all current medications, chronic illness, and any illness in past 2 weeks.

Patient healthy before exercising? ☐ YES ☐ NO

(Continue on additional page if needed)

MEDICAL RECORDER (print) _____

Chapter 8

EXERTIONAL HEATSTROKE IN THE ISRAELI DEFENCE FORCES

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INTRODUCTION

Most victims of exertional heatstroke (EHS) are highly motivated, healthy, young individuals who exert themselves beyond their physiological capacity (Exhibit 8-1). In 1989, Sir Roger Bannister commented on EHS in the armed forces in a letter to *The Times* of London:

> The notion that courage and esprit de corps can somehow defeat the principles of physiology is not only wrong but dangerously wrong; life can unnecessarily be lost.[1]

Very often military personnel, especially in their basic training regimen, exert themselves to the edge of their physiological ability. We in the Israeli Defence Forces (IDF) also intentionally encourage motivation and push the soldiers to perform to the edge of their ability, with very limited safety margins. Although motivation is very important in "courage and esprit de corps," it might become a noxious agent. Therefore, commanding officers should be aware of their subordinates' ability, monitor them during training, identify potential risk factors for heatstroke, and watch that their subordinates do not exert themselves beyond their capacity.

In a review of 82 cases of EHS that occurred in the IDF during the years 1988 through 1996,[2] most cases were found to have occurred during basic training (57%); an additional 21% occurred during screening tests for special forces—in which motivation is a key issue. Most of the cases of EHS occurred in highly motivated and relatively unfit soldiers during short marches lasting less than 2 hours (46% of all cases that occurred during marches) or during short distance (< 5 km) runs (57% of all the cases that occurred during runs).

EXHIBIT 8-1

EXERTIONAL HEATSTROKE: THE ILLNESS OF THE OVERMOTIVATED

A squadron of highly motivated special forces soldiers was engaged in an intense physical training regimen under desert conditions; the temperature of the ambient air was 40°C to 45°C. Two of the soldiers, who had been absent for several weeks from regular training with the squadron, joined their friends only on the fourth day of the maneuver. At the end of this day, these soldiers suffered from dizziness, headaches, disorientation, nausea, and confusion; their friends described their behavior as "irrational and strange." They were given fluids (intravenous infusion of Hartman's solution) but no measurements of body temperature were taken.

After a 4-hour night rest, and without seeking medical advice, the soldiers continued with regular training, which was carried out in very hot climatic conditions and in difficult terrain. At midday both soldiers collapsed within 15 minutes of each other. Their heart rates were high, they vomited and were tachypneic, but body temperature was not measured. The medic made a diagnosis of "heatstroke," which was later confirmed in the emergency department and at the postmortem examination.

A PREVENTABLE CONDITION

The Israeli experience indicates that prompt recognition, attention, and treatment usually result in complete recovery from EHS, and that EHS can be prevented by following simple regulations and proper health education.

Exertional heat illness is a sporadic phenomenon. It follows that an individual's tolerance to heat is compromised by some underlying factors, many of which have been identified in the literature.[3] However, based on military experience in the young active population, four factors are of major importance in the development of EHS: low physical fitness, improper acclimation to heat, acute febrile illness, and hypohydration.[2] Although these factors relate to the individual, they can be identified by commanders and thus be controlled.

Preventive Measures

The understanding that human ability has its limits led to the issuing of simple regulations that proved to be helpful in reducing the number of heat casualties.[4] Cumulative experience suggests that

- physical efforts should match the individual's capacity,
- physical activity should be limited under severe heat load,
- rest periods should be scheduled during activity, and
- adequate hydration should be emphasized.

These measures are effective if they are targeted to the individual as well as to the organization. The mainstay is education.

Medical personnel who supervise events that involve strenuous activity must, prior to the exercise, examine, evaluate, and when necessary, eliminate a subject who is predisposed to heatstroke, or who, during the exercise, exhibits prodromal signs of heatstroke (discussed below). Medical personnel should also have the authority to cancel maneuvers and other strenuous activities when weather conditions are adverse. These simple measures, which were implemented in the IDF and have been in practice for many years, are also the official position of the American College of Sports Medicine.

In the IDF, each case of EHS is subjected to an inquest by an investigation board, whose members are nominated ad hoc. Its primary aim is to examine the circumstances of the case in detail and to draw the necessary conclusions. If the inquest finds that regulations have been disregarded, commanders are considered responsible, and disciplinary measures—which may include those resulting from courts martial—are taken.

The Heat Tolerance Test

Soldiers who have recovered from EHS undergo a standard heat tolerance test (HTT) 6 to 8 weeks after the injury to screen for congenital or acquired factors that might compromise body temperature regulation.[3] The test consists of walking on a treadmill at 3.1 mph (~ 5 km/h), 2% elevation, for 2 hours, in a climatic chamber set to 40°C, 40% relative humidity (rh). A normal thermoregulatory response results in the return of the soldier to training. If the thermoregulatory response is abnormal (~ 5% of cases), the soldier is scheduled for a second test 2 to 4 weeks later. If this test is again abnormal according to our standards, he is regarded as heat intolerant, and cannot continue his service in a combat unit. It is noteworthy that we would rather occasionally discharge a soldier from combat duty unnecessarily than have a heat-intolerant soldier return to it. (We do not advocate testing for malignant hyperthermia because the etiologies of malignant hyperthermia and EHS are different.)

Potential recruits to military service undergo the HTT as part of their medical evaluation if heat susceptibility is suspected based on family or medical history (eg, previous heatstroke, ectodermal dysplasia).

A QUESTION OF DIAGNOSIS

Theoretically, the diagnosis of EHS is simple: in a previously healthy individual who collapses while exerting in a hot environment for long periods and whose rectal temperature is above 40.5°C,[5–7] the diagnosis of heatstroke is virtually clinched. However, heatstroke is misdiagnosed when medical personnel adhere too rigidly to simplistic diagnostic schemes that require warm climate, very high body temperature, and lack of sweating.[5,7] It follows that the correct diagnosis of heatstroke should depend on the understanding of the composite clinical and pathological picture. The following case study is illustrative:

> Four days after recruitment, a healthy, 18-year-old military recruit participated in a 4-km march (average speed 4 km/h). The march started at 20:00 hours under a moderate heat load; the ambient temperature was 26°C, the rh, 78%. During the march he volunteered to carry a 10-L water canteen in addition to the 20-kg backpack that was carried by every soldier. On arrival at the base gate, the soldier collapsed. His face was red and he sweated excessively. At the base clinic, the patient was delirious, alternating with aggressive reaction, and he vomited several times. Body temperature was not measured.
>
> Hyperventilation, aggressive reaction, the history of a previously healthy young man, and the short distance of march led the physician to the misdiagnosis of conversion reaction. He was wetted with about 40 L of tap water. About an hour after collapse, 10 mg diazepam was administered intramuscularly and another dose of 10 mg was administered intramuscularly 1 hour later. The patient's condition continued to deteriorate and convulsions, accompanied by dark-colored vomit, appeared. At 00:30 hours he was evacuated to a medical center.
>
> On admission 4 hours after collapse, the patient was comatose, heart rate was 150 beats per minute, systolic blood pressure was 60 mm Hg, and breathing was shallow at a rate of 24 per minute. Rectal temperature (the first time measured!) was 39.6°C. Neurological examination revealed coma; spontaneous hand movements; low muscle tone; "doll eye" movement; dilated pupils, which reacted sluggishly to light; and diminished tendon reflexes.
>
> Laboratory values for the arterial blood gas analysis were as follows: pH, 7.32; P_{O_2}, 58 mm Hg; P_{CO_2}, 30 mm Hg; and base excess, –9.5. Chemical and hematological findings were as follows: creatinine, 4.4 mg/dL; glucose, 20 (and later, 426 and 546) mg/dL; aspartate serum aminotransferase, 550 IU/L; alkaline

phosphatase, 114 IU/L; amylase, 165 IU/L; hemoglobin, 8.4 g/dL; hematocrit, 38%; white blood cell count, 16,000/mm^3 with neutrophilia and many immature forms; platelets, 55,000/mm^3; prothrombin time, 10% of normal; partial thromboplastin time, 85 seconds; and blood specimen, did not clot.

The patient suffered severe watery diarrhea. Urethral catheterization produced a small amount of residual urine, and during the next hours the patient remained comatose and totally anuric. Syringe needle punctures resulted in long-lasting bleeding, and spontaneous mouth and nose bleeding appeared. Dark material ("coffee grounds") was aspirated from the duodenal tube. The patient was infused with 13 L of crystalloid fluid, albumin, blood, fresh frozen plasma, and cryoprecipitate. Dopamine and steroids were administered. However, blood pressure remained low, bleeding diathesis did not abate, and bloody diarrhea appeared. The patient died 27 hours after the collapse.

Autopsy was performed several hours later. The body and brain were edematous and subcutaneous petechiae were seen all over the skin. Lungs were congested with interstitial hemorrhages. Small gas bubbles were noted in the heart. Liver was necrotic with massive bleeding. Hemorrhages were seen all along the intestinal wall. Multiple foci of hemorrhage were seen in the kidneys and the urinary bladder. A remarkable amount of blood was aspirated from the retroperitoneal space. Autopsy confirmed the diagnoses of heatstroke, disseminated intravascular coagulopathy (DIC), acute renal failure, and shock.

The performance of strenuous physical exercise (athletic events, military training, hard labor) in the heat has been notorious as the cause for heatstroke. However, in many cases heatstroke occurs also at relatively low ambient temperature.[8–11] Proper measurement of body temperature is essential, and hyperthermia should be expected. It should be acknowledged, however, that adherence to a diagnostic yardstick requiring that rectal temperature be high has proven to be inadequate and misleading. Although body temperature probably exceeds a critical temperature at the moment of collapse, in many instances lower temperatures are recorded; the first recordings of body temperature may be delayed, carried out by untrained individuals, or measured incorrectly.[4,12–14]

Contrary to earlier beliefs,[6,7,15] in most cases sweat glands are still active at the stage of heatstroke collapse, and profuse sweating is likely to be present.[13] Dry skin (*a*) is evident in situations where climate is very dry and sweat evaporates very easily or (*b*) is a late phenomenon of heatstroke, which usually coincides with a severe degree of dehydration.[16]

The loss of consciousness is a constant feature of heatstroke. This abruptly puts to an end the physical effort. Once activity ceases, body temperature falls and the victim will usually then spontaneously regain consciousness. Heatstroke patients are thus likely to present at the emergency ward with only mild hyperthermia and mild central nervous system (CNS) disturbances.

THE CLINICAL AND PATHOLOGICAL PICTURES

A typical presentation of heatstroke is the sudden collapse of a highly motivated, relatively untrained subject during physical exertion carried out in a warm climate (see Exhibit 8-1). The collapse is accompanied by loss of consciousness, very elevated body temperature, rapid pulse, tachypnea, hypotension, and shock. Nevertheless, this "optimal" scenario does not always occur. When an individual collapses under circumstances of physical exertion, the working diagnosis should be heatstroke, unless another cause is obvious.[4,5,17]

The early clinical signs of heatstroke are nonspecific. Therefore, any systemic disease or condition that appears with fever and manifestations of brain dysfunction must always be kept in mind but considered only after the diagnosis of heatstroke has been excluded (Exhibit 8-2).[4,18]

Prodromal Symptoms

The presentation of heatstroke is usually abrupt, but about 20% to 25% of all casualties have prodromal symptoms lasting minutes to hours.[8,19] These include dizziness, weakness, nausea, confusion, disorientation, drowsiness, and irrational behavior. Lack of recognition of the first signs of disability, and in some cases an assumption that the victim is malingering, have led to misassessment of the true physiological status.

Clinical Picture

The clinical manifestation of EHS reflects the result of a direct thermal injury and cardiovascular collapse. The high temperature will precipitate cellular biochemical disturbances, and CNS and secondary noncardiovascular changes. Usually a very distinct pattern of events, which can be categorized into three phases, is evident: the hyperthermic phase, the hematological and enzymatic phase, and the renal and hepatic phase.

EXHIBIT 8-2

DIFFERENTIAL DIAGNOSIS OF HEATSTROKE

- Severe dehydration
- Encephalitis, meningitis
- Coagulopathies
- Cerebrovascular accident, epilepsy
- Hypothalamic hemorrhage
- Hypoglycemia
- Drug intoxication
- Envenomization (eg, a bite or sting from a snake or bee)
- Allergic reaction, anaphylactic shock

Adapted with permission from Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients. *Med Sci Sports Exerc.* 1990;22:8.

Hyperthermic Phase

CNS disturbances are present in all cases of heatstroke, as the brain is extremely sensitive to hyperthermia. Signs of depression of the CNS often appear simultaneously in the form of coma, stupor, or delirium, irritability, and aggressiveness.[5,20–22] Persisting coma after returning to normothermia is a poor prognostic sign.[5,19] Seizures occur in approximately 60% to 70% of cases.[13,19] Other findings include fecal incontinence, flaccidity, and hemiplegia.[5] Cerebellar symptoms, including ataxia and dysarthria, are prominent and may persist.[5,23,24] In more than two thirds of cases, pupils are constricted to pinpoint size.[19,25] Papilledema is present in cases of cerebral edema.[5] The cerebrospinal fluid and pressure are usually normal.[19,20]

CNS dysfunction is usually directly related to the duration of the hyperthermic phase and to circulatory failure. In most cases, coma persisting for as long as 24 hours, with subsequent seizures, may be followed by full recovery, without evidence of mental or neurological impairment.[20,26] Chronic disability may prevail for several weeks or months in the form of cerebellar deficits, hemiparesis, aphasia, and mental deficiency.[12,26,27] Only in rare cases, usually related to coma persisting for more than 24 hours, mental and neurological impairment may be chronic and persist for years.[28]

Gastrointestinal dysfunction, including diarrhea and vomiting, is a common occurrence. This may reflect poor perfusion or CNS impairment.[5]

Hyperventilation and elevation in body temperature are associated with primary respiratory alkalosis, which in cases of EHS, is masked by metabolic acidosis as a result of increased glycolysis and the development of hyperlactemia.[17,29–33] Hypoxia may be present in cases of respiratory complications.[6,29,34]

The Hematological and Enzymatic Phase

Heatstroke is associated with leukocytosis and significant alteration in absolute number and percentage of circulating lymphocyte subpopulations.[35] The white cell count may be in the range of 20,000/mm^3 to 30,000/mm^3 or even higher.[8,36] Hemorrhagic diathesis is frequently observed. It is clinically manifested by purpura, conjunctival hemorrhage, melena, bloody diarrhea, signs of hemolysis, hematuria, and pulmonary or myocardial hemorrhage.[5,22,37] The etiology is complex and multifactorial. It may be related to direct thermal effect on clotting factors (V, VIII),[38] decreased production of clotting factors due to hepatic dysfunction,[5,39] and thermal damage to the megakaryocytes in the bone marrow.[5,37] The most important mechanism may be a result of endothelial damage that leads to the release of thromboplastic substances, with resultant intravascular thrombosis and secondary fibrinolysis.[38,40] Hypofibrinogenemia, prolonged prothrombin time and partial thromboplastin time, elevated fibrin split products, and thrombocytopenia indicate the presence of DIC.[8,13,41,42] Clotting dysfunction peaks at 18 to 36 hours after the acute phase of heatstroke,[38] with prothrombin levels that reach a nadir on the second to third day.

One of the characteristic and almost pathognomonic features of EHS is the exceptionally high levels of various cellular enzymes. Evidence of skeletal muscle damage, displayed by elevation of serum creatine kinase (CK) activity (which is not always associated with myoglobinuria), occurs in a large percentage of patients with EHI. CK activity in the range of 10^3 to 10^4 IU/L is common (~ 5% of total CK may be of the myocardial isozyme, or MB, fraction [normal: 0–7 μg/L][12]). Peak values of CK occur 24 to 48 hours after collapse.[43] Although the dynamics in CK levels are not pathognomonic, they are helpful in making the differential diagnosis of heat-

stroke (see Exhibit 8-2) because in most other febrile states the enzyme levels will be within normal range. Elevated levels of serum aspartate aminotransferase (> 35 U/L), serum alanine aminotransferase (> 35 U/L), lactate dehydrogenase (> 190 U/L), and bilirubin (> 22 μmol/L) are also consistent features of heatstroke.[13,44] Levels of alanine aminotransferase greater than 1,000 U/L (normal: 10–35 U/L) are common in severe cases.[13,30]

Hypokalemia is found in the early stages of heatstroke.[5,32,45,46] Hyperkalemia may later develop as a result of metabolic acidosis, rhabdomyolysis, general cellular damage, and decreased renal perfusion.[12,13] Sodium levels are usually normal or slightly elevated due to dehydration.[47] In some cases, however, sodium levels may be slightly low, reflecting a state of hyperhydration (mainly because of unbalanced fluid replenishment during early treatment), or related to rhabdomyolysis.[48,49] Hypophosphatemia is frequently described as a result of the respiratory alkalosis[31,50] but has been observed also in patients with heatstroke who exhibit metabolic acidosis.[34,50,51] Hypocalcemia may reflect calcium phosphate or calcium carbonate deposition in damaged muscle.[12] In cases of acute oliguric renal failure associated with severe rhabdomyolysis, hypercalcemia has also been reported.[52] Hypomagnesemia may occur as a result of the increased loss of both calcium and magnesium in urine and sweat.[47]

Renal and Hepatic Phase

Disturbance in renal and hepatic functions characterize this late phase. High bilirubin levels, which may last several days, may reflect both hepatic dysfunction and hemolysis. Aminotransferases may originate from liver and muscle tissues.

Acute renal failure is a common complication of severe cases of EHS, occurring in approximately 25% to 30% of patients with EHS.[12,13,22] The etiology of acute renal failure is complex.[53] Decreased renal blood flow secondary to hypotension, following hypohydration and peripheral vasodilatation, is the major cause. Direct thermal injury may lead to widespread kidney tissue damage.[54] Intravascular clotting due to DIC may further contribute to acute oliguric renal failure.[40,54] Additional renal damage may also be inflicted by myoglobinuria.[55,56] Oliguria and anuria are characteristic features, the color of the urine being described as "machine oil," and with a low specific gravity.[13,53–55] Red and white blood cells, hyaline and granular casts, and mild to moderate proteinuria are commonly present.[54,55]

Pathological Picture

Degenerative changes and hemorrhages characterize the pathological picture. The former consist of swelling and degeneration of tissue and cell structures; the latter is often presented by widespread hemorrhages, which may vary in size from microscopic to massive bleeding.[57] The organs are generally described as congested or edematous, with increased weights and swollen cells.[5,8,13,22]

Brain edema or congestion is generally present. Swelling of nerve cells and degenerative neural changes are constant findings and could be traced through the successive stages from acute and chronic cell alteration to disappearance of neurons and their replacement by glia. Petechiae are common in the walls of the third ventricle and in the floor of the fourth ventricle.

Subendocardial hemorrhages, with predilection for the left side of the ventricular septum, are common, as is focal necrosis of the muscle fibers. Widespread necrosis of heart muscle, with a bleeding diathesis and myocardial infarction, are also described, with no abnormalities in coronary arteries. Striated muscles display degeneration, necrosis, and disruption of muscle fibers more prominently than in the myocardium. The lungs are congested and edematous. Numerous small pulmonary hemorrhages and massive pleural and intrapulmonary bleeding are presented. Pneumonia is occasionally evident.

The kidneys are always congested, with above-normal weights, and macroscopic hemorrhages are present in 20% of cases. Parenchymal damage is scant in very severe cases with early death. Pigmented casts, degeneration, and necrosis are often present in the lower nephron.

In the liver, centrolobular necrosis and hepatocellular degeneration are present around the widened centrolobular veins. Serial liver biopsies reveal hydropic swelling, extensive cholestasis, and leukocytic cholangitis.

Engorged intestinal vessels are a common finding, and the gastrointestinal tract may be the site of massive hemorrhages and ulcerations.

Subcutaneous petechiae are seen all over the skin. Degenerative changes are found in megakaryocytes, and basal cells and endothelial cell damage can be demonstrated. In small blood vessels, intravascular coagulation with thrombi is present.

MANAGEMENT AND PROGNOSIS

Heatstroke is the most serious of the syndromes associated with excess body heat.[58] It is defined as a condition in which body temperature is elevated to such a level that it becomes a noxious agent, causing damage to the body's tissues and giving rise to characteristic clinical and pathological syndromes affecting multiple organs.[13] The severity of the illness depends on (1) the degree of hyperthermia and (2) its duration, and is related to the duration of the temperature above a critical temperature.[59] Heatstroke is considered an extreme medical emergency that might result in death if not properly diagnosed and treated.

Management

To prevent or minimize expected complications, cooling should be initiated energetically immediately after the victim of EHS collapses, and minimally delayed only for vital resuscitation measures. In the field, the victim should be placed in the shade and his or her restrictive clothing removed. The skin must be kept wet with large quantities of tap water, and the body constantly fanned. These measures should not delay the rapid evacuation of the heatstroke victim to the nearest medical facility, which is of utmost importance.

Any time-consuming diagnostic procedures should be postponed until body temperature is controlled. At a medical facility, rapid cooling can be achieved by the use of ice packs, ice water baths, alcohol sponge baths, and with fans or air-conditioned rooms.[60–62] Sophisticated and expensive equipment, as suggested by Weiner and Khogali,[63] is unnecessary. The most practical and efficient method of cooling the body is by drenching it in large quantities of tap water. Tap water is readily available and does not require any complicated logistical arrangements. It eliminates the hazard of cold-induced vasoconstriction, which reduces the efficiency of heat dissipation.[61]

In heatstroke, the high body temperature is neither a fever nor the result of a hypermetabolic reaction but is rather the result of excess heat load (metabolic and environmental). Therefore, no drug is effective in reducing body temperature. Antipyretics should not be considered, as there is no shift in the thermoregulatory set point. Dantrolene is not effective because EHS is not linked to the uncontrolled release of calcium ions from the sarcoplasmic reticulum.

Rectal temperature must be measured to confirm the diagnosis of EHS. It is noteworthy, however, that in many instances the initial measurement will be lower than the critical core temperature (~ 41.0°C). This is due mostly to a delay in getting the first measurement, or due to faulty measurement.[4,12–14]

Drastic drop in skin temperature may induce violent shivering. Chlorpromazine (50 mg administered intravenously[18]) or diazepam[60] have been found to be effective in depressing the shivering and preventing an increase in metabolic heat production.

Blood pressure and pulse must be checked, a quick clinical examination performed, and if possible, urine and blood should be obtained for examination, prior to infusion of fluids.

Electrocardiographic findings are nonspecific and may include ST segment and T wave abnormalities and conductive disturbances. However, the evidence for such abnormalities in young patients with EHS is scarce. Nevertheless, continuous cardiac monitoring is recommended during the first 24 hours of hospitalization because of the potential rhythm disturbances that may emerge from electrolyte and acid–base imbalance.

The airway must be kept clear, and if the patient is comatose, insertion of a cuffed endotracheal tube should be considered because aspiration is common, especially when seizures occur.[4] If available, oxygen administration is advisable as metabolic demands are high and oxygenation may be hampered by pulmonary complications.[6] Positive pressure ventilation is indicated when supplemental oxygen alone does not suffice.[4,64]

A large-bore (eg, 18 or 20 French) intravenous catheter should be inserted without delay. When seizures occur they add to body heat storage, and thus cooling is inefficient; repeated doses of diazepam (5–10 mg) should be administered intravenously until the convulsions are controlled. Two liters of Ringer's lactate or saline should be infused in the first hour; later, fluids should be administered in accordance with the state of hydration.

Depending on the patient's hydration state, continuous fluid monitoring is required during the first 24 hours of hospitalization. A central venous catheter may be required to identify hypovolemic patients. A Swan-Ganz pulmonary artery catheter can provide more accurate guidance to fluid therapy when the circulatory state of the patient is unclear.[60] Monitoring the urinary flow using an indwelling urinary bladder catheter provides important information on the patient's state of hydration. Furthermore, urinary output reflects the kidneys' perfusion level and allows an early diagnosis of acute oliguric renal failure. Intravenous mannitol (0.25 mg/kg) or intravenous

furosemide (1 mg/kg) may be used to promote diuresis and prevent damage from myoglobinuria and hyperuricemia.[6,60] Anuria, uremia, and hyperkalemia are indications for peritoneal dialysis or hemodialysis.[65]

Acid–base abnormalities are usually corrected by cooling and by proper hydration not requiring specific treatment. Serum electrolyte imbalances should be followed closely, especially with regard to their relation with possible cardiac arrhythmias. Glucose levels should be checked repeatedly, as both hypoglycemia and hyperglycemia have been reported following heatstroke.[47]

Coagulation tests must always be performed on admission and at 12-hour intervals afterward. DIC may be recognized only after 24 to 72 hours. It starts abruptly and may be severe. Bleeding from venipuncture sites is characteristic; other common sites are bleeding into the skin, from the nose, gums, and respiratory and gastrointestinal tracts. The deficiency in clotting factors should be corrected by transfusion of fresh frozen plasma, cryoprecipitate, or platelet concentrates.

Acute hepatic dysfunction is exhibited by elevated levels of transaminases and bilirubin; peak levels are seen 36 to 72 hours after collapse, and high levels may last for several days.[13,43] Muscle damage is displayed primarily by marked elevation of serum CK activity levels, which peak 24 to 48 hours after collapse and usually recover spontaneously within 5 days.[43] Muscle and liver enzymes and bilirubin should be monitored but usually no drastic intervention (such as liver transplant) is necessary or advocated.

Prognosis

Since the end of the 1960s, it has been shown that more than 95% of victims of EHS survive when the following four measures are implemented[17,25]:

1. rapid decrease of body temperature,
2. arrest of convulsions,
3. proper rehydration at the site of the event, and
4. quick evacuation to a medical center.

The noxious effect on the tissues caused by heatstroke is directly and closely correlated with the degree of hyperthermia and its duration, and is a function of both the *elevation* and the *duration* of the elevation of the temperature above the critical value (the integrated time–temperature area).[59] Predictors of poor prognosis include (1) body temperature above 42°C,[13,25] (2) prolonged duration of hyperpyrexia, (3) prolonged coma, (4) hyperkalemia, (5) oliguric renal failure, and (6) high levels of aminotransferases.[17] The vast majority of survivors from EHS recover without sequelae.[6,13,26] However, some neurological deficits may be apparent in some patients, usually for only 12 to 24 months and only in rare cases for longer.[28]

According to the IDF experience, only six fatal cases of EHS were recorded in soldiers since 1980; in all cases, misdiagnosis, ineffective primary treatment, and delay in evacuation were the major causes for deterioration of the casualty's condition.

CONCLUSION

EHS is a state of extreme hyperthermia that occurs when heat that is generated by muscular exercise exceeds the body's ability to dissipate it at the same rate. The elevated body temperature becomes a noxious agent causing damage to the body's tissue, affecting multiple organs, and may be fatal if misdiagnosed or diagnosis is delayed. EHS is underdiagnosed because of adherence to simplistic and misleading diagnostic schemes that require long exertions in very warm climates and with very high body temperatures (> 40°C). Prompt diagnosis and treatment result in full recovery of most patients. Primary treatment should focus on clearing the airway, measuring rectal temperature, cooling the whole body, controlling convulsions, and prompt evacuation to a hospital.

The Israeli experience has proven that EHS can, in most cases, be prevented, education being the mainstay of this prevention. By providing proper instructions and following some simple regulations, the incidence of EHS has been reduced markedly. These instructions and regulations include grading training programs and matching effort to the individual's capacity, limiting physical activity under severe heat load, scheduling proper work/rest cycles, and providing adequate rehydration routines. Also, regulations for monitoring the medical status of soldiers participating in physical activities and evaluating the soldiers' medical histories should be implemented.

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Chapter 9

PRACTICAL MEDICAL ASPECTS OF MILITARY OPERATIONS IN THE HEAT

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INTRODUCTION

Military operations in hot environments present a special set of challenges to the medical personnel who must support them. Hot environments can affect both personnel and equipment performance and introduce special considerations into the planning and execution of a military operation. Vast resources have been committed to warfare in the 20th century, and in support of this, a significant body of research now exists on human performance in hot environments. Countermeasures to heat, extrapolated from physiological studies and mathematical models, have been developed and implemented by the US Army.[1,2] However, considerably less data have been gathered on how effective these countermeasures really are when used by military units under operational conditions.

It is impossible to write a textbook chapter on operations in the heat that does full justice to the wide variety of missions and challenges that are likely to be encountered by the different branches of the armed forces. Furthermore, the available countermeasures vary somewhat with mission, doctrine, and resources. For example, microclimate cooling vests may be highly practical for sailors working on the deck of an aircraft carrier, yet would be entirely inappropriate for light infantry engaged

TABLE 9-1

COMMON HAZARDS OF MILITARY OPERATIONS IN HOT ENVIRONMENTS

Hazard	Countermeasure
Decreased exercise capacity[1–7]	Prior physical fitness, acclimatization
Increased requirements for	
Water	Water discipline
Sodium and other electrolytes[8,9]	Acclimatization, food intake; supplementation is rarely necessary
Increased risk of certain types of injury	
Heat injuries	Water consumption, acclimatization, work/rest cycles, shelters, proper clothing, etc
Sunburn	Sunscreen, shelter, protective clothing (hats)
Miliaria rubra ("heat rash")	Personal hygiene, availability of cool environments
Decreased psychological performance on some tasks[10]	Work/rest cycles, cross-checking (buddy system), redundancy in key assignments
Involuntary dehydration*	Water consumption, cross-checking (buddy system)

* The tendency of individuals to replace less than their complete fluid deficits when allowed to drink ad libitum[11–15]

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in jungle warfare. Nonetheless, the success of all military operations requires careful attention to the fundamental problems of personnel, intelligence, training and operations, and logistics; and a detailed analysis of how ground forces cope with these problems may be enlightening to medical officers (MOs) in other branches of the armed forces. Accordingly, this chapter will detail practical considerations for the battalion-level MO who is supporting a unit deployed to a hot weather environment.

HEAT AS A THREAT TO MILITARY OPERATIONS

Many studies have measured the incidence of heat casualties among military personnel in a training environment.[3–8] The reported incidence of heat injury at basic military training facilities is typically about 5 to 8 cases per 10,000 troops per week,[3,5,7] although higher rates were reported in the Marine Corps prior to implementation of heat-related training restrictions.[4,7] New recruits and reservists typically have the highest rates of heat injury[3,4,6,7,9]; reservists appear to be at particularly high risk. Reported incidences of heat illness among reservists range from 33 to 49 cases per 10,000 troops per week (in the era before heat-related training restrictions)[4,7] to as high as 210 to 455 per 10,000 troops per week over a 2-week annual training period.[8]

By contrast, less is known about the incidence of heat illness during actual military operations, in part due to underreporting bias and the lack of uniformly accepted case definitions.[8] As a result, estimates vary widely; for example, the reported incidence of heat casualties was 11 per 10,000 per week in the Persian Gulf Command during World War II, whereas in Vietnam it ran as high as 378 per 10,000 per week.[8] Because these figures do not include soldiers who were adversely affected by heat but did not come to the attention of the reporting system, the true incidence of heat illness may have been much higher.

The effects of heat on personnel performance have been reviewed in detail elsewhere in this section of heat chapters. Table 9-1 recapitulates some of the most significant hazards encountered by units deployed to hot environments and some of the countermeasures available. As in all aspects of military operations, leadership and training are key to successful implementation of these countermeasures. Even in the best-trained unit, however, there is always a risk that the high motivation that soldiers typically bring to a mission will override the protective behaviors needed (water intake, rest, shelter, etc) to overcome the environmental threat. In this regard neither commanders nor troops seem to have changed—certainly not since this anonymous mid-19th century passage was written:

> At this season it is especially necessary for commanding officers to be alive to the dangers of prolonged over-exertion. … Commanding Officers of volunteers are very apt to err in this particular: and the spirit of their men is such that they shrink from complaint, and persevere in efforts which may easily, under a burning sun, become dangerous to life.[10(p627)]

Soldiers and their leaders must understand that—no matter how motivated they may be—failure to take protective measures against hot environments may result in mission failure. For example, it takes four troops to carry a single heat casualty any kind of distance. Thus, one heat casualty effectively means the loss of a fire team; two casualties mean the loss of a squad; four to six casualties can significantly degrade the combat power of an entire platoon (Exhibit 9-1).

PLANNING FOR DEPLOYMENT TO HOT ENVIRONMENTS

In the US Army, a battalion-level MO serves three roles: staff officer, leader in the medical platoon, and healthcare provider. Each of these roles affords the MO an opportunity to help the battalion cope with the environmental threat. As a staff officer, he is an advisor to the commander and is involved in the planning and day-to-day management of operations. As a leader in the medical platoon, he has a duty to make the platoon proactive and competent in preventing and treating heat casualties. Finally, as the primary healthcare provider to the battalion, he can help educate individual soldiers and leaders about the medical aspects of operations in hot environments. Obviously, as healthcare provider, he will also be involved in the medical management of heat- and sun-related injuries.

When planning for a deployment to a hot environment, it is important to remember that most of the threats that a military unit faces in temperate climates will still be present in a hot environment. A unit that is well-prepared to perform in the heat will nonetheless find its mission performance unduly hampered if inadequate provisions have been made for managing the illnesses and injuries that occur in all climates. Accordingly, although heat must be treated as a very serious threat to any op-

EXHIBIT 9-1

THE MOTIVATED POINT MAN

During a summer field training exercise in 1996 in Puerto Rico, a light infantry platoon was to conduct a surprise evening raid on the camp of (simulated) guerrillas. The approach to the camp required cutting a path through brush with machetes. This particular platoon was reputed to have some of the most fit and motivated soldiers in the battalion. They had been careful to increase their activity slowly over several days, to allow for acclimatization; had been briefed by a medic on the environmental threat; and were diligent about water supplies and intake.

The weather that evening was hot and humid when the lead squad left the staging area to cut through the brush. The soldiers were wearing summer-weight battle dress uniforms and ranger caps, and were encumbered with a typical combat load. Good progress was made initially, thanks to the vigorous efforts of Private Smith,* the point man (who was wielding the machete). The squad leader reportedly enforced water consumption and, to prevent Private Smith from overheating, replaced him as the point man when he started to look fatigued. Unfortunately, the replacement point man, Private Jones,* did not progress as rapidly as was desired in cutting through the brush. After considerable insistence on the part of Private Smith, the first point man, the decision was made to switch them. After a few minutes as point man, Private Smith complained of severe fatigue, muscle cramping, and light-headedness. He could no longer walk. The medic was called, who noted that Private Smith had an oral temperature of 101°F. Now a casualty, Private Smith was doused with cool water, and the medic started an intravenous infusion. It was also decided to evacuate the casualty to the battalion aid station. Access to the nearest road required further travel through uncut brush, which required the assigning of four soldiers as litter bearers and yet another as point man. Unfortunately, in the effort to evacuate their colleague promptly, one of the litter bearers developed symptoms of heat exhaustion, and four more troops had to be detailed to carry him. Having now lost an entire squad and recognizing that the loss of two soldiers to heat raised the possibility of even more heat casualties, the platoon leader decided to call off the exercise. Because the environmental threat to the light infantry platoon was inadequately managed, the opposition force prevailed that night without having to fire a single shot.

The foregoing illustrates how unforgiving the environmental threat can be. Even though the unit had been taking reasonable precautions against heat all along, the decision to allow a soldier who had already shown signs of fatigue to prematurely resume high-intensity activity in the heat led to a series of events that eventually resulted in scuttling the mission. Medical officers need to wonder whether Private Smith's insistence on being allowed to resume the point position was not itself an indication that his judgment was clouded by overheating and dehydration. Because individuals can be poor judges of their own hydration status[1] and core temperature,[2] medical officers must not allow a soldier's assurances (vehement as they may be) to be the sole deciding factor when making a duty assignment.

* Pseudonym

Sources: (1) Sawka, MN, Modrow HE, Kolka MA, et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1994. Technical Note 95-1. (2) Yaglou CP, Minard D. Control of heat casualties at military training centers. *AMA Arch Ind Health.* 1957;16:302–316.

eration, we must not allow the attention given to planning for heat to cause us to neglect other important operational issues. The general and environmental issues relating to deployment are discussed in Volume 3 of this textbook, and in another volume in the Textbook of Military Medicine Series, *Military Preventive Medicine, Mobilization, and Deployment.* For now, however, we shall focus on issues specific to hot environments.

Battalion MOs can and should play an integral role in the planning of military operations. An MO can provide a battalion staff with meaningful input into all four areas of staff concern: Personnel (S-1), Intelligence (S-2), Operations and Training (S-3), and Logistics (S-4).

Personnel Issues

In preparation for a deployment, the adjutant (S-1) of the battalion will need to provide the commander with an estimate of the number of combat-ready personnel available in the battalion and to

identify any critical unfilled positions. Therefore, in addition to advising the S-1 about the deployability of soldiers with known medical problems, the MO should also aim to identify personnel who may be at risk for heat or solar injury. Risk factors are listed in Exhibit 9-2; some of these (such as obesity and poor physical fitness) are modifiable and should be identified and managed as part of a unit's routine,

EXHIBIT 9-2

RISK FACTORS FOR POOR HEAT TOLERANCE AND HEAT-RELATED INJURY

- Obesity[1–4]
- Lower level of physical fitness[3,5,6]
- Prior history of heat injury[7,8]
- Being a new recruit or reservist[1,2,4,9,10]
- Febrile illnesses[11]
- Dehydration from any cause[12] (including diarrhea)
- Skin disorders:
  Miliaria rubra (heat rash)[13,14]
  Sunburn[15]
  Psoriasis[16]
  Burns
- Older age*[17,18]
- Medications that decrease sweating:
  Anticholinergic agents
  b-Blockers[19] (these also act by decreasing cutaneous vasodilation)
  Antihistamines
  Phenothiazines
- Medications that increase heat production:
  Amphetamines
  Cocaine
  Neuroleptics
- Medications that decrease thirst:
  Butyrophenone
  Angiotensin-converting enzyme (ACE) inhibitors†[20]
- Medications that cause hypohydration:
  Alcohol
  Diuretics

*Age may not be a risk factor for heat illness per se, once adjustments are made for confounders such as lower level of aerobic fitness, higher adiposity, etc.[21]However, because older individuals often do have one or more of these confounding risk factors, it is prudent to view them as belonging to a population that is at increased risk for heat injury.

†ACE inhibitors have been shown to decrease thirst in dialysis patients; if this holds true in normal patients, it could theoretically increase the risk of heat injury.

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ongoing healthcare program. Others, such as a prior history of difficulty acclimatizing to heat, may not be easily modified but are often manageable with proper preparation and planning.

Furthermore, the S-1 will probably want to review the unit's medical personnel assets. Although a well-maintained personnel roster should contain the names and military occupational specialties of all medical personnel in the battalion, a realistic assessment of the unit's medical capabilities should include an evaluation of the professional strengths and weaknesses of the medical personnel available. The MO is, by training and experience, in a position to provide valuable insight into these capabilities. By identifying areas of weakness early, the medical platoon can design an intensive and focused training effort in preparation for deployment.

One important but commonly overlooked medical asset in the US Army is the combat lifesaver. Combat lifesavers are nonmedical troops trained to perform advanced lifesaving tasks as a secondary mission.[11] They are given advanced first-responder training and additionally are taught how to start intravenous (IV) lines as an early step in the management of shock. They are issued a small medical kit that includes two 500-mL bags of saline for IV infusion. Although combat lifesavers cannot replace a medic, they do serve as important medical capability multipliers. This is especially important in units that have a limited number of medics. For example, in a light infantry battalion, the table of organization and equipment assigns only three medics to each rifle company; this means that each rifle company medic is responsible for an entire platoon of soldiers. Three or four combat lifesavers acting under the guidance of a medic can greatly enhance the platoon's ability to cope with heat casualties, which typically occur in clusters,[3–5,12] and to treat soldiers in a mass casualty situation. The training of combat lifesavers can be accomplished during peacetime with little extra effort; course materials and guidance on how to set up a training program are available from the Army Institute for Professional Development (Exhibit 9-3).

Intelligence Issues

To better assist the planning process, the MO will need intelligence on the environmental and medical threats the unit is likely to face in the theater of operations. An excellent source of this information comes in the form of country dossiers prepared by the US Armed Forces Medical Intelligence Center (AFMIC) located in Fort Detrick, Maryland (see Exhibit 9-3). Among other information, the dossiers include ambient temperatures (maximum, minimum, and average) by month, average monthly precipitation, geographic and demographic information of medical importance, discussions of medically significant plants and animals, and lists of endemic and epidemic diseases. The MO should plan to obtain a country dossier and summarize its contents for the battalion staff early in the planning process.

The battalion MO will also need to discuss how weather information is to be obtained and transmitted to medical personnel during the operation. It is well within the capabilities of the medical platoon to set up wet globe temperature (WGT) monitoring at the battalion aid station (BAS),[1] and this can provide timely and immediately accessible information. This can be done with a Botsball device (National Service Number [NSN] 6665-01-103-8547), a field-expedient instrument that measures the WGT.[1] It should be remembered, however, that the Botsball device can significantly underestimate the actual wet *bulb* globe temperature (WBGT) under some conditions (eg, hot, dry, windy weather) and should always be corrected accordingly, using the following formula[1]:

$$\text{WBGT (°F)} = (0.8)\ \text{Botsball temperature} + (0.2)\ \text{dry bulb temperature} + 1.3$$

However, weather forecasts will also be needed. Possible sources include the intelligence (S-2) sections of supporting units, air force weather liaison teams, national meteorologic agencies, civilian sources (newspapers, almanacs), and even Internet

EXHIBIT 9-3

USEFUL RESOURCES FOR THE BATTALION MEDICAL OFFICER

Combat Lifesaver Course Materials and Guidance:
Army Institute for Professional Development
Newport News, Virginia 23628-0001

Medical Intelligence:
Armed Forces Medical Intelligence Center
Fort Detrick
Frederick, Maryland 21702-5004

Environmental Medicine Resources:
US Army Research Institute for Environmental Medicine
42 Kansas Street
Natick, Massachusetts 01760

sources (when available).

Training and Operations Issues

Every heat casualty prevented thanks to peacetime training will potentially be one less patient who needs acute care during a deployment, and thus one more soldier available to accomplish the mission. Accordingly, MOs should help train the unit for the hot weather threat, as part of the unit's preventive medicine program. In addition, during a field deployment, the MO may be asked to provide ongoing advice on how to cope with the heat threat. Training and operations planning for a battalion are coordinated by the S-3 section; therefore, an MO who learns how to work effectively with this section will be in a position to have a broad impact on unit performance. Although the staff position is named Operations and Training (S-3), training occurs before deployment; so for purposes of this textbook, training is discussed before operations planning.

Predeployment Training

Table 9-2 describes a training program for hot weather operations. Some of the elements of this program (such as how to avoid insects and diarrheal diseases) should be a routine part of any unit's ongoing training plan, but they take on extra importance when a unit is faced with a deployment to a hot environment. Others, such as training that is specifically directed at acclimatizing the troops to heat, can be implemented if there are 7 to 10 days of advance notice. The preventive measures listed here have been discussed in greater detail in Chapter 6, Prevention of Heat Illness; we shall, therefore, highlight only a few important points.

Acclimatization Training. The most practical way to acclimatize troops to the heat remains a program

TABLE 9-2

GUIDELINES FOR PREDEPLOYMENT TRAINING FOR HOT WEATHER OPERATIONS

Training Component	Description
Physical Training	Safe, daily exercises in the heat of sufficient duration and intensity to produce profuse sweating
Water Consumption Practices	1–2 L/h of monitored water intake while physically active (depending on activity, environment, and protective clothing being worn). Consider prehydration with 300–500 mL of water prior to starting physical activity. Thirst: *not* a reliable indicator of dehydration. Urine: should be clear, colorless. Avoid massive water ingestion, as this can precipitate hyponatremia. Symptoms that do not respond to ingestion of 2–3 L water should be referred to a medic. Water consumed is far more effective as a coolant than water splashed on the skin. Water discipline is the first but by no means the only measure needed to prevent heat injury
Other Protective Measures Against Heat	Use work/rest cycles whenever possible. Keep clothing dry, loose. Use shelter and entrenchment (ground is cooler below the surface)
Skin and Eye Care	UV protection (sunscreen, sunglasses, lip ointment). Daily personal hygiene. Goggles in sandy or dusty terrain. Foot care
Countermeasures to Diarrheal Diseases	Hand washing. Daily personal hygiene. No eating or drinking from unapproved sources. Review field sanitation and hygiene practices
Countermeasures to Insects	Use of insect repellent. Shake out bedding, clothing, and boots before using. Use of insect netting
First Aid for Heat Injuries	Water intake. Rest. Shelter. Field-expedient cooling methods. Early evacuation, if indicated. Assess others in the unit for signs of impending heat injury
Environmental Awareness	Beware of sandstorms, flash-floods, nighttime cold
MOPP Training	Training in all levels of MOPP gear

MOPP: mission-oriented protective posture
UV: ultraviolet light rays

of daily exercise in hot weather, with increasing intensity and duration. The goal of each daily exercise session should be to produce profuse sweating without causing heat injury; it is therefore important for leaders to monitor their troops carefully during this period and to reduce the intensity at the first sign of trouble. Individuals vary in their rate of acclimatization, but substantial acclimatization will occur in most soldiers after 7 to 10 days of exercise in the heat.[13]

As part of acclimatization training, it is important to point out to troops that indigenous forces and allied units who have deployed earlier to the hot environment may, at least at first, be much better acclimatized than they are themselves. Underestimating the advantage that an acclimatized enemy has over unacclimatized friendly troops can be lethal. However, even when not faced with immediate enemy opposition, the period that follows a deployment tends to be very busy and physically demanding as the unit secures and establishes an area of operations. Under these circumstances, unit pride and high-spiritedness may lead soldiers who are not yet fully acclimatized to adopt activity levels that produce needless heat casualties, sometimes in an effort to impress others with their prowess and strength. Unit leaders may need to be reminded that troops will not be able to perform at their optimum level until full acclimatization takes place, and that taking heat casualties probably does very little to enhance a unit's reputation for ferocity and toughness.

Water Consumption Practices. The days preceding a deployment to a hot weather environment are an excellent time to review water consumption practices, particularly if this is done concurrently with acclimatization training. This training should include, first and foremost, a strong emphasis on monitored intake of water. The amount of water consumed should be at least enough to make the urine colorless and may range from 4 to 14 liters per day for active troops.[14–17] Troops should be reminded that thirst is not a reliable indicator of dehydration, as exercising soldiers who are allowed to drink ad libitum will typically not drink enough to replace a significant portion of their water deficit.[18–22] Accordingly, troops should use the "buddy" system to ensure that sufficient quantities of water are being ingested. Forced overhydration provides no extra advantage in the heat,[23] however, and has resulted in cases of life-threatening and even fatal hyponatremia.[24] Accordingly, the US Army's current water replacement guidelines for hot weather training[17] state that hourly intake should not exceed 1.5 qt (1.4 L), and that daily intake should not exceed 12 qt (11.4 L). Soldiers must also be trained to understand that water consumption alone may not be enough to prevent heat injury, and that other protective measures should be used as well (see below).

In addition to ongoing water intake, prehydration with 300 to 500 mL of water prior to engaging in the day's activities can be useful to replenish any underlying deficits.[14,25,26] It should be made clear to the troops that this is a replacement of accumulated deficits, as the human body cannot "store up" water for future use.

Other Important Protective Measures. Whereas troops generally recognize the importance of water consumption as a countermeasure to heat injury, the importance of scheduled work/rest cycles is often underestimated. Troops and their leaders need to incorporate planned rest periods into their training and operations whenever circumstances permit. Tables of recommended work times under various heat conditions are found in a 1999 article by S. J. Montain and colleagues, "Fluid Replacement Recommendations for Training in Hot Weather,"[17] and in Chapter 3, Physical Exercise in Hot Climates: Physiology, Performance, and Biomedical Issues, of this textbook. Although the tables offer useful guidelines, they suffer from several important limitations:

1. The tables are inherently conservative, as they are designed to protect most soldiers from heat injury and cannot therefore account for individual variations in tolerance to heat and degree of acclimatization.
2. They are based on mathematical models and reasonable inferences made from controlled laboratory conditions,[17,27–31] not from data obtained from in the field, where conditions may be more or less strenuous than in the laboratory and where levels of activity may vary considerably during the work cycle.
3. The models used to construct these tables may be less accurate at extremes of temperature and humidity.

Accordingly, these tables should be used as a reference point for planning and training but should not replace common sense and close monitoring of the troops for signs of heat injury once they are deployed.

Operations Planning

A detailed description of the process by which

the battalion staff develops a plan is beyond the scope of this chapter, but it can be summarized as follows. First, on receiving a mission from a higher headquarters, the commander will develop a general concept of what the unit is expected to accomplish and how (the Commander's Intent). The battalion staff will then develop a number of different possible courses of action, each of which must be analyzed in detail to determine the manpower, combat support, and logistical requirements needed. A final recommendation is made to the commander, who has the option to accept it, modify it, or adopt a different course of action. The course of action chosen is then refined further to produce the battalion's Operational Order.

To have an impact on the final plan, the MO should strive to develop a close working relationship with the rest of the battalion staff so that he knows and understands well each of the proposed courses of action. The MO should help analyze each proposal to determine its likely medical implications for the troops. It is appropriate at this stage to recommend changes that would enhance both the welfare of the troops and the medical platoon's ability to care for them. Some factors to consider when evaluating a proposed course of action in a hot weather environment are found in Exhibit 9-4. This list is by no means comprehensive, but it should serve as a guideline to the MO who must advise a combat unit's staff.

Once a recommended course of action has been chosen and a tentative plan developed, the MO may be asked to brief the commander on the medical implications of the proposed and recommended courses of action. It is important to remember that medical implications of an operation are only one of many factors the commander weighs when deciding on a final course of action. Also, it is the commander, not the MO, who is ultimately responsible for the welfare of the troops. MOs must therefore master the art of advising without appearing to be second-guessing or undermining the commander's authority.

EXHIBIT 9-4

PLANNING FACTORS TO CONSIDER IN A HOT WEATHER ENVIRONMENT

- Mission, enemy, troops, terrain, time
- Water availability:
  - Sources of water
  - Water supply lines
- Water requirements, based on:
  - Weather and other environmental conditions
  - Intensity, duration, and punctuation of proposed activities
  - MOPP level and other protective clothing
  - Likely enemy courses of action
- Are work/rest cycles possible? If so, are they planned for?
- Predicted numbers and types of casualties (national and foreign)
- Location and capabilities of medical support assets
- Primary and alternate means and routes of evacuation
- Primary and alternate means of resupply
- Communications
- Chain of command
- Special medical requirements (equipment, supplies, drugs)
- Management of prisoners of war

MOPP: mission-oriented protective posture

Logistical Issues

MOs should be familiar with how hot weather affects some of the unit's key logistical requirements. For example, the MO should know how much water a soldier needs to survive in hot weather, and be able to provide this information to the S-4 officer on request. MOs may also be asked about the advisability of certain practices that have an effect on the unit's supply requirements (eg, Is salt supplementation necessary? Should caffeinated beverages be forbidden?). Finally, although a professional medical logistician (the medical service corps officer) is assigned to every infantry medical platoon, the MO must be able to articulate clearly the special medical supply requirements that the platoon will have during a hot weather deployment. Some of the common logistical issues that arise during deployment to hot weather (water, caffeine, IV solutions, supplemental salt, protection from the sun and heat, the effects of heat on medical equipment and supplies, and latrines) are discussed below.

Water

Water is always a key and limiting factor, and is even more so than usual in hot environments. Depending on the environmental conditions, the work load, and the uniform and protective clothing worn, it has been estimated that individuals will need between 2.5 and 14 liters of potable water per day when the mean ambient temperature is above 27°C (81°F).[14–17] The lower amount (2.5 L/d) is appropriate only for troops at rest in weather cooler than 35°C (95°F); with even mild work, each soldier should be supplied with at least 3 to 4 liters of water per day. Furthermore, during brief periods of intense activity, water requirements may increase to as much as 1 to 1.5 liters per *hour*.[2,14,25,26] Finally, we must consider the effect of acclimatization. As individuals acclimatize, their sweat rate with activity tends to increase,[13,22] as does their average water intake.[22] Because the effect of even a moderate increase in water intake per individual is multiplied by dozens of troops, a unit's water requirements may increase substantially during the first 2 weeks of deployment to a hot environment.

Whenever feasible, additional water rations should be provided for personal hygiene (ideally enough to allow handwashing and a daily sponge bath). The medical and mess sections of a battalion have special needs that logisticians must plan for, such as handwashing stations, food washing, wound cleansing, equipment cleaning, and the cooling and oral rehydration of heat casualties. Guidelines for estimating water supply requirements in a theater of operations can be found in Field Manual 10-52.[32]

Procurement of water does little good if distribution methods are ineffective. Planning to equip each vehicle (medical and nonmedical) with several 5-gal containers of potable water is an easy way to facilitate ongoing resupply efforts, and, in addition, gives each vehicle at least an inherent minimal heat and dehydration treatment capability.

Lyster bags (standard US Army equipment) may enhance the palatability of drinking water and hence its consumption.[2] These containers use an evaporative process to cool the contents down to the prevailing wet bulb temperature. Some water is lost in this process, however. Other strategies to enhance palatability include providing soldiers with citrus flavoring and sweetening for their water[14]; the beverage base powder contained in the prepackaged Meals, Ready-to-Eat (MREs) is an example of this. It may be counterproductive to provide carbonated beverages, however, as these tend to produce a sensation of stomach fullness, which may inhibit drinking. Furthermore, carbonic acid can itself stimulate both diuresis and sweating.[14]

It has long been known that in the desert, much of an individual's total daily water intake occurs during meals.[33] This is presumably true in other hot weather environments as well. It is therefore very important to make sure that an ample supply of water is available to troops at meal times. Providing extra water at meals also allows troops to refill their canteens before resuming field operations.

Caffeine

The MO may be asked whether caffeine consumption should be banned during a deployment to hot weather, owing to caffeine's reputation for having diuretic properties. While it is true that any degree of dehydration is undesirable in hot environments,[34] caffeine is at most a mild diuretic, and unless water is in very short supply, it should be possible to compensate for any additional urinary losses by increasing water consumption. Caffeine may also have desirable ergogenic effects on physical performance.[35,36] Furthermore, abrupt cessation of caffeine intake by even moderate users can produce a withdrawal syndrome[37] that might adversely affect performance. Thus, until outcome data are available that specifically address this issue, a ban on moderate caffeine consumption during hot weather operations does not appear to be necessary (and may even be detrimental), unless the unit's ability to meet a small increase in water requirements is in question.

Intravenous Solutions

Extra IV fluids and infusion sets should be planned for and procured; both the medics and the combat lifesavers in the unit must be supplied. The unit's total on-hand stock of IV fluid can be greatly enhanced without unduly encumbering the aid station section, the medics, or the combat lifesavers by simply procuring and distributing extra IV bags to individual line soldiers in the unit. Soldiers who are asked to carry IV bags should be given basic instructions on how to care for them. For example, they should be told that the IV bags should not be removed from their outer wrapping until just before they are used, and that care should be taken to avoid accidental puncturing of the bag. However, even under the best of circumstances, saline for IV infusion tends to be a limited resource. It is therefore prudent to plan to attempt oral rehydration of heat casualties whenever feasible, reserving the IV saline for the emergency cases and for those who cannot take liquids by mouth. Field-expedient oral rehydration solutions can be made by adding one

salt packet and one beverage base powder from a standard MRE to a 1-qt canteen of water[2]; medics and combat lifesavers should be trained to prepare and use this solution.

Supplemental Salt

Although unacclimatized soldiers will lose a significant amount of sodium and chloride (as well as some potassium and magnesium) in their sweat, supplemental salt administration is generally not necessary. Most diets (and US military rations in particular) will supply more than enough to meet a soldier's needs, even in a hot weather environment.[13,38] Furthermore, supplemental salt administration may be deleterious, as it may (for example) force obligatory urinary losses of water to excrete the excess sodium load.[38]

Protection From the Sun and Heat

A unit deploying to a hot environment will need to procure an ample supply of sunscreen, ultraviolet ray–blocking sunglasses and lip ointment, bandanas, insect repellent, mosquito netting, and skin and foot powder. Broad-brimmed hats ("ranger," "jungle," or "boonie" caps) can provide better protection from the sun than standard battle dress uniform caps[39] and should be authorized and obtained if possible.

Shelter from the elements is an important aspect of the prevention and treatment of heat injury. Shelters that are entrenched can be cooler than those that are erected on the surface, as ground temperature drops fairly quickly below the surface.[2] For static facilities that are likely to treat a large number of heat casualties, air-conditioning units can be useful. However, even an infantry BAS can be equipped with a fan, provided a generator is available. Furthermore, many ground ambulances are equipped with air conditioning in the patient bay. These units should be checked and be working properly before deployment; if they are not working, repair should be a high priority.

Effects of Heat on Medical Equipment and Supplies

Medical equipment and supplies can be highly vulnerable to the conditions faced in a hot weather environment. For example, sand, dirt, and dust can clog filters and tubing, and can damage or disable medical equipment. Bacteria and fungi may proliferate in hot, humid climates and can rapidly degrade untreated fabric and rubber components. Critical equipment should therefore be inspected and cleaned frequently to identify and correct problems before they become disabling.

Drug shelf life may be significantly reduced, especially once a container has been opened; it may therefore be preferable to obtain some drugs in blister packs instead of bottles. Medics must also understand that dressings and other items packaged in paper sleeves are no longer considered sterile if the sleeve gets wet. A torrential rain can soak an aid bag or an incompletely sealed medical supply box and ruin much of its contents. A practice of wrapping individual moisture-sensitive items in airtight plastic sandwich bags prior to deployment can help salvage supplies under even the worst conditions.

Latrines

Latrines should be procured or built for unit elements that are expected to remain in relatively static positions. Latrines are obviously important for sanitary reasons. They are also important because when clean, sanitary latrines are not available, some troops may drink less (to void less).

In the desert, care should be taken to avoid placing latrines in either a flash-flood zone or in a location that could contaminate the limited water supply.

TWELVE TIPS FOR MEDICAL OFFICERS

1. Always Have a Functioning Evacuation Plan.

The best MO in the world will be of little use to the troops if there is no way to get the casualties to him. Furthermore, being able to get a casualty who needs advanced care to a facility that can provide it in a timely manner often makes the difference between life and death. A functioning medical evacuation plan is always a top priority for the medical platoon. In a hot weather environment, this should include the means to cool and hydrate a patient during transport.

2. Establish a Medical Treatment Capability as Early as Possible.

A unit deploying to a hot weather environment should be prepared to suffer some early heat casualties, when water supplies are still tenuous and troops are not yet fully acclimatized. Enemy attempts to disrupt deployment can also produce early casualties. It is therefore a top priority for a medical platoon to establish capabilities to treat casualties with trauma or heat injury as early as possible. This can be

EXHIBIT 9-5

THE CASE OF THE MISSING CANTEENS

Two National Guard infantry battalions conducted their annual training in the middle of a record-setting heat wave at Fort A. P. Hill, Virginia. For the sake of efficiency, the medical assets of both battalions were pooled into an enhanced aid station under the direction of a single medical officer. Because of the short time available for training, it was not possible to preacclimatize the troops before deploying into weather hotter than 100°F. Within 48 hours of arrival at the training site, the aid station had treated about a dozen heat casualties, several of whom also had signs of dehydration. Accordingly, the medical officer made a point of attending the evening's command briefing. During the briefing he noticed that, aside from himself and the chaplain, none of his fellow officers were carrying canteens of water, despite evening temperatures that remained in the high 90s (°F). He pointed this out to the group, in front of the commander, and posed the question: What message does this send to the troops about how seriously their officers take water discipline? The importance of being a role model for water discipline was vigorously reaffirmed by the commander; within 24 hours, the casualty rate had diminished to about one per day.

accomplished by designating a ground ambulance as a makeshift battalion aid station while the BAS tent is being erected. Distributing extra supplies to medics prior to deployment also may buy a little time.

3. Keep Track of the Number, Type, and Disposition of Heat Casualties.

Heat casualties tend to occur in clusters[3–5,12]; therefore, it is wise to view the first heat casualty as a herald of others to come. A cluster of casualties coming from the same unit should prompt the MO to seek a constructive discussion with the leadership of that unit. The focus of this discussion should not be to ascribe blame but rather to identify remediable causes of heat injuries and to find countermeasures that are tailored to the reality the unit is facing.

The adjutant (S-1) will need a daily tally of the number of casualties incurred and a rough estimate of how soon they will be able to return to duty (if at all). Furthermore, the numbers and types of casualties suffered can provide the intelligence officer (S-2) important insights into enemy activities and capabilities. It is therefore important for the medical platoon to keep a systematic count of these casualties and to provide a daily summary report to the battalion staff. Finally, keeping a log of soldiers who were evacuated to a supporting medical facility and the facility to which they were evacuated can help the process of returning them to the unit once they are treated.

4. Attend the Daily Staff Meeting and Commander's Briefing.

Participation in staff and command briefings serves several purposes:

- these gatherings allow the MO to bring key information to the attention of the commander and the staff,
- they are efficient places to get first-hand information on the unit's situation (including the weather forecast) and the latest updates to the operation, and
- they provide an excellent forum for giving general medical recommendations to the unit.

At these meetings, the MO should be prepared, at a minimum, to brief the commander and the other staff members on the numbers and types of casualties treated, any identified clusters of cases (including those that are environmentally caused), and any recommended countermeasures. The MO should be prepared to answer questions in direct, nontechnical language. Brevity and clarity are essential to effective communication in these meetings and are greatly appreciated by the nonmedical personnel who must attend them.

Exhibit 9-5 illustrates how an MO used the commander's daily briefing to improve water discipline in the unit.

5. Monitor the Weather.

Knowing the weather forecast allows the MO to tailor the recommendations for addressing the environmental threat. Methods of obtaining and disseminating weather information should be planned for prior to deployment (as discussed above) and implemented once the unit is in the field.

6. Never Pass Up an Opportunity to Resupply a Unit.

We should view every medical *evacuation* mission

as an opportunity to be a medical *resupply* mission. Every vehicle in the battalion (including ground ambulances) should carry several 5-gal containers filled with potable water. This practice will afford the unit an impromptu water resupply capability that can be especially important for elements in isolated locations. Furthermore, ambulances should maintain a small surplus of items required by the frontline medics. In hot weather environments, this often means extra bags of IV fluids.

MOs must never forget that if a ground ambulance is to be used for any nonmedical purpose, the medical distinguishing characteristics (in the US Army, the red crosses) must be obscured.

7. Monitor Your Own Soldiers for Signs of Environmental Illness and Combat Fatigue.

Medical units are expected to deliver services and support around the clock and are thus highly vulnerable to the stresses of continuous operations. Furthermore, the ethic of self-sacrifice and "patients first" that characterizes medical personnel can result in self-neglect and unnecessary environmental injury. The physical demands of being a medic can be quite high (eg, when transporting casualties to and from an evacuation vehicle). It is therefore incumbent upon leaders in medical units to ensure that their own soldiers follow the heat injury prevention guidelines that are given to the rest of the unit.

Combat fatigue is an ever-present threat to medical units. In a modern battlefield, no unit is immune to attack by indirect fire. Even when spared attack, medical units often face, daily, the grisly human consequences of war: injury, illness, dismemberment, and death. The environmental threat only compounds the inherent psychological stresses of the occupation. Heat can diminish the capacity to perform physically demanding tasks[40–46] as well as some mental tasks,[47] and is often perceived as an especially unrelenting environmental condition, thus adding to any underlying frustrations. Countermeasures to combat fatigue are discussed in Volume 3 of *Medical Aspects of Harsh Environments* and elsewhere,[48] and must be tailored to the realities of the local situation. For example, in a hot environment, we must make extra efforts to find comfortable sleeping arrangements for soldiers who will be working night shifts and must therefore sleep during the hottest part of the day. Identifying a relatively cool and safe location for rest periods can also be a boost to morale. Of course, we must never pass up an opportunity to provide the troops appropriate rest and recreation, especially if it involves a chance to obtain respite from the heat. Finally, opportunities to obtain Class VI (personal need) items can provide a significant boost to troop morale.

8. Dead Medics Cannot Save Lives.

Medical personnel who do not know the fundamentals of battlefield survival quickly become a burden to those around them. Medical units must therefore pay close attention to the realities of operations in a combat zone. All personnel must maintain physical fitness and proficiency in basic soldier skills, including how to move, shoot, and communicate; how to set up and maintain a defensive perimeter; how to use mission-oriented protective posture (MOPP) gear at all its levels, and how to cope with the environmental threat.

Casualties themselves can be a serious and even lethal threat to medical personnel. For example, a single, armed grenade that a confused casualty is clutching but happens to drop while being loaded into an ambulance can result in unnecessary tragedy and loss of a limited asset. It is important to make sure that casualties are relieved of their weapons before loading them into an ambulance or bringing them into a medical treatment facility. Do not take at face value the word of a sick or injured soldier that all weapons are secured; search him. A search policy is especially important in areas where terrorist and guerrilla threats are high, as unconscious casualties may be deliberately booby-trapped in an effort to kill and injure those who may try to help them.

9. Always Be on the Lookout for Opportunities to Improve the Area of Operations.

Every day that a unit is not on the move should be viewed as an opportunity to improve the surrounding area of operations. These improvements include the quality and quantity of the water supply, field sanitation and hygiene, cooling and shelter, defensibility of the area of operations, and better access to routes of evacuation and helicopter landing zones.

10. Be a Good Role Model.

Like it or not, an MO will probably be viewed as a role model by the troops. As a result, our actions and attitudes, as mimicked by others, may have unanticipated effects. In addition, behavior that is viewed as capricious or arbitrary can rapidly sap the morale of the unit. We must therefore be care-

ful to maintain high personal standards of conduct and ethics, to be just, and to try to set the example that we wish others to follow. This is especially important when members of the unit are under environmentally induced stresses such as those imposed by hot weather.

11. Use Your Noncommissioned Officers Wisely.

The advice offered here is often second nature to a good noncommissioned officer (NCO). Furthermore, NCOs are expected to be adept at implementing these recommendations. The MO who makes a point of identifying and developing strong NCOs in peacetime will be richly rewarded with individuals he can trust and to whom he can confidently delegate responsibility in wartime. Never be ashamed to learn from an NCO, especially one who has experienced battle firsthand.

12. Keep a Positive Mental Attitude.

Always!

SUMMARY

Hot weather poses special challenges to the MO who must support a combat arms unit. However, the elements of success are similar to those encountered in any military operation: a strong peacetime training program, careful planning, a good working relationship with the battalion commander and staff, development of and trust in the NCOs, and, above all, a very healthy respect for the threat that hot weather poses.

Although the advice given here may be helpful to the MO deploying to a hot weather environment, the ultimate test of success is whether the unit accomplishes its mission with a minimum of casualties. No amount of writing or reading can replace the need for training, resourcefulness, wisdom, and flexibility on the part of the medical personnel who must support troops in the field.

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MEDICAL ASPECTS OF HARSH ENVIRONMENTS
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Olin Dows *Medics Moving In Near Bastogne* 1945

The events depicted here are consistent with more than one scenario. The watercolor could show a medical unit retreating into the perimeter of the surrounded 101st Airborne Division at Bastogne, Belgium, during the German siege in the third week of December 1944. More likely, it shows a medical unit accompanying Patton's Third Army as it broke through the German ring on the day after Christmas 1944. The medical unit would have assisted with the treatment and evacuation of some 1,500 casualties who had been trapped at Bastogne during the Battle of the Bulge. The official history of the Bastogne campaign contains the following passage, which nicely sums up some of the medical problems of warfare in cold weather:

> The cold caused even slightly injured men to go into shock so that litter squads had to carry extra blankets, and aid stations had to administer larger than usual amounts of plasma.[1(p397)]

Quotation: (1) Cosmas GA, Cowdrey AE. *Medical Service in the European Theater of Operations*. Washington, DC: US Army Center of Military History; 1992: 397. Watercolor: Reproduced courtesy of Army Art Collection, US Army Center of Military History, Washington, DC.

Chapter 10

COLD, CASUALTIES, AND CONQUESTS: THE EFFECTS OF COLD ON WARFARE

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INTRODUCTION

On a bitter, cold night during the Korean War, a US Marine sentry, huddling in a ditch alongside a road near the Chosin Reservoir, peered nervously into the darkness. In the stillness he heard a rhythmical "click-clack, click-clack," slowly becoming louder and louder. Not knowing what the sound could be, he waited, his finger on the trigger. The noise came closer until, around the corner, staggered a dazed Chinese soldier walking on bare feet, frozen so hard that they clattered on the road with each step.[1]

Of all the factors that influence the outcome of wars and battles, the natural environment has been one of the greatest forces for disaster. Cold environments have decided the fate of armies, often causing the deaths of hundreds of thousands of soldiers, confounding plans, and turning victory into defeat. Knowledge of the campaigns in which weather and winter have played a critical part should, rationally, help to avoid similar catastrophes in the future; but even a superficial study of history shows that these lessons have been hard to learn. Faulty command decisions,[2] inadequate logistical preparation,[3] failure to learn from the past, and unwarranted optimism[4] have, time and time again, led to disaster.

The lessons learned have been both military and medical. As casualties have decimated armies, doctors have been stimulated to seek a better understanding of the pathology of cold injuries, and this knowledge has been translated into better management.

Although the mud of spring and fall has had a greater effect on mechanized warfare than the frozen terrain of winter,[5] countless wars have been started in summer and brought to a slower pace during winter. The best-laid plans often came to naught if armies were caught in the grip of winter,[6] ill-equipped and unable to obtain food, far from a home base, and defeated. In the 20th century, better equipment and more sophisticated logistical and supply systems have enabled armies to alleviate the effects of the environment, whether hot or cold. On occasion, armies experienced in winter warfare have used the environment to their advantage. In the battle for Stalingrad, in World War II, the Russians made their final attack during bitter cold,[2] knowing that they were attacking an enemy demoralized by cold and starvation and lacking the resources to resist, despite their courageous defense.

ANCIENT HISTORY TO WORLD WAR I

Xenophon and Hannibal

The greatest epidemics of cold injury have occurred during wars. Xenophon (434–355 BCE) was a young Athenian officer in an army of mixed Greek mercenaries led by Cyrus on a campaign into Asia Minor. At the Battle of Cunaxa (401 BCE), not far from Babylon, the Greeks won but Cyrus was killed.

EXHIBIT 10-1

GLOSSARY

Frostbite, also called Freezing Cold Injury (FCI)	A cold injury involving freezing of tissues. Damage may be temporary or permanent. Gangrene and loss of digits or limbs may result. Long-term sequelae, including pain on exposure to cold, are common.
Nonfreezing Cold Injury (NFCI)	Includes trench foot, immersion foot, sea-boot foot (an injury that results from multiday exposure of limbs [usually the lower limbs] to a cold—but nonfreezing—wet environment. The primary injury is to nerves, but damage to blood vessels and other tissues also occurs. Gangrene may result. Long-term, painful sequelae are common.
Warm Water Immersion Foot (WWIF)	An injury caused by prolonged exposure of feet and legs to a warm, wet environment. The condition responds to drying the affected parts, and long-term sequelae are uncommon.

Xenophon was elected one of the new leaders and became the main inspiration and driving force, leading 10,000 men on a 1,000-mile retreat through the mountains of what is now Armenia, in the depths of the winter of 401/400 BCE.[7] Only 4,000 soldiers survived; most of the others died from exposure and frostbite (Exhibit 10-1).

Hannibal, the famous Carthaginian general, planned to invade northern Italy in 218 BCE by leading an army of 38,000 infantry, 9,000 cavalry, and 80 elephants up the valley of the Rhone, across the Alps, and into the valley of the Po. When he made the passage of the Alps in October 218 BCE, heavy snow had already fallen in the mountain valleys.[8] The altitude, bitter cold, and fierce mountain tribes extracted a heavy toll. Only 19,000 men survived, half-starved and frozen. Surprisingly, some of the elephants also survived to play an important part in victorious battles against the Romans.

Charles XII of Sweden

In 1697 a charismatic young man, aged only 14 years, ascended the throne of Sweden.[6] Charles XII soon won the hearts of his people and, supported by a well-trained army, won a string of victories in the Great Northern War (1700–1721), which only came to an end when he was defeated by the Russians at the battle of Poltava (July 1709). The campaign leading up to that defeat was conducted in one of the coldest winters ever experienced in Europe, and by spring only 20,000 men were fit for action to face 50,000 Russian troops led by Peter the Great. Only 1,500 Swedish troops survived the battle. Charles XII was not the first, and certainly not the last, conqueror to be enticed into the vastness of the Russian steppes, there to be defeated. He fled to the Ottoman Empire, present-day Turkey, where he served in the Ottoman army before returning to Sweden in 1714.

A few years later Charles decided to invade Norway.[9] His first attempt was a failure, but in the autumn of 1718 he returned to battle again, mounting a two-pronged attack on the Norwegians: a southern thrust against Oslo, and a northern attack across the mountains to Trondheim. Both attacks reached their objectives. In the north, the Swedish army, which was composed largely of Finns, surrounded Trondheim without much difficulty because the Norwegians, being wise locals, did not want to become involved in a pitched battle as winter descended on the bleak, treeless ridges.

King Charles, who was renowned for leading from the front, commanded the southern army. While inspecting a forward position on the evening of 11 December 1718, the king was hit in the temple—some reports claimed by a silver button—and died almost instantly. The news of his death spread rapidly and caused consternation in the army. Some of the generals in the south immediately abandoned their troops and fled for home. The news did not reach the northern army until Christmas Eve, when General Carl Gustaf Armfeldt, the commanding general of the northern army, received an order to retreat and return to Sweden.

A severe storm hit on December 27th, a harbinger of what was to come. General Armfeldt struck his headquarters on 4 January 1719 and started up the valley of the Tydal toward the border. The Norwegians used guerilla tactics to attack the retreating army, constantly picking at the edges of the columns. On 13 January, when the main body of the retreating army was crossing the highest ridge, another storm struck and lasted 3 days. By the time the blizzard had abated, 3,700 men (half of Armfeldt's army) had died of hypothermia and frostbite, and another 600 were permanently crippled by the cold.

Contemporary accounts of the disaster told of soldiers who were found lying in the snow with few clothes, and it was thought that the survivors had stripped their freezing comrades to keep themselves warm. A more likely explanation, in light of our 20th-century experience, is that this was an early account of "paradoxical disrobing," that strange phenomenon that makes hypothermic victims take off their clothes because of a feeling of intense warmth.[10] One of the casualties, a young medical officer, froze his feet badly but refused to allow them to be amputated. He recovered and later became a general in the Swedish army—still with two feet. His insistence on waiting saved his feet, showing the advantage of conservative treatment.

This defeat, brought about more by a blizzard than by an enemy, ended Swedish military dominance of the Nordic countries.

General George Washington and Valley Forge

In the annals of American military history few stories equal that of the winter of 1777/78, when Washington's army bunkered down in Valley Forge. Eleven thousand raggle-taggle, retreating men marched into the valley on 18 December 1777.[11] Two thousand were without shoes or boots. During those winter months the hardships endured were severe, although the winter was relatively mild. At times, snow lay on the ground and the temperatures

dropped below freezing. Six months later, after long, hard training, a tough, disciplined force marched out to defeat the British.

Accurate records were not kept of the number of cold injuries, but Lafayette wrote:

> Feet and legs froze until they became black and it was often necessary to amputate them.[12(p45)]

There were many other medical problems. Diarrhea was rampant because of improper sanitation, although the need for latrines and their correct use was well recognized. Smallpox was a threat, and the inoculation campaign that protected thousands of men must have been one of the first large-scale attempts to prevent the disease. The vaccine used was primitive and obtained from men with mild cases. Amazingly, of the 4,000 men vaccinated, only 10 died from the immunization.[12]

Exposure, overcrowding, poor food, scabies, typhoid, and typhus haunted the Continental Army during that fateful winter. Had the army been moving and fighting rearguard actions for those months, the casualties would have been many times greater. As it was, the lack of fighting and the chance to stay in one place and reorganize reduced the casualties and laid the foundation for future victory.

Napoleon in Russia

A century after the army of Charles XII came to grief in Russia, the disaster was repeated on a grander scale. In 1812, Napoleon invaded Russia to suppress Tsar Alexander and consolidate his hold over Europe.[4]

Napoleon studied the problems he might encounter in minute detail and repeatedly read the accounts of the campaign of Charles XII of Sweden, vowing that he would not make the same mistakes.[4] The planners of Napoleon's Grande Armée understood that they would not be able to obtain supplies from the countryside and, therefore, made preparations to set up huge depots along the route and carry many of the supplies in trains of wagons.[4(p757)] The invading army was organized into three lines amounting to 612,000 troops, of whom fewer than half were French. Twenty-six transport battalions with nearly 25,000 wagons pulled by oxen and horses trailed the army, accompanied by herds of cattle to be slaughtered for meat. There were 200,000 animals, including 30,000 horses for the artillery and 80,000 for the cavalry, to be fed and cared for.[4(p758)] Because of the need to feed the animals, the invasion had to be delayed until June, when the plains would be lush with summer grasses.

Despite the massive preparations, one factor—apart from the extremes of weather—had not been considered and could not be controlled. Napoleon, although only 42 years old, was no longer at the height of his military powers. His health was not good and it deteriorated during the campaign.[13] He could not possibly maintain control over a vast multinational army, spread across hundreds of miles, when the fastest means of communication was determined by the speed of a horse. Where Napoleon foresaw only glory, however, others saw disaster. Carl von Clausewitz—who was himself serving in the Russian army in 1812—had forecast that if Napoleon were ever to invade Russia, he would be destroyed.[14]

The River Niemen in Poland was the starting line for the invasion, but no arrangements were made to feed the troops before they reached this point, although some of them came from as far away as Austria and Spain. The soldiers had to live off the country. As a result, many arrived at the Niemen already malnourished and suffering from diseases that included diarrhea, diphtheria, and typhus. In addition, insufficient warm clothing had been ordered. Napoleon believed that he would defeat the Russians quickly in one decisive battle and, unrealistically, hoped that the campaign would be over in 20 days. The Russians had other ideas, and three times they slipped away in the night before a major battle, drawing Napoleon farther and farther from his bases into the emptiness of the plains and forests. The Russians held fast at Borodino in a battle that cost them 44,000 casualties. In the same battle Napoleon lost 30,000 troops, including 47 generals.[4]

The route to Moscow was haunted by frustration and the loss of thousands of men. Two thirds of the army were lost during the advance, due to death from wounds and illnesses, desertion, and capture. The invasion started in June, and Napoleon entered the Kremlin in September. A month later he decided to retreat; by this time, the populace had fled, the city had been reduced to ashes, the Tsar had refused to negotiate a peace, and winter was approaching. The retreat, Napoleon's first, became a frozen nightmare. It was, ultimately, the start of his downfall.

By the time the gates of Moscow were reached, the Grande Armée had been reduced to 110,000 men, and this force started back to France on 19 October 1812. As the troops left Moscow, in a train of 40,000 wagons loaded with loot and supplies, rain

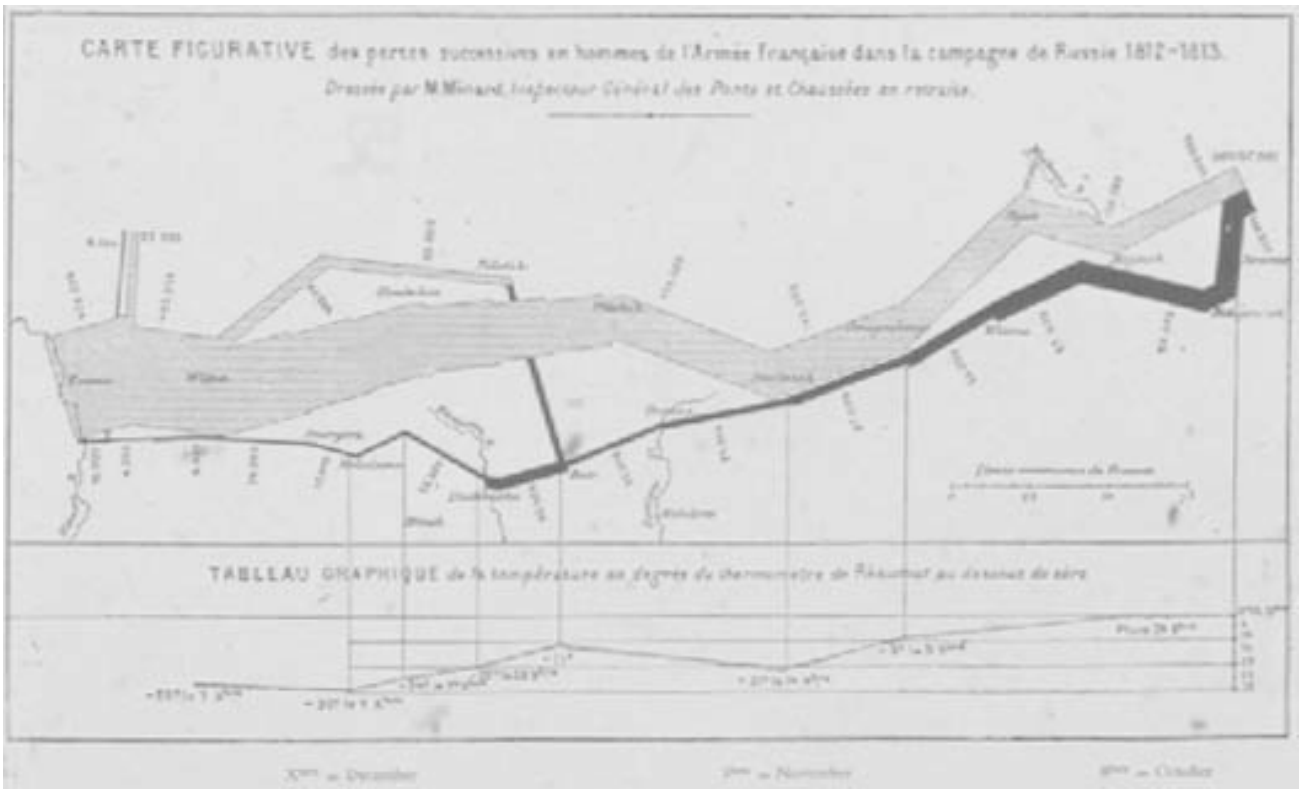

Fig. 10-1. This diagram of Napoleon's Russian campaign in 1812–1813, drawn in 1861 by M. Minard, retired General Inspector of Roads and Bridges for the French Army, has been praised as "the best statistical graphic ever drawn."[1(p40)] Reading from left to right, the shaded area indicates the diminishing size of Napoleon's Grande Armée during its advance to Moscow. Reading from right to left, the dark line shows the size of the Grande Armée as it diminishes further during its retreat from Moscow. The narrower the line, the smaller the army. Interested readers will note that approximately 75% of the Grande Armée had been lost before the retreat from Moscow; the remainder was destroyed during the fall of 1812. The representations of the size of the Grande Armée are superimposed on a map with the names of places and battles. The lower part of the diagram shows a graphical representation of the temperature in degrees Réamur (a temperature scale similar to Celsius, which superseded it) at different points during the retreat. Scale in leagues; 1 league equals approximately 2 mi. (1) Tufte ER. *The Visual Display of Quantitative Information.* Cheshire, Conn: Graphics Press; 1983: 40. Reproduced with permission from Tufte ER. *The Visual Display of Quantitative Information.* Cheshire, Conn: Graphics Press; 1983: 41.

was falling and the temperature was just above freezing. A month later, by the time the army reached Vyazma, only 160 miles from Moscow, the force was down to 50,000 men (Figure 10-1).

The first snow storm hit on 6 November[13] and was followed by intermittent snow, rain, and thaw until early December, when the weather became bitterly cold. During the next 6 weeks the temperature fell to nearly –40°C, and the size of the Grande Armée was further depleted to 12,000 men (Figure 10-2). During one battle, the crossing of the ice-filled Berezina River, the French army had 30,000 casualties, many of whom drowned in the river, having been swept off the hastily built bridges by a flood of retreating men and horses. When the army finally crossed the River Niemen into Poland, fewer than 10,000 effective soldiers remained.[4]

More than 100,000 men had been killed in action, 200,000 died from other causes, 50,000 were left sick in hospitals, 50,000 deserted, and 100,000 became prisoners of war. The exact number who died from cold can never be known, but in addition to those who died from cold alone, thousands of wounded who might have lived must have died from the combined effects of their wounds and the cold. Russian casualties numbered at least 250,000, and must also have included many who died from cold and exposure.

Count Phillipe-Paul de Ségur, a brigadier and aide to Napoleon, who wrote a definitive history of the campaign that was both a literary and military

Fig. 10-2. *Retreat From Moscow;* hand-colored contemporary aquatint after Johann Adam Klein; circa 1815; originally published in Vienna by Artaria. Napoleon, huddled in a cape and riding his horse, marches alongside his defeated troops during the retreat from Moscow. The ground is littered with frozen bodies and abandoned artillery. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island.

masterpiece,[13] described the first snow as it fell on the retreating army:

> Russian winter in this new guise attacked them on all sides; it cut through their thin uniforms and worn shoes, their wet clothing froze on them, and this icy shroud molded their bodies and stiffened their limbs. The sharp wind made them gasp for breath, and froze the moisture from their mouths and nostrils into icicles on their beards.[13(p170)]

The depth of the final defeat and degradation of the Grande Armée can hardly be described. According to de Ségur, one division that started from Moscow with 12,000 men arrived in France with fewer than 400 in good health:

> Here were the same valleys down which had poured those three long columns of dragoons and heavy cavalry, three streams of steel and brass, flashing in the hot sunlight. But now, men, weapons, eagles, horses, sunlight, even the frontier river they had crossed in such ardor and hope - everything had disappeared. The Niemen was just a long mass of blocks of ice piled up and welded together by the breath of winter. In place of the three French bridges, brought fifteen hundred miles and erected with such daring speed, there was only one Russian bridge. Instead of the four hundred thousand companions who had fought so many successful battles with them, who had pushed so valiantly into Russia, they saw issuing from the white, ice-bound desert only one thousand foot soldiers and troopers still armed, nine cannon and twenty thousand beings clothed in rags, with bowed heads, dull eyes, ashy, cadaverous faces and long ice-stiffened beards. Some of them were fighting in silence for the right to cross the bridge which, despite their reduced number, was still too narrow to accommodate their precipitate flight. Others had rushed down the bank and were struggling across the river, crawling from one jagged cake of ice to another. And this was the Grand Army.[13(p280)]

Napoleon's Surgeon-General, the Baron Larrey, wrote graphically of the retreat and the cold injuries of the soldiers:

> Woe to the man benumbed with cold ... if he entered suddenly into a too warm room, or came too near to the great fire of a bivouac. The benumbed or frozen extremities ... were struck with gangrene, which manifested itself at the very instant, and developed itself with such rapidity that its progress

> was perceptible to the eye. Or else, the individual was suddenly suffocated by a kind of turgescence, which appeared to seize upon the pulmonary and cerebral systems: he perished as in asphyxia. Thus died the chief apothecary of the guard. … Weakened by cold and abstinence, a refuge was offered him in a very warm room of the hospital pharmacy, scarcely had he passed a few hours in this warm atmosphere, which was new to him, when his limbs, which he no longer felt, became bloated and swollen, and he expired soon afterwards in the arms of his son and one of his colleagues, without being able to utter a single word.[15(p83)]

The mechanism of death in these men can only be conjectured. They were in the final stages of exhaustion. They had probably received little food or drink, and what they drank may well have been polluted. Terror and exhaustion were their constant companions. Their feet were excruciatingly painful, afflicted with a combination of trench foot and frostbite. Severely depleted of fluids, and perhaps suffering from infections—for typhus was rife in the army—they exposed themselves to heat and vasodilated both centrally and peripherally. They placed their frozen feet too close to a strong source of heat, now recognized to be disastrous treatment.[16] The combination of a burn and frostbite guaranteed massive tissue loss and destruction. Massive extravascular extravasation further reduced their intravascular volume. They probably died from hypovolemic shock, peripheral and pulmonary edema, and, in many instances, infection.

Larrey not only described the condition of the men but also advocated treatment for frostbite that, because of the authority of his position and experience, would become the standard for the next hundred years. He believed vehemently that rapid warming was fatal and advised rubbing frozen limbs with snow. His explanation, although understandable, sounds strange to our ears:

> For it is well known that the effect of caloric on an organized part, which is almost deprived of life, is marked by an acceleration of fermentation and putrefaction.[15(p84)]

Larrey's contributions to military surgery and the handling of battle casualties, his astute observations on the pathogenesis of frostbite and his analytic approach to the understanding of trauma, make him a true surgical giant. He observed that cold weather, by itself, need not necessarily result in casualties. When the weather warmed up—and when the roads and battlefields turned to mud—cold injuries appeared. He described how the soldiers, after one battle in February, had no cold injuries although the temperatures at night dropped to –20°C. But when there was a sudden thaw with heavy rain, the temperature rising to 8°C to 10°C, soon many soldiers complained of

> vivid pains in the feet, congestion, heaviness, and unpleasant creepings in the extremities. ... All of those patients who had gone to the town or to the camp fires to warm themselves became the most mistreated.[15(p67)]

The description fits the early symptoms of trench foot, a problem that must have been very common (although it was not yet separated clinically from frostbite).

Larrey also recognized that the external appearance of gangrene in a frozen extremity does not conform to the deeper damage:

> One must not confuse the gangrene of the foot with the gangrene of the skin. Indeed it often happens that a more or less extended part of the skin is mortified without loss of life to the tendons, ligaments, vessels, deep nerves or the bones.[15(p74)]

Larrey was not expounding a new theory. Hippocrates had warned of the danger of heat:

> In some instances blisters arise as if from burning with fire, and they do not suffer from any of those unpleasant symptoms until they become heated.[17(p22)]

Crimean War

Trench foot has afflicted armies for centuries, although the association between prolonged exposure to wet cold and gangrene was not made until World War I.[18,19] (Trench foot, immersion foot, shelter foot, and sea-boot foot are all now classified as nonfreezing cold injuries [NFCIs], but for purposes of this historical overview, the earlier terminology will be used.)

During the Crimean War (1853–1856), prolonged exposure to the cold mud in which the combatants fought was ideal for the development of trench foot.[20] The British and French sustained thousands of cold injuries (Figure 10-3).[21] During the first winter of the war the British had 1,942 cold casualties out of an army of 50,000 soldiers. The doctors recognized, as had Baron Larrey, that injuries often appeared when the weather warmed and a thaw started. Then the frozen ground on which the troops could stand turned to icy mud into which they sank. The soldiers spent more than 24 hours in the

Fig. 10-3. *Before Sebastopol;* November 1854. *The Sentry,* an unsigned ink and watercolor drawing by Miss H. J. Wilkinson after one done by Captain Wilkinson of the 9th Foot, British army, shows a cold, wet sentry, the hood of his jacket wrapped around his neck, his hands in his armpits. He is standing in a mud puddle, and the bones of a carcass can be seen near his feet: a picture of misery and inattention to duty. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island.

trenches, often in mud and snow up to their knees. Their boots were inadequate and their clothing unsuited to cold weather. As would be expected, the infantry had many more casualties than the cavalry, but even the horses suffered from ill-treatment, the terrible cold, and mud:

> The horses had one handful of barley each day as their day's food and the same the next day. They were standing knee deep in mud, with bitter Crimean wind cutting their emaciated bodies. … An order had been issued that no horse was to be destroyed ... [so] ... they lay in the mud in their death agony for three days while no one dared to shoot them.[20(p262)]

By the second winter of the war the army had begun to learn its lessons (Figure 10-4). Supplies had improved, and commanders understood the connection between length of exposure to wet, cold conditions and the injuries. Soldiers did not have to spend such long periods in the line, and their

Fig. 10-4. *Huts and Warm Clothing for the Army;* original watercolor by William Simpson, 1855. Soldiers march through the frozen landscape of the Crimea. On the left, a man driving an ox before him is lucky enough to wear a sheepskin cover. In the background, a camp is being raised with a tent and a few wooden huts. The artist, Simpson, went to the Crimea in fall, 1854, and spent several months sketching the campaign for a series of lithographs published in London, England. This watercolor was the subject of one of the lithographs. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island.

experiences of the previous winter had taught them how to care for themselves. The case mortality death rate fell from 23.75% during the first winter to 1.3% in the second.[21] The decreasing mortality was not entirely due to a reduction in cold injuries. Cholera and typhus also extracted a high death toll, and a storm—which on 14 November 1855 had destroyed 30 transports carrying food and clothing for the first winter—had added to the high early mortality.[22] William Howard Russell, the correspondent for *The London Times,* reported such a grim picture of privation and inefficiency that the public uproar resulted in intensive efforts to improve the lot of the troops and to reorganize the medical facilities under Florence Nightingale.[20]

Wars of the Late 19th and Early 20th Centuries

During the American Civil War (1861–1865) there were more than 15,000 cold injuries, of which 1,075 were serious. Amputations were common, with a mortality rate of about 30%; many were for frostbite. Of 259 amputations that were carefully documented, 44 were for frostbite.[21]

During the next few decades the Franco–Prussian War (1870–1871), the Russo–Turkish War (1877–1878), the Sino–Japanese War (1895), the Russo–Japanese War (1904–1905) (Figure 10-5), and the Boer War (1899–1902) all extracted their tolls of thousands of cold injuries. Much was learned by the military in various countries about the treatment and prevention of cold injuries. The Japanese, according to a British observer,[23] learned to wear greased waterproof boots and to change their socks and boots at regular intervals. "Foot care" became an important part of military discipline. Casualties were reduced significantly. Nevertheless, the Russians had more than 1,000 frostbite injuries in one battle[24] and claimed that the Japanese had many more than were reported. Although the Japanese certainly improved the care of their soldiers and reduced the number of men incapacitated by cold,

Fig. 10-5. *Japanese Troops in a Snowstorm During the War With China;* original wash drawing by John Schönberg, 1895. The bitter winter conditions of the Sino–Japanese War of 1895 are vividly portrayed in this drawing of Japanese troops during a snowstorm. One man, exhausted and cold, has fallen to the ground and is being helped to his feet by his companions. Others appear indifferent to his trouble. The artist, Schönberg, covered the war for the *Illustrated London News*. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island. Jap. O. 1895 F-1.

the lessons were slow to be learned by other nations. World War I, only a few years in the future, would show, once again, how the medical lessons of one war are slow to be transferred to the next.

The Balkan War of 1912, between the Turks of the Ottoman Empire and the Balkan Alliance, one of the seemingly endless conflicts in that area, was another example of war under difficult, cold, wet conditions with the inevitable toll of thousands of injured soldiers. Page[25] reported many cases of symmetrical gangrene of the legs, often associated with enteritis. All these men had spent at least 24 hours in wet trenches with a ground temperature below 5°C; the cases were considered to be almost identical to frostbite.

By this time theories about treatment were beginning to change. Passive rewarming was being advised, although rubbing with snow was still commonly used.[26]

WORLD WAR I

World War I—the Great War, as it was known for a generation—began on 4 August 1914, and within a short time the politicians and generals were talking about "being home before Christmas." Disillusionment was soon in coming. By Christmas, after a German attack that nearly captured Paris had been turned back on the River Marne, both sides had settled into 4 years of unrelenting, stalemated trench warfare that claimed millions of lives and even more millions of wounds and injuries.[27]

Trench Warfare

Hundreds of thousands of soldiers spent days, even weeks, in trenches filled with near-freezing mud, sometimes above their knees. Within 4 months of the outbreak of war, the British army had 9,000 cases of what was named "so-called" frostbite. By the end of the war they would have sustained 115,000 cold and frostbite injuries (Figure 10-6).[28]

The conditions under which the troops, both Allied and German, had to live and fight were a prescription for disaster. Pierre Berton,[29] describing the conditions under which Canadian troops lived on Vimy Ridge, wrote:

> For most of that record winter the Canadians were cold, wet, hungry, tired, and, although they did not admit it, frightened. The cold was unbelievable. The temperature did not rise above zero Fahren-

Fig. 10-6. *Winter Conditions in the Yser Country: A Scene on the Snow-Covered Road Leading to the Rear;* uncolored photogravure after Fortunino Matania, 1916. In October 1914, the German army attacked the valley of the Yser River in Flanders to reach the coast of the North Sea. They were halted by a much smaller but determined British army. This photogravure shows British troops escorting German prisoners of war along a frozen, poplar-lined Belgian road. One of the Germans is blowing on his hands, while the two men at the head of the column seem to be holding each other for support and perhaps for warmth. The artist, Mantania, visited the British troops on the western front in 1915 on behalf of *The Sphere,* an illustrated newspaper of London, and produced a series of 12 photogravures (of which this is one) which were originally published in *With the British Army on the Western Front* by *The Sphere* in 1916. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island.

> heit for one month. The ground froze two feet deep, making it impossible to bury the horses and mules that died of cold and exposure. This was not the dry cold that the men of the prairies and Northwest were accustomed to. Fog and rain mingled with snow and sleet, the water in the shellholes froze overnight, the mud turned hard as granite so that the men were actually wounded by flying chunks of earth. They clumped along the duckboards swathed in greatcoats and jerkins, hooded by balaclavas under their steel helmets, their rifles wrapped in sacking; and they took their boots to bed with them to keep them from freezing stiff. It was so cold the bread froze after it came from the ovens and had to be cut apart with a hacksaw. Colds were so common that before a man could be sent to the rear he had to be suffering from pneumonia.[29(p85)]

When the mud was not frozen the trenches became a hell of slush and water, in some places so deep that a man falling into a shell hole could drown (Figure 10-7):

> ... and the mud, the dreadful clinging mud, reappeared. Nothing sapped the soldier's morale more than this ever-present gumbo, so glue-like that the strongest boots had their seams wrenched apart by men's efforts to struggle out of the morass. … The mud flowed like gruel around men's puttees, filled their boots, squeezed into their socks, and had to be scraped from between the toes with a knife.[29(p86)]

Conditions on the eastern front were equally harsh. Colonel Knox, British Military Attaché with the Russian armies in 1914, noted: "If we have to advance in the winter our losses will be three times as great."[27(p107)] Sentries froze to death at their posts, and it was impossible to provide hot food for the troops. Not only the men were affected: sometimes their weapons did not work either. Rifles and machine guns became clogged with mud and, at every level, military efficiency was reduced to a primitive struggle to survive.

On the eastern front the Russians reported 12,000 cases of frostbite.[27] Eight percent of all casualties were due to cold, and the Germans reported 10,000 cases of frostbite in *a single night*.[30] Only rudimentary treatment could have been applied to so many cases in a single night. Most of the frozen limbs must have thawed spontaneously. Those with lesser injuries made satisfactory recoveries; those with severe injuries lost their limbs.

In the Dardanelles, where the British made an abortive attempt to bypass the conflict in France and relieve the pressure on Russia, the troops, many from Australia and New Zealand, were unprepared

Fig. 10-7. *Extricating a British Soldier From the Mud of the Somme District;* drawing by Christopher Clark based on material received, 1917. In World War I the mud of France, churned into deep pools by constant bombardment, claimed the lives of thousands of men. *The Sphere,* a well-known British illustrated newspaper, printed this picture on its cover on 10 February 1917 during the Battle of the Somme, with the following legend:

> This man has slipped into a particularly soft spot en route to the trenches. He has become firmly stuck in the soft clinging soil, so much so that his efforts to liberate himself are quite unavailing. A rope is fastened to his arms and planks are pushed into the oozy hole. On these the rescuers slip and pant as they haul away at the unfortunate man. No result is obtained until others with spades begin to dig and loosen the mud. Then gradually the upward pressure tells, and the man is carried, not a little exhausted, to firmer ground.

What the original legend does not state is that even as the soldiers worked to free the trapped man, they were in danger of falling into the same morass themselves. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island. Originally printed by *The Sphere.* London, England; 10 Feb 17; LXVIII(890): cover 1.

for cold weather. On 27 November 1915, a freezing thunderstorm hit the peninsula. More than 100 soldiers were drowned in flooded trenches.[27,31] The rain turned to snow. There were more than 14,000 cases of frostbite, with a death rate of 0.58/1,000, a rate 10-fold greater than the death rate associated with frostbite in Europe. Dysentery was a common concurrent problem, as it had been in the 1912 Balkan War,[25] and other wounds or illnesses added to the dangers of the cold injuries. The snowstorm brought one blessing, though: it killed the swarms of flies that flew in thick clouds around the dead.[27]

In 1916 the Russians were locked in bitter fighting against the Turks. The city of Erzerum was heavily defended and surrounded by snow-filled valleys with few roads.[27,32] The British newspaper correspondent Philips Price wrote:

> The Russian troops had to cross mountains with deep snowdrifts at 10,000 ft and go for at least three days cut off from food supplies, with nothing but the few crusts of bread they could carry with them.[32(p209)]

Two thousand Russians died from frostbite and exposure in two nights.

In northern Italy a hotly contested mountain war was fought between the Italians and Austrians. Both sides had specially trained mountain troops, and scaling peaks and hauling artillery up and lowering wounded down cliffs were part of the regular pattern of fighting. Killian[30] quotes an Italian authority who claimed that there were 300,000 Italian cold injuries. The official number (38,000) was much smaller (Table 10-1).

Definition and Treatment of Trench Foot

At first the distinction between "true" (or "ground") frostbite and trench foot (or immersion foot) was not clear, but as World War I progressed it became clear that "trench foot" was distinct from "true frostbite."[33] In trench foot, the limbs were not frozen, and the water in which the men stood was not freezing, whereas in frostbite, tissues are frozen

TABLE 10-1

COLD INJURY IN WORLD WAR I

Army	Theater of War	Dates	Cold Injuries (N)
British	France	1914–1915	29,000
	Dardanelles	1915–1916	14,584
	All theaters	1914–1918	115,361
French	France	1914–1918	79,465
Italian	Italy / Alps	1915–1918	38,000
German	East and West	1914–1918	12,848*
United States	France	1917–1918	1,994†

*The accuracy of this number is doubtful because another report[1] indicated 10,000 cases in one day on the eastern front. (1) Gilbert M. *The First World War*. New York, NY: Henry Holt & Co; 1994.
†Most of these cases occurred in one battle on the Meuse / Argonne front in October–November 1918. An additional 67 cases occurred during training in the United States.

and ice crystals develop between cells. The salient points in the development of trench foot were

- prolonged exposure to cold and wet;
- dependency of the feet; and
- pressure around the lower leg caused by boots or the encircling, bandagelike "puttees," which were too tight.

Johns[33] thought that the condition was due to pressure on anesthetic feet. The men entered the trenches with their feet and legs swollen and hot from rapid marching and then stood up to their knees in ice-cold water and mud, which caused shrinkage of the boots and puttees. Localized gangrene, due largely to circulatory interference, developed in the feet.

Treatment of trench foot was largely symptomatic; amputation was rarely needed, but long-term disability was common. Tetanus and gas ("emphysematous") gangrene were constant threats because most of the trenches had been dug in farmland heavily fertilized with manure. The mixture of human and animal carcasses in the stinking mud only added to the risks of infection.[34]

One form of treatment was to rub the feet with whale oil every day. Berton[29] describes how the freezing whale oil was brought to the trenches every day, and the loathing with which the men applied the obnoxious cure to their feet. The penalties for not using the oil daily were severe, not only for the man who failed to apply the oil but also for his senior officer. Other oils were suggested: eucalyptus; methyl salicylate; and a mixture of castor oil, glycerine, and atropine.[34]

Delépine,[35] professor of public health in Manchester, England, devised a waterproof sock of oiled silk that was thin enough to be worn between the ordinary sock and the boot. The idea was ingenious but impractical for men standing in water and mud several feet deep.

One of the salient symptoms of trench foot was a feeling of intense warmth in the feet and lower legs. The casualties with trench foot were nursed in a cool atmosphere, with feet uncovered and protected from the bedclothes by a cradle.[36] The treatment of true frostbite, in contrast, was to put the casualty in a cool room and rub the frozen parts with snow or cold water.[37] Only later was the patient transferred to a warmer environment. The official advice to the US Army and Navy in 1917, still reflecting the teaching of Larrey, was to rub the frozen part "vigorously" with wet snow or ice water, "never with dry snow as the temperature of dry snow may be much below freezing."[38(p125)]

COLD INJURY RESEARCH TO 1939

Investigations During World War I

The number of cold injuries in World War I (see Table 10-1) must have taxed the medical services of the armies on both sides. If anything was learned it was that conservative management was better than precipitate amputation. The gangrene that followed trench foot was more likely to be "wet" (ie, ischemic

necrosis with bacterial infection), whereas the gangrene that followed "true" frostbite was more likely to be "dry" (ie, shriveled or mummified necrosis). But amputation owing to gangrene was relatively rare.[34] The number of injuries, and the impact on the fighting strength of the armies, stimulated research into an increased understanding of the pathology, particularly of trench foot.

Lake[39] investigated the effects of freezing on cell cultures and determined that –6°C is a critical temperature, below which serious damage occurs. With the acute interest at the time in trench foot, much of his work was directed to elucidating the pathogenesis of the lesions. High capillary pressure was found to be an important secondary factor, although cold was the primary etiologic influence.

Lorrain-Smith, Ritchie, and Dawson,[40] from the University of Edinburgh, studied 51 cases of "trench frostbite" of the feet and legs. They described how soldiers spent 48 to 72 hours in the front line, although the period was occasionally as long as 6 to 11 days. The mud was frequently mixed with ice crystals, and fires were not available in the dugouts for warming or drying. They advised that soldiers massage their legs before going up to the front line, that duties in the trenches should be restricted to 48 hours, and that all constricting clothing be avoided. They also advised that at the development of the first symptoms soldiers should not be allowed to walk before receiving treatment. The feet were to be rested and not warmed.

Page[41] tried a controlled study in 332 patients, treating one foot with dusting powder and the other with a variety of counterirritants, hot fomentations, massage, or electrical stimulus. He found that counterirritants relieved pain for a short time, hot baths accelerated the development of hyperesthesia, massage sometimes became difficult to bear but it expedited cure, and stroking the legs with a faradic current cured anesthesia. It was not possible to draw conclusions about the efficacy of one treatment over another.

The advice given by these authorities and others was heeded and the incidence of cold injuries decreased steadily throughout the years of the war.[21] When the war started there were few medical officers who had either knowledge or experience of cold injuries. It did not take long, however, for the field commanders to become aware that prevention was possible. Under the conditions described by Berton,[29] prevention was difficult if not impossible, but to some extent it was successful with the introduction of rubber boots and wooden duckboards for the men to stand on. The British army's trench foot rates for 1914 and 1915 were 33.97 and 38.43 per 1,000 hospital admissions, respectively; but in 1916 the rate was down to 12.82, in 1917 it was 11.34, and in 1918 it had plummeted to 3.82.[21]

Between the Wars: 1918–1939

Between the two world wars the treatment advised for frostbite was, almost universally, rubbing with snow or the application of cold. The reasoning behind this advice was that rapid warming caused more pain, swelling, and tissue destruction than did very slow rewarming.

Robert Greene,[42] a doctor with several major British mountaineering expeditions in the 1930s, advised that frozen feet be exposed to a cold environment, although there was little experimental evidence to support this view, with which not all experts agreed. Scott,[43] of Antarctic fame, had in 1905 described warming frozen parts by placing them on the warm, bare skin of an associate. Other famous polar explorers, such as Byrd in 1935, similarly advised the use of body heat and condemned the use of friction with snow.[44] But, overall, the advice was still to use cold, rubbing with snow, or gentle friction with cloth or fur.

During the mid to late 1930s, a group of scientists at the Kirov Military Medical Academy of the USSR Army, including T. V. Ariev, proved that rapid warming was preferable to older methods using cold.[45] Ariev was both a surgeon and scientist, and he found that frozen rabbit ears, rewarmed rapidly, sustained less damage than ears allowed to warm spontaneously in air. Historically unsubstantiated evidence against rapid warming did not deter Ariev from looking at the possibility of using this method. In 1940 he wrote:

> The great majority of recent observers and some of the older observers claim that rapid warming of frozen extremities aids (or may even lead directly to) development of gangrene, and that rapid warming in cases of generalized freezing causes death. … Although there is no basis for this argument, it is widely accepted. ... 1) No concrete evidence proving the injurious effect of rapid warming which could withstand criticism has been encountered in the literature. And, 2) The experiments used to prove the value of slow warming are insufficient in number and the methods used are inadequate.[46(p99)]

WORLD WAR II

On 1 September 1939, when Germany invaded Poland, war once more engulfed Europe. Before the war ended in 1945, more than 55 million people had died. The Russians had more than 10 million military and civilian casualties. Cold injuries alone numbered in the millions. The Germans fighting in Russia had more than 250,000 frostbite injuries, most in one winter.[30] The Americans, in other theaters, lost the equivalent of several divisions temporarily or permanently because of cold injuries.[21] The "phony war," as the first months were called in Europe, produced a sizable crop of cold injuries despite the absence of serious fighting (the winter of 1939/40 was unusually cold, one of the coldest in recent memory[47]). The French, sitting in the relative comfort and security of the Maginot Line, sustained 12,000 cold injuries during the first winter of the war.[20]

The Russo–Finnish War

On 30 November 1939, Stalin invaded Finland. Four Russian armies launched a preemptive strike into untracked forests. By the end of the campaign, 1 million Russian troops had been thrown against 300,000 Finnish soldiers, most of whom were reservists.[2] The Finns were fighting to protect their homeland and their soldiers were trained to fight on skis; they appeared suddenly, attacking Russian convoys that were bogged down on forest roads, and then blended back into the forests (Figure 10-8). In the battle for Suomussalmi, the Russians lost 27,500, killed or frozen to death.[8] The Finns lost only 900 killed and 1,770 wounded. The Russians lost 200,000 men in the campaign, many from exposure to cold or starvation, compared with 25,000 lost by the Finns.

Fig. 10-8. Sketch by an unknown Finnish artist. During the Russo–Finnish War of 1939/40, the Finns were vastly outnumbered but made up for the disparity in numbers with their skill as ski troops. Finnish troops, known as the "White Death," were able to make quick attacks on the Soviet columns laboring along the poor forest roads. The Red Army troops, who proved inadequately armed and poorly trained for the winter campaign, could not reply. For a time, it was thought that the Finns might win the war. Eventually the USSR overwhelmed the Finnish army, but not before the White Death had extracted a terrible toll in the Russian campaign. Reproduced with permission from McCormick K, Perry HD. *Images of War: The Artist's Vision of World War II.* New York, NY: Orion Books; 1990: 9.

The massive weight of the Russian attack finally wore down the Finnish defenses, but the Russian military leadership, decimated and weakened by Stalin's prewar purges, discovered that their troops were ill-trained for winter warfare, and that their clothing and equipment were inadequate. They also learned the value of ski troops and how to manage cold injuries. The Russians would benefit from this experience a year later.

German Invasion of Russia: Operation Barbarossa

On 22 June 1941, 3 million German troops smashed into Russia in the start of Operation Barbarossa.[2,48] The Russians fell back on all fronts. A German victory seemed certain before the summer ended, but the advance petered out in the vastness of the Russian plains. A winter campaign became inevitable. The German High Command was aware of impending problems, but it was not until August 1941 that an order went out from Generaloberst Franz Halder, chief of staff of the German army, that a report should be made on the winter clothing needed.[2]

Confusing Decisions: The Start of Winter, 1941

According to a postwar report written by Colonel Paul Hagendorn,[3] a staff officer involved with supplies and logistics, the German High Command made a fateful decision on 15 October 1941, at 4:30 pm, that marked a turning point in the war. The original plan of the General Staff was to advance as far as Moscow and then dig in for the winter, conserving troops and supplies for a campaign in the spring of 1942. After the invasion slowed and the autumn rains turned the Russian roads into impassable quagmires, members of the German High Command disagreed over the correct course to take. After much indecision, Hitler personally reversed the plans to dig in for the winter. There would be no pause, although the troops were already struggling and the first cold winds of winter were blowing across the steppes. Moscow must be taken. The commanders were ordered to fill their transport vehicles only with ammunition, food, and fuel, and to leave winter clothes behind. With typical thoroughness, the German supply corps had accumulated enough winter equipment for the 50 divisions expected to occupy defensive winter positions, but they had neither prepared for nor obtained sufficient winter clothing for the 150 divisions required for the attack. The fate of thousands of men was sealed by a dictatorial afternoon decision.

The attack stalled a mere 18 miles from the gates of Moscow.[48] Some advanced German patrols even entered the suburbs,[49] but the main body of troops was exhausted, cold, and short of food and fuel. Before the winter of 1941/42 was finished, 200,000 German soldiers had been frostbitten.[21] General Winter reigned supreme. The ghost of Napoleon, who said he had been defeated by Generals January and February, must have been saying, "I told you so."

The early part of the winter was not unduly severe, but as December progressed into January and February the weather became bitterly cold. The Panzer chief, General Guderian, reported the temperature to be –63°C. In a moment of depression he wrote to his wife:

> The icy cold, the lack of shelter, the shortage of clothing, the heavy losses of men and equipment, the wretched state of our fuel supplies, all this makes the duties of a commander a misery.[2(p174)]

Learning from the Finnish experience, the Russians had thousands of ski troops who out-maneuvered the Germans, attacking from many sides. The snow was so deep that horses pulling the German guns floundered up to their bellies, and retreating soldiers had to spend their nights digging roads along which they could retreat the next day. (The German army used 2,750,000 horses during World War II, most of which died.[50])

The conditions under which the Germans fought were cruel. No one described the conditions better than General Guderian:

> Only he who saw the endless expanse of Russian snow during this winter of our misery ... who drove for hour after hour through that no-man's land only at last to find too thin shelter, with insufficiently clothed half-starved men; and who also saw, by contrast, the well fed and warmly clad and fresh Siberians, fully equipped for winter, only a man who knew all that can truly judge the events that now occurred.[2(p175)]

Winter clothing never reached the front—a frequent complaint in many armies—and when it arrived the quantities were totally inadequate. Complaints were made at the highest levels: Deighton[48] recounts a telephone conversation between Generalfeldmarschall Fedor von Bock, commander in chief of Army Group Center in Russia, and Generalfeldmarschall Walter von Brauchitsch, commander in chief of the German army, in which Bock complained bitterly that all the cold weather supplies were stored in areas hundreds of miles

from the front. Brauchitsch's response was, "The Fuehrer wants to know when Moscow will be captured."[48(p213)]

One battalion of 800 men received only 16 winter greatcoats and 16 pairs of fur-lined boots (Figure 10-9).[2(p217)] The temperature fell to –40°C to –60°C. Men urinated on their freezing hands to warm them. The cracked skin bled with every movement.[51] Soup that came boiling from the pot was frozen before it could be eaten. The tank engines could not be started. The recoil mechanisms of the guns froze,[48] and fingers froze to exposed metal. The wounded or the exhausted who fell froze to death if they could not be roused into action.

After the war, Colonel Hagendorn claimed that German winter equipment was good, but that inadequate supplies resulted in too few soldiers being appropriately dressed; in other words, they were ill-equipped for the Russian winter (Figure 10-10).[3] When the temperatures were bitterly cold and the snow was dry, the Russians fought in felt-lined boots,[49] while the Germans wore leather, calf-high

Fig. 10-9. Painting, title and date unknown, by W. F. Gebhardt; captured German art, formerly held at US Army Center of Military History, Washington, DC, has been returned to the German government. Two German sentries of an SS unit stand watching over the bleak Russian landscape. Because they were SS troopers they were well supplied with thick winter clothing; most German soldiers were inadequately clothed and never had access to the type of clothing worn by these troopers. Reproduced with permission from McCormick K, Perry HD. *Images of War: The Artist's Vision of World War II.* New York, NY: Orion Books; 1990: 142.

Fig. 10-10. *Ill-Equipped for Russian Soil, December 1941;* photograph by Galina Sankova. The picture tells the story: two dead Germans, one hatless and the other wearing the standard open-topped boots of the German army. The soles of the boots were thin, with metal studs that conducted heat from the feet to the ground. The open tops of the boots allowed snow to blow in and surround the feet. Frostbite was inevitable. Reprinted with permission from Mrazkova D, Remes V, eds. *The Russian War: 1941–1945.* New York, NY: Dorset Press; 1975: unpaginated.

boots filled with rags and paper. The Russians had quilted clothes; the Germans, standard woolen field uniforms; and the Germans had to increase the insulation of their uniforms by stuffing newspapers between the layers. One German recalled how he used propaganda leaflets telling the Russians to surrender as insulation.[2]

The Russians, and especially the Siberian troops brought from the East to support the winter counterattack, knew how to live in the cold and treat cold injuries. They knew how to improvise stoves to keep themselves warm. The Germans burned precious gasoline. Shelter was impossible to find because the ground was too hard to dig. Everything confirmed the well-known experience that in winter, the losers have more casualties and a greater number of frostbite victims than the victors.[21] The number of casualties alone made treatment impossible in the retreating troops. As Professor Hans Killian, a surgeon with the German 16th Army, later wrote, the occurrence of cold injuries in every war waged in the cold should not be taken as an indictment of the medical officers, quartermasters, and commanders, but is a consequence of war itself. The cold injury is "the badge of depression, demoralization and defeat."[30]

In 1942 a German medical officer who had served in Russia outlined the treatment used.[52] Warming was gradual. Frozen parts were elevated to reduce edema. Blisters were incised and covered with antiseptic powders. Hasty amputations were avoided and surgery used sparingly. The same account described how the frozen parts were "bathed" in water at 25°C to 30°C, which was gradually heated until it reached 40°C. Rewarming in water was, therefore, already being used, although more conservatively than later. Alcohol and coffee were given as stimulants.

The Germans learned the same bitter lessons as had previous armies that had invaded Russia. Training, personal discipline, supervision by the noncommissioned officers of the troops, strong morale, good food, and equipment reduced the number of casualties. Some factors, such as the decisions of the High Command, were beyond the control of the fighting men and, sometimes, the press of battle made protection against the cold impossible. Given equal circumstances, a well-disciplined unit had fewer cold injuries than one with lax control and lazy supervision.

Professor Killian personally supervised the treatment of 5,243 cases of first-degree frostbite, 12,937 cases of second-degree, and 1,455 cases of third-degree with 393 amputations, all in the winter of 1941/42. The German army pathologists at that time reported that one third of the autopsies showed evidence of frostbite. Of the German troops who were frostbitten, 40% were out of commission for 3 months, 10% for 6 months, and 2% were fit only for service at bases in Germany.[30]

The Germans suffered in every winter of the campaign. Early in 1944 a retreating German unit was halted by a river 30 feet across and 6 feet deep. Floundering horses drowned. Men flung themselves into the river only to have their clothes freeze on them. When they came out on the far side, "they

were turned into blocks of ice."[2(p377)] Many of them tore off their clothes. A witness described how

> soon hundreds of soldiers, completely naked and red as lobsters were thronging the other bank … . Under the fire of tanks, thousands upon thousands of soldiers, half clothed, streaming with icy water or naked as the day they were born, ran through the snow to the distant cottages of Lysianka.[2(p377)]

After the war a young German soldier, Hans Burtscher, who had been a member of the No. 4 High Mountain Infantry Battalion in Italy, wrote a bitter account of his earlier experiences with the German mountain troops in the Caucasus and Italy.[53] His unit had defended a high (9,100-ft) pass against repeated Russian attacks for 2 months. Their supplies could not reach them and they were starving:

> Winter clothing consisted of two sheepskin coats, teeming with lice, for the sentries. They probably had been on loan from the Italians, because the German fur coats, as well as other useful items, reportedly were found only about 350 miles behind the front lines where they kept party functionaries warm and comfortable. But, as mentioned above, such subjects could not be discussed openly, because there always existed the likelihood of being turned in by a snitch.
>
> For weeks there was no way to prepare warm meals in positions above an altitude of 9,000 ft, no firewood was to be found, and portable cook stoves were not available. The daily ration consisted of a thin slice of bread, a small chunk of blood sausage, a tablespoonful of jam and dab of margarine, two pieces of candy and 10 French cigarettes. Bread and sausage were usually covered by ice crystals, and it all barely filled a man's hand. We were occasionally able to break up captured weapons and dismantle enemy grenades and mortar rounds to get powder to start a fire from broken rifle stocks and melt snow to prepare some tea. Deplorable conditions such as these, as well as others too numerous to list here, and the unfolding Stalingrad disaster, increasingly fueled doubts, even among the younger soldiers about the strategic talents of the Fuhrer. But he had his informers everywhere, who made sure that any mutineer was sentenced without delay to do time in a murderous penal battalion, or worse. No wonder the preferred conduct was therefore to remain a silent coward, instead of becoming a dead martyr, or traitor, accused of undermining the morale of the army.[53(pp2–3)]

A photograph in his account of a group of soldiers has the caption: "Of the group seated in front, only one was alive, four weeks later, with hands and feet amputated."[53(following p3)]

German Cold-Weather Training Manual, 1942

Despite the terrible conditions and heavy casualties from the cold, the official German army training manual, entitled *Winter Warfare*,[54] published in 1942, and based on experience in the Russian campaign, failed to sound the warnings that might have been expected and gave a falsely optimistic impression of how the German soldier had coped with the winter conditions of the previous year:

> Experience teaches us that the German soldier knows how to master the difficulties of the Russian winter, and that he is superior to the enemy even in winter. He is capable, not only of defending himself against the Russian but also of annihilating him in the attack.
>
> Prerequisites for this superiority are as follows: psychological preparation for the hardships of winter warfare, appropriate training and adaptation, familiarity with winter combat methods, and proper equipment and employment of expedients.
>
> In building up endurance against the rigors of the Russian winter, mental discipline is the determining factor. Many cases of freezing are caused by the slackening of attention and by indifference. The danger of freezing is especially great when one is exhausted after great exertion or after a long period on guard. Then the soldier must summon all his will power in order to keep awake and alert. The code of comradeship demands that soldiers must assist each other in this effort and in stimulating the will to live. The most serious danger begins when confidence in one's own strength is extinguished.[54(pp4–7)]
>
> . . . .
>
> In addition to the fight against the enemy in winter, there is also the struggle against nature—against cold, snow, wind, poor visibility and prolonged darkness. ... Knowledge of the following fundamental subjects is necessary: clothing and equipment—rations—maintenance of health—care of weapons, equipment and ammunition, care of motor vehicles—care of horses—heating facilities. Generally speaking the danger of illness resulting from cold is slight, provided blood circulation is normal. It is impossible to warn too forcefully against the use of alcohol as protection against the cold. It dilates the pores and simply stimulates a feeling of warmth. It abets exhaustion and death by freezing and, therefore, must never be taken prior to physical exertion. If alcohol must be used, it is best administered in hot beverages such as tea.

> It may be issued only if a subsequent protracted stay in heated accommodation is expected. Those who must again go out-of-doors (sentries) must not receive alcohol.[54(p27)]

The authors of this manual must have been aware of the inefficiency and inadequacy of the logistical system that led to so many casualties. But the bitter pill had to be sugared:

> If supplies issued to the unit are not sufficient, they must be augmented by improvisations and substitutes of all kind. The ingenuity of the individual soldier and of the leader keeps the unit efficient and reduces casualties. … In case of a shortage of felt boots, sentries and drivers may wear shoes of straw over their regular footwear. Use the natives to manufacture straw shoes. … Nevertheless it is necessary that the unit should try to overcome the cold independently by using winter clothing as effectively as possible and by devising additional expedients.
>
> Emergency precautions against the cold. Put felt lining inside the steel helmet—preferably the crown of an old felt hat. If nothing else is available, use a handkerchief or crumpled newspaper. … The feet are especially susceptible to frostbite. Socks should be changed frequently. A proved measure for preventing frostbite of the feet is to use inner soles of straw, cloth or paper. The straw should be cut to the right size and arranged carefully, if newspaper is used it should be wrapped carefully around the foot to avoid wrinkles. An especially effective measure for protecting the feet is to wear paper between two pairs of socks and another layer of paper or foot cloths over the top pair of socks. All wrinkles must be smoothed out.
>
> Special protective measures for the genitals should be taken if the weather is very cold or the wind very strong. The soldier should wear short trunks, if they are available, in addition to his underwear or should place paper between his drawers and his trousers.[54(p131)]

Some of the advice bore little relation to the realities of warfare in the Russian winter:

> Always have warm water ready for drinks. ... If it is not possible to feed the troops from a field kitchen, they should be given instant coffees or tea. Every man should know how to cook.[54(p128)]

It was noted that red wine does not keep well in the cold and that supplies of flour could be supplemented:

> Flour rations can be stretched by adding sawdust flour, made preferably from the pine tree, but birch may also be useful for this purpose.[54(p140)]

US Army: 10th Mountain Division

Mountain warfare demands special skills and training. Deep snow and icy slopes and cliffs pose unique problems in the evacuation of casualties. Freezing temperatures and strong winds add to the difficulties. Standard US infantry battalions were not prepared to fight in this environment. While the Russian campaign was being fought, the US Army was training its first mountain warfare troops. During World War I there had been hard fighting in the mountains of northern Italy, in the Carpathians, and the mountains of Asia Minor. For more than a century the British had been fighting the mountain people of Afghanistan and the Pathans, who controlled the Khyber Pass. The Germans entered World War II with four specially trained mountain divisions, and added four more as the war progressed.[55] The US Army, however, had no mountain troops until 1940, when a group of US civilians—Robert Livermore, Roger Langley, C. "Minnie" Dole (founder of the National Ski Patrol), and Alexander Bright—urged the Department of Defense to start a mountain unit capable of fighting in extreme conditions of cold and altitude, trained in all the skills of survival and battle necessary for that environment.[56]

On 15 November 1941, the 87th Mountain Infantry Battalion (Reinforced) was activated at Fort Lewis, Washington. The National Ski Patrol was one of the official recruiting agencies, and each candidate for the regiment required three letters of recommendation even to be considered. Major Robert Cook described the first days at Fort Lewis when he was assigned there to be the supply officer:

> About noon a solder walked in with a pair of skis on his shoulder. He said he had been captain of the Dartmouth ski team. I gave him some keys and requested that he open one of the barracks and turn on the heat. It was getting cold in November. The next soldier was carrying an ice axe. He was from the Teton Mt area. The third day a 1st Lt from the Reserves reported. I asked him if he knew anything about army paperwork. Yes, he replied. I told him to take a seat as he was now the Adjutant of this outfit. His name was Townsend.[57]

By 8 December 1941, one day after the Japanese attack on the United States at Pearl Harbor, Hawaii, 8,000 recruits had been accepted out of 15,000 applicants.

The 87th Regiment was later joined by the 85th and 86th Regiments to form the 10th Mountain Division, which trained in Colorado and saw action in the Aleutians and Italy. The selection, training, and equip-

Fig. 10-11. Troops of the US 10th Mountain Division train in the Colorado mountains in winter 1944 at Camp Hale. They are wearing specially designed mukluks on their feet and large mittens on their hands. Their anoraks are designed to keep out wind and snow but permit ventilation. Despite good clothing many men were frostbitten on training exercises, usually owing to their inexperience in dealing with winter conditions. Reprinted with permission from Denver Public Library. Call Number TMD-905.

ping of this new division was a huge endeavor that made use of a combination of civilians, expert in skiing and mountaineering, and career military personnel, capable of molding an unusual group of men into a fighting unit. A camp, which eventually housed 20,000 men and thousands of mules, was built at Camp Hale, in the middle of the Colorado mountains on the site of an old mining railroad stop (Figure 10-11).

Special equipment had to be tested and manufactured. Well-known mountaineers were recruited to conduct expeditions in the Saint Elias Range of the Yukon, Mount McKinley, and Mount Rainier to test new sleeping bags and tents, socks and boots, skis and ski bindings, and compact food and stoves.[58] The tests revealed that the first single-layer sleeping bags supplied by the US Army Quartermaster Corps provided inadequate protection against the cold, so a double-layer bag was designed. In addition, special boots—suitable for both climbing and skiing—became standard issue, and the felt-lined "shoepac" boot, with a rubber foot and leather, calf-high top, which is still popular today, proved to be useful but was not always available.

The winter conditions under which the 10th Mountain Division trained were severe and the men sustained more frostbite during the training exercises than during subsequent campaigns. In the early days, maneuvers were started before the men were fully trained:

> From the onset the maneuvers were a fiasco. It was midwinter in Colorado. Icy blizzards were sweeping the mountains. Because of their lack of training men became casualties from the elements with terrifying rapidity. The tactical situation was forgotten, lost in the simple struggle for survival, bewildered troops staggered down the mountains with frost-bitten feet hands and faces. Some of them were raw recruits with only a few days of military training of any kind.[59]

A soldier recorded in a letter home:

> Some of the fellows froze their toes or feet while in Colorado. Our Lt H. froze his hands and foot, and they wanted to amputate them, but he took a discharge from the service instead. Lt H. was one of the smartest officers I [have] ever known, so I hated to see him go.[60]

In 1944 a notorious exercise, named Series D, was held to test the ability of the division to function in extreme conditions. The maneuvers lasted 3 weeks. The temperature fell to –36°C and there were numerous cases of frostbite. The men spent most of the period camping but, despite problems, the exercise proved that the division was ready to fight in the mountains under all conditions (Figure 10-12). They were able to carry packs that contained 58 items and weighed 80 to 100 lb, and still travel on skis. They had become skilled at cliff and rock climbing. Their artillery was carried by mules that could operate as effectively in 4 ft of snow as in the heat of summer. "I really like them critters,"[61] said the officer in charge of the mule trains. With characteristic military logic, the division's final training was on the hot, dry flatlands of Texas.

During the period of training, great emphasis was placed on the principles for keeping warm and preventing freezing to death:

- avoid overheating,
- reduce sweating,
- minimize evaporation and condensation, and
- layer clothing.

Recognition was also given to psychological factors. Conditioning to exposure to cold and freezing temperatures, to thirst, hunger, and exhaustion, and above all, to the demoralizing effects of cold was the basis of all training. At that time the pathogenesis of frostbite was ascribed to vasoconstriction and stagnation of the circulation. The etiologic importance of dampness was recognized and, with one exception, the standard warning about treatment was not to rewarm rapidly.

One winter warfare manual (1944) went against traditional advice and stated:

> In treating freezing the frozen part should be thawed as quickly as possible without injury to the

Fig. 10-12. Camp Hale, high in the Colorado Rockies, housed more than 20,000 men training in the 10th Mountain Division. A memorial with a picture of the camp as it once was now stands along the nearby highway, but there are few remains of the camp except the concrete bases for huts long-since removed. Not far away, in the ski resort of Vail, Colorado, one of the most popular runs, Riva Ridge, is named after the Division's most famous battle, in Italy. Reprinted with permission from Denver Public Library, Denver, Colorado. Call Number TMD-784.

> tissue. This can best be accomplished by putting the frozen part of the body in luke warm water.[62]

No attribution was given for this advice, which was contrary to all current opinion, but it predated by 6 years the advice on rapid rewarming given by Furhman,[63] although it came several years after the work of Ariev.[46]

There were many similarities between the advice given to German troops and that given to the 10th Mountain Division. One essential factor in the training of both armies was the emphasis on personal responsibility for keeping warm and preventing frostbite. One US manual finished with this injunction:

> Let there be no mistake about who must do the learning. It is not the squad leader, not the aid man, not the commanding officer, but you [the] soldier.[62(p114)]

In May 1943 an American offensive was launched to retake the island of Attu in the Aleutian Islands from the Japanese. The amphibious assault met stiff resistance and the battle was viciously contested. Although the operation was conducted during the spring, daily temperatures were low and fell below freezing at night. There was almost constant rain or snow, and cold injuries were as frequent as wounds. The operation was, perhaps unrealistically, planned to last 3 days but lasted for 20, and ended with the mass suicide of the last 500 Japanese defenders. Garfield, a historian, wrote later of the operation:

> The cold was intense. Men limped on frozen feet and vomited silently. In training, W. had lectured them on health measures; but, W. recalls, "The ones who suffered were the ones who did not keep moving. I tried to keep everyone on the move, but I didn't catch some of them. They stayed in their holes with wet feet. They didn't rub their feet or change socks when they needed to."[64(p163)]

In the battle for Attu, 1,148 soldiers were wounded and there were 1,200 cold injuries, a 1:1 ratio that could not have been sustained for a longer campaign.[64] The troops involved were inexperienced and some were not equipped for the cold, wet environment in which they had to fight. It is unlikely that the commanders had taken into account the possibility of so many cold injuries.

In August 1943, the Aleutian island of Kiska, which had also been occupied by the Japanese, was retaken without resistance because the island had been evacuated 2 weeks earlier. The assaulting force, which included the 87th Mountain Infantry Regiment from Camp Hale, was unaware that they were attacking an empty island. The troops, none of whom had been in action before, climbed the steep mountains in swirling mist and oncoming darkness. Cold, wet, nervous soldiers imagined that they saw Japanese troops advancing to attack them.[65] They could not distinguish between the vague figures of their own men and possible enemy. Firing was undisciplined and haphazard. Casualties included 23 deaths from friendly fire, 45 wounded or severely sick, and 130 cases of trench foot.[64]

In 1945 the 10th Mountain Division fought with distinction in Italy, but only in the battles for Mt. Belvedere and Riva Ridge were their mountaineering skills required.

COLD INJURY RESEARCH DURING WORLD WAR II

The magnitude of the morbidity and mortality rates among troops, aircrews, and submarine crews due to cold was a compelling stimulus to the major powers to embark on research programs to solve the problems of the etiology and treatment of frostbite, trench foot, and hypothermia.

German and Japanese Research

The Germans and the Japanese were deeply concerned about cold injuries. The Germans not only had catastrophic casualties in Russia, but their U-boat crews also had the highest mortality and casualty rate of any branch of any service in World War II.[48] Of 40,000 men in the U-boat service, 28,000 (70%) lost their lives. Many of those deaths were due to exposure or a combination of hypothermia and drowning. In attempting to find methods for rewarming men and preventing frostbite, both nations carried out ethically abhorrent cold research.

German experiments in the concentration camp at Dachau were related more to the treatment of hypothermia than frostbite; their experiments with frostbite contributed nothing to our knowledge. The experiments on immersion hypothermia, designed to find a way to rewarm airmen and U-boat crews, confirmed that soaking in warm water was the best and safest way to rewarm victims of immersion cooling.

Soon after the war ended these experiments were investigated by Major L. Alexander, MD, Medical Corps, US Army.[66] He had access to Himmler's per-

sonal copy of the report on experiments, which was found, when hostilities ceased, in a cave full of archives. The prime investigator was named Dr Rascher, who was married to Himmler's former secretary. Through this relationship he obtained permission to use concentration camp prisoners as experimental subjects. Although large volumes of data were obtained, the experiments were ill conceived, and there has been considerable discussion about whether results obtained under such terrible conditions should be used, or even believed.[67]

The Japanese also used thousands of prisoners, including at least 37 Americans, in experiments on biological warfare. These experiments were carried out at a secret establishment in Manchuria called Unit 731.[68] Although the main purpose of the work was to develop methods of spreading plague and anthrax, Miyaoshi Watanabe, a midlevel scientist, was assigned the task of investigating frostbite.[69] He was interested in two aspects of cold injury: first, the best treatment of frostbite, and second, the development of a test that would distinguish between men who were resistant to cold and men who were susceptible to cold. The hope was that military units could be assembled in which all the soldiers would be resistant to cold.

Men were lined up in temperatures of –20°C to –30°C with their hands extended in front of them. Observers went up and down the lines measuring the time taken for fingers to become dead white. The times ranged from 1.5 minutes to 1.5 hours. Various methods of rewarming were then tested. The Japanese, as had the Russians, found that immediate thawing in warm water was best. Legitimate experiments by Yoshimura, which had been done during the hostilities but published after the war, confirmed the same findings.[70]

At the end of the war Unit 731 was overrun by the Russians, who did not realize the importance of the place they had captured. Most of the records had been destroyed or removed, and the workers were naturally reluctant to say what they had been doing. Watanabe was interviewed by US military intelligence, but the interrogator had no medical background and probably did not realize the significance of what he was hearing. Watanabe reported that he had used soldiers (not prisoners) as experimental subjects. This is unlikely because of his position as a scientist at Unit 731, where prisoners were routinely used for experiments. Once again, thousands of humans were subjected to unethical experiments to obtain information that was already known to the Russians and was being rediscovered by the Americans. (German and Japanese experimentation on prisoners is discussed in detail in *Military Medical Ethics*, a forthcoming volume in the Textbook of Military Medicine series.)

US Army: Management of Cold Injuries

The US Army, Army Air Corps, and Navy had 90,000 cold injuries during World War II. The land casualties occurred mostly in the Aleutians in 1943, in Italy in the winter of 1943/44, and in the European theater in the winter of 1944/45.[21] Thousands of cases of immersion foot occurred in the US Navy and merchant marine, in sailors who had tossed for days in lifeboats and rubber rafts in the North Atlantic. In the Army Air Corps, gunners in blister turrets on the flanks of Flying Fortresses unjammed their machine guns with bare hands, and only a few seconds of exposure to the cold and metal resulted in frostbite. "High-altitude" frostbite became a recognized entity.[71] The first recorded case of frostbite in a flyer occurred in World War I in 1915 and was reported in the *Journal of the Royal Naval Medical Service* in a pilot at 15,000 ft, where the temperature was calculated to be –36°C.[72]

Cold injury, particularly trench foot, was early recognized by the US military to be a major and, in many instances, a preventable cause of troop losses. The drain on German resources during the Russian campaign may have influenced the final outcome of that campaign. While the extent of American cold casualties never reached those proportions, thousands of servicemen were incapacitated. According to Whayne (discussed below),[21] 55,000 cases of cold injury were reported, with an average loss of time per case of 50 days: 2,757,300 man-days.

The general policy in the US military services during World War II was to warm frozen parts slowly. The *Military Medical Manual* (1942)[73] advised:

> No temperature higher than the normal temperature of the body should be used in the treatment of frostbite. … The person suffering from freezing should be removed to a moderately warm shelter and permitted to reestablish gradually the normal circulation without undue disturbance.[73(p560)]

A 1943 document entitled *Burns, Shock, Wound Healing and Vascular Injuries*,[74] prepared by the Committee on Surgery of the Division of Medical Sciences of the National Research Council, advised:

> The frostbitten individual should be kept in cool, not hot, surroundings. … The frostbitten part must (be) warmed slowly—it cannot respond to heat as yet by vasodilation—by gentle kneading with the

> normally warm hand or by contact with the normal body. Never apply heat.[74(p248)]

In 1945 the *US Maritime Service Hospital Corps School Training Manual*[75] read:

> Treatment of these conditions (frostbite) consists of elevation of the affected part to improve the return of blood flow and the gradual elevation of the temperature by allowing it to thaw in a cool room at room temperature. … Friction or heat should never be applied to these injuries.[75(p308)]

US Army: Cold Injuries in Europe, 1944–1945

A detailed study of trench foot casualties among American servicemen in World War II was written in 1950 by Thomas F. Whayne, Colonel, Medical Corps, US Army, and submitted as a thesis to the Harvard School of Public Health for the degree of Doctor of Public Health.[21] Colonel Whayne analyzed all the cold injuries in the European theater in US and British troops during the last months of 1944 through March 1945. Statistics on the number of casualties were correlated with time of year, temperature, weather, battle activity, clothing, supplies, and other factors that influence the incidence of injury. Whayne analyzed data obtained from the records of 21 divisions that served in Italy in the European theater. When circumstantial factors were correlated with the incidence of cold injury, combat action and shelter were the two with the greatest statistical significance. Terrain and rotation out of battle were marginally significant. If the weather was bad enough to make trench foot likely, the incidence depended more on the battle action than the terrain or shelter available.

Whayne collected statistics down to regiment and platoon levels, and from them he drew important conclusions:

- Training. Better trained and experienced units had fewer cold injuries than units with less training. One unit that had been stationed in Iceland before transfer to the European theater had received extensive training and practical experience with cold. They had few cases of cold injury except during a period of heavy fighting. On the other hand, some inexperienced units had many cases of trench foot even before reaching the active zone. One battle-tried division with excellent leadership had only 164 cold injuries during the entire winter of 1944/45. Another unit, a regiment with inexperienced leaders, had 400 cases in their first week of fighting; these men were fully equipped with winter clothing and overshoes. The connection between discipline and training is obvious.[21] Experience and an understanding of the potential problems, with good personal discipline, reduced the incidence of injuries.
- Command decisions. There are military situations in which prevention of cold injuries has to play second fiddle to the exigencies of battle. There are other situations, however, in which overly optimistic or rash decisions have resulted in thousands of casualties and influenced the course of battle. Hitler's decision to invade Russia in the face of opposition from the general staff stands as a warning to all high command officers.
- Personal factors. Fatigue is related to nutrition and increases the possibility of cold injuries. Most of the US troops in the European theater in World War II were well fed, even though they sometimes spent many days on "K" rations. In contrast, many German units were very badly fed.[76]
- Previous cold exposure. Individual susceptibility to cold has been of interest to the military of many nations. In Japan, Watanabe[69] believed that he could distinguish between cold-susceptible and cold-resistant men. A first cold injury can lead to an increased chance of subsequent injury; and the more severe the first injury, the greater the chance of a second injury. In Italy in 1943 and 1944, 50% of cold-injured men returned to duty. But in 1944 and 1945, only 2.1% of injured men were returned to full duty because many men had recurrent injuries.[21] Men who had suffered once from trench foot were not able later to undertake long marches. Soldiers who had been frostbitten or had trench foot in Italy had an increased incidence of disability in the invasion of the South of France when the weather became cold. Of all men returned to duty after suffering trench foot, 15% had recurrent problems.[21]
- Race. There were no data to substantiate a view that racial differences in susceptibility exist. There were instances in which troops from Brazil and Hawaii had a high incidence of cold injury, but other factors such as training and experience may have been more important than racial back-

ground. Later data from the Korean War[77] suggested that black soldiers sustained more frostbite than white soldiers.

- Weather, temperature, and combat. While most cold injuries occur in cold weather, the more important factor is the influence of the weather and environment on heat loss. A cool, wet environment, maintained for several days, may be more damaging than a short, dry snap of very cold weather. Trench foot has an incubation period of 3 days. Frostbite develops in a few minutes to a few hours, and the colder the temperature, the shorter the period required to inflict damage.[21] (A detailed correlation of temperature and battle conditions enabled Whayne to pinpoint the days when trench foot or frostbite injuries were frequent, and to show that this often happened when weather conditions joined with intense battle conditions to increase the chances of injury.—B.C.P.)
- Clothing. The US Army researched the design of clothes for many environments.[58] The delivery of appropriate winter clothing did not always meet the needs of the troops, however, and the result was an increase in injuries in those units that did not receive winter clothing. In February 1945 a representative of the War Department was sent to the European theater to investigate the clothing situation. He reported that winter clothing had been supplied in insufficient quantities and was often too late in arriving.[21]

During the winter of 1944/45, units supplied with winter clothing in November had an incidence of only 4.3 per 1,000 men per year of trench foot in the following months. Units supplied later, in December, had an incidence of 10.5 per 1,000 men per year, and units still not equipped by January had an incidence of 11.5 per 1,000 men per year.[21]

The incidence of cold injury was much lower in British and Canadian units than in American units. The British 21st Army Group had fewer cold injuries during the European campaign than comparable American units fighting in the same areas.[21] Many factors, some of them small but perhaps significant, were thought to be responsible:

1. The British troops were well supplied with small hexamine stoves that enabled them to heat drinks and food when hot food could not otherwise be provided.
2. They drank, on an average, four cups of hot tea per day. Many American troops went days without hot food.
3. The rotation of British troops in and out of the line was more frequent, so that periods of rest and reorganization, during which the soldiers could change clothes and warm up, were more frequent than in the US Army.

In Italy it was the British policy to have troops spend a total of 400 days in the combat theater but with no more than 10 to 12 days continuously in the front line at a time. The American policy was to have troops spend 40 to 80 days at the front, but with only 200 days in the theater.

The woolen "battle-dress" of the British and Canadians was "indisputably superior,"[21] and their woolen-lined leather jerkins were a warm, windproof outer layer that could be worn in battle. Many British troops were supplied with rubber "Wellington" boots when serving in wet environments. The British also wore thick socks and loose boots that could accommodate two pairs of socks. Most American soldiers, perhaps based on their civilian tastes, tended to wear boots that fit snugly and would only take a single pair of socks.

In his final chapter, Whayne evaluated cold injury as a military problem and concluded that the cost of cold injuries cannot be weighed in dollars and cents, but by the number of men removed from active duty and the length of time they are out of commission (Tables 10-2 and 10-3). During World War II, the estimated time lost was 50 days per case, which calculates to a total of 2,757,300 man-days, or 7,579 man-years, lost. The loss was equivalent to a whole division's being eliminated for 6 months, or an army of 250,000 being out of action for 11 days.[21]

Staggering as these numbers are, they become even more important when we consider that cold injuries affect the frontline infantryman more than any other branch of the service. If the losses, based on Whayne's statistics,[21] are calculated in relation to an infantry division and not to the whole army, the population at risk is much smaller, and the losses proportionately greater. The cold injury losses in the winter of 1944/45 alone were equivalent to three divisions of 15,000 men each. As those lost were nearly all riflemen and a division has about 4,000 riflemen, the loss really amounted to the fighting strength of 12 divisions.

The lessons of the winter campaign in Italy in 1943 were only partially passed on to the commanders in Europe, although they were well understood by the medical corps. It is also possible that the initial victories and rapid advances in the European theater after

TABLE 10-2

FROSTBITE IN THE US ARMY DURING WORLD WAR II

Year	Geographical Area	Total Cases	Days Lost* (Total)	Average Duration† (Days)
1942	Total Army	1,021	11,360	11.1
	CONUS	717	7,880	11.0
	Overseas	304	3,480	11.4
1943	Overseas	665	10,402	16.1
1945	Total Army	11,510	553,420	46.8
	CONUS	385	4,610	12.0
	Overseas	11,125	528,810	48.0

CONUS: continental United States
* Includes days lost for readmissions
† Days excluded from duty
Adapted from Whayne TF. *Cold Injury in World War II: A Study in the Epidemiology of Trauma*. Boston, Mass: Harvard School of Public Health; 1950: 218. Thesis.

D day (6 June 1944) encouraged commanders to believe that the war would be over before the winter.

The number of men out of action at any one time varied from season to season and month to month, depending on weather and battle conditions. Between 30 December 1944 and 2 March 1945 there were never fewer than 20,000 men hospitalized or in rehabilitation, and another 1,000 to 6,000 similarly decommissioned because of frostbite,[21] many from the Ardennes, known as the Battle of the Bulge (Figure 10-13). For example, 28,042 men were affected by trench foot and 7,382 disabled by frostbite during the week of 3–9 February 1945. Calculated another way, and based on 4,000 riflemen per division, this was equivalent to sidelining the fighting strength of more than 8 divisions.

No commander can ignore numbers such as these. The need for adequate training, proper equipment, good discipline, and command awareness of the problem are self-evident. If any of these factors are missing, then weather, terrain, and battle—over which a commander has limited control—combine to ensure a high, and potentially disastrous, rate of casualties.

TABLE 10-3

TRENCH FOOT IN THE US ARMY DURING WORLD WAR II

Year	Geographical Area	Total Cases	Days Lost* (Total)	Average Duration† (Days)
1942	Total Army	32	220	6.9
	CONUS	—	—	—
	Overseas	32	220	6.9
1943	Overseas	489	5,167	11.0
1945	Total Army	24,555	1,380,160	56.7
	CONUS	110	3,985	36.2
	Overseas	24,445	1,376,175	56.8

CONUS: continental United States
* Includes days lost for readmissions
† Days excluded from duty
Adapted from Whayne TF. *Cold Injury in World War II: A Study in the Epidemiology of Trauma*. Boston, Mass: Harvard School of Public Health; 1950: 217. Thesis.

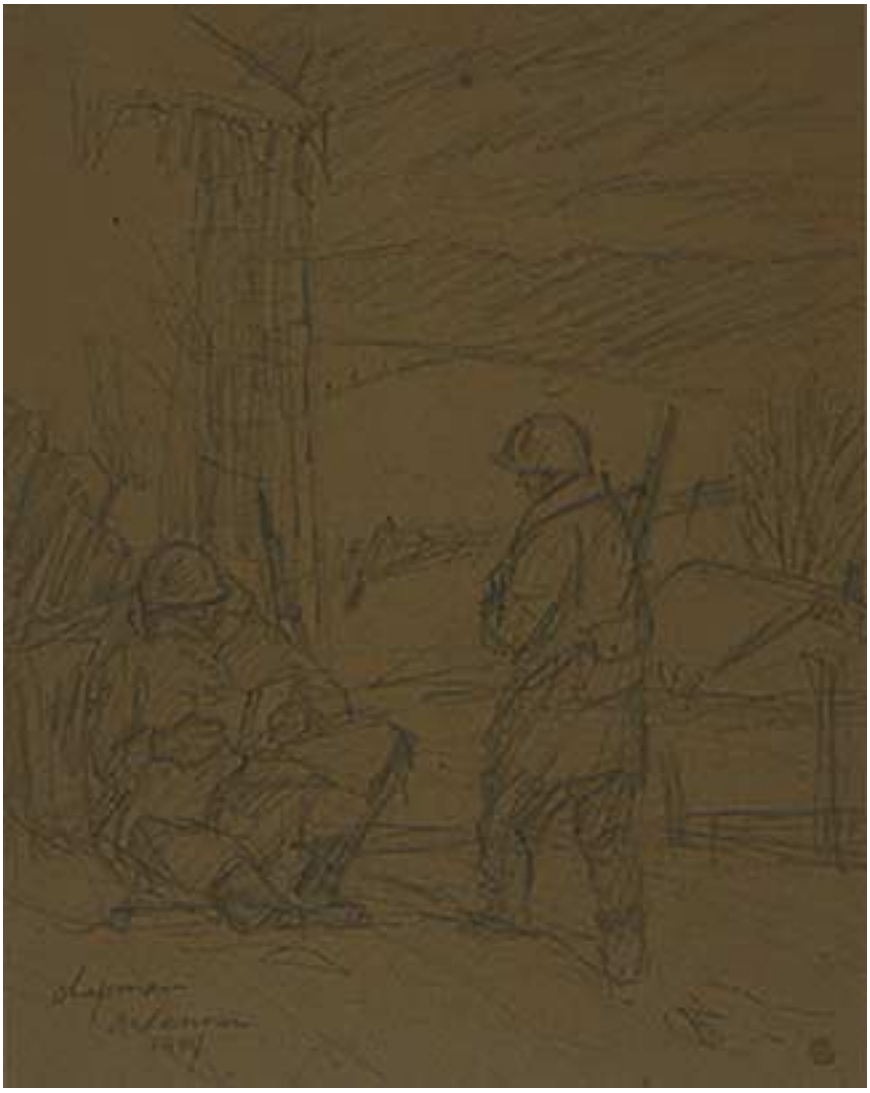

Fig. 10-13. *US Troops in the Ardennes, Winter 1944;* pencil sketch by Walter Chapman, 1944. This rough sketch, done on the spot, shows the cold conditions experienced by the US Army in the Ardennes Forest region in France and Luxembourg during the Battle of the Bulge in 1944 in World War II. One soldier, sunk into his greatcoat, sits reading, while another seems to walk, perhaps to keep warm, while standing guard. The artist, Chapman, worked for the Public Relations and Historical Section of the 84th Infantry Division in 1944 and 1945. Reproduced with permission from Peter Harrington, Curator, Anne S. K. Brown Military Collection, Brown University Library, Providence, Rhode Island.

KOREAN WAR: 1950–1953

The scientific knowledge acquired after World War II would soon be put to the test in the Korean War, where there were between 5,000 and 6,000 cases of frostbite. Many of them occurred during the first winter of the war, when the army was ill-equipped to deal with the extreme cold.[77]

During the night of 25/26 November 1950, 180,000 Chinese Communist troops swarmed across the Yalu River to hammer the right flank of the US Army, sending a flood of men through the gap between the 8th Army and X Corps. On 27 November, X Corps was hit by the advancing Chinese, who made a pincer thrust around the Chosin Reservoir. The 1st Marine Division was surrounded and isolated, and, in what was called "attacking in a different direction," fought their way out of the trap to join the 3rd Division. The Marines had thousands of frostbite injuries.[78]

In February 1951, Captain Norman Allen of the 5th Cavalry wrote:

> The weather is terrible; cold wind and driving rain. I was soaked to the skin for two days and nights and just froze. Despite the weather the lads fought hard.[79(p83)]

And Private First Class James Cardinal, in the same unit, wrote home:

> It's a cold gray day and the wind chills me to the bone … and the cold hurts them [the Chinese] much more than us.[79(p83)]

In a classified report written in 1951 and later declassified, S. L. A. Marshall[78] critiqued the actions of the Chinese Communist forces and also commented on the effects of cold. He held that the 1st Marine Division was as well prepared for cold-weather fighting as any US division could be with existing weapons and equipment. Strong discipline and the availability of warming tents maintained the fighting efficiency of the men. The wounded were kept in warming tents; men suffering from extreme shock or exhaustion from the cold were similarly kept in warming tents for 24 hours before returning to their units. Of the 2,700 nonbattle casualties, 2,000 (74%) were cases of frostbite, of which 95% affected the feet. A careful study indicated that only 20% of the frostbite injuries were due to carelessness and 80% were due to the conditions of battle.

Many men became sick from eating half-frozen C-rations, and the rations did not supply the nutritional needs of troops in combat under conditions of extreme cold, conditions that may have contributed to some injuries.[78]

Medical officers noted a shocklike state in hundreds of men due to the cold and unrelated to other battle stresses.[78] This condition was similar to that described 140 years earlier by Larrey.[15] Respirations were suppressed and stumbling men stared, unresponsive, into space; some sobbed. Warmth and a shot of brandy restored them to normality. After a few days of fighting in these extreme conditions this state of shock disappeared as though the troops had, in some way, become acclimatized physically or psychologically to the conditions.

The shoepac was condemned because it was impermeable to moisture, and excessive sweating during marching froze when the troops came to rest. Ice formed within the shoes and battle conditions made changing socks—the only effective treatment—impossible. Weapons were affected by the cold, and the Marines cleaned their weapons in gasoline rather than with oil, which congealed and rendered weapons useless.[78]

The US Army Medical Corps was prepared to study what had happened and, in 1952, Orr and Fainer outlined the injuries and their two-phase treatment[77]:

1. Emergency. Rapid rewarming was known as an effective treatment but was not feasible under the conditions of battle. Only 2.1% of the casualties were seen before blisters developed, and the majority of limbs had thawed spontaneously before treatment could be started.
2. Hospital. Treatment included bed rest, no smoking, room temperature at 70°F to 78°F, bullae left intact, and penicillin.

Many adjunctive methods were used, but in their report, Orr and Fainer stated that no specific therapeutic agents or procedures conclusively reduced the tissue damage, shortened the process of healing, or prevented sequelae. Parts that became cold and remained cold were lost, and parts that were vesiculated were usually not lost. Nonvesiculated areas distal to the vesicles and bullae were frequently lost.[77]

A follow-up paper[80] demonstrated a high incidence of late symptoms that included hyperhidrosis, deformities, and sensitivity to cold, and radiological evidence of damage to the bony surfaces of small joints. The survivors of the battle at the Chosin Reservoir still meet and many have residual symptoms and damage from their injuries sustained during the Korean War (see Chapter 14, Clinical Aspects of Freezing Cold Injury).

FALKLAND WAR: 1982

This description of the Falkland Islands, from a guide to the birds of the islands, can hardly have been in the minds of the British Royal Marines and troopers of the Parachute Regiment landing at San Carlos on the west coast of East Falkland on May 21st, 1982:

> The land appears bleak due to the absence of native trees, but the white sand-beaches, brilliant green grass around ponds or the coastal clumps of tussock-grass, contrast pleasantly with the wind-

> swept uplands. … The silence, broken only by the cries of animals and birds and the thunder of the surf, the solitude, and the strong, cool winds, combine to give a sense of exhilaration and freedom.[81(p14)]

Although the fighting stopped less than a month later, it left behind a huge legacy of cold injury in the armies of both Great Britain and Argentina.

The battlefields were boggy moors where trenches and foxholes were dug—when they could be dug—in oozing peat. In the assault on Mount Longdon, British Royal Marines and Paratroopers marched 50 mi in 3 days across rough, wet terrain. Their feet were never dry and there was no opportunity to rest, dry their feet, or put on dry socks. The high incidence of injuries affected the fighting ability of the entire force. Many of those injured still suffer from residual symptoms. The Army of Argentina did not keep statistics, but it can be presumed that they suffered as many, or more, cold casualties, because many of their units were stranded without supplies and without adequate cold-weather clothing. For further information, interested readers should also see Chapter 15, Nonfreezing Cold Injury, especially Exhibits 15-1 and 15-2.

TRENDS IN MANAGEMENT

Until the end of World War II, the management of cold injuries had remained remarkably unchanged for more than 100 years. But during the war, research in many countries had demonstrated the usefulness and importance of rapid rewarming of frozen limbs. Within the space of a few years, rapid warming became the treatment of choice. Later, basic laboratory research, growing out of an increasing understanding of inflammation, wound healing, and reperfusion injuries, transformed our ideas about the pathology of frostbite.

Rapid Rewarming in the Field

Until Fuhrman and Crismon's work in the late 1940s,[63] rapid warming was generally thought by authorities in North America and Western Europe to be harmful. There seemed to be more pain, swelling, and subsequent tissue loss if the frozen body part was warmed quickly.

The 1950s and 1960s were a golden era in frostbite research. The US defense organizations, motivated by the possibility of war against the USSR, invested heavily in research and produced numerous reports on the pathology, physiology, and treatment of frostbite. As early as 1952, rapid rewarming had become the official method for treating frostbite. The North Atlantic Treaty Organization handbook, *Emergency War Surgery*,[82] published that year, instructed:

> If the injured parts are still frozen they should be rapidly rewarmed by immersion in water at 90–104°F, by placing warm hands on the part or merely by exposure to warm air. Walking, massage, exposure to an open fire, cold water soaks, or rubbing with snow are contraindicated.[82(p43)]

In the 1940s and early 1950s there was little clinical evidence to back up the laboratory work, and there was criticism that animal experiments did not imitate the clinical situation with sufficient accuracy. Most experiments had been done with very rapid, deep cooling of small areas in small animals—rabbit ears or feet, rat tails or feet—with immediate warming, a situation quite different from clinical frostbite. In the early 1960s, William J. Mills, Jr., an orthopedic surgeon in Anchorage, Alaska, who had been a student at Stanford University, in Palo Alto, California, under Professor Fuhrman and later served in the US Navy during World War II, had a unique opportunity to see many cases of frostbite. A succession of clinical reports and conference transcripts, backed by an ever-increasing number of treated patients, confirmed that rapid warming of a frozen part resulted in a better outcome than slow or spontaneous warming.[83]

Many factors influence the possibility and severity of damage, and numerous therapeutic methods were tested: anticoagulants,[84] hyperbaric oxygenation,[85] medical or surgical sympathetic blockade,[86,87] vasodilators,[88,89] antispasmodics,[90] agents to affect viscosity,[91] steroids[92] and other antiinflammatory agents,[93] and compression[88,93] to reduce edema. None were shown to be consistently successful.

The best technique, which is essentially followed to this day, was warming of the frozen limb in water at 42°C to 48°C for about 20 to 30 minutes; followed by careful drying; care in a sterile environment; daily whirlpool bath debridement; and late, conservative surgery.[83] Mills discovered both what was and what was not effective. In a report in 1960 of 51 patients, 21 treated with rapid warming and 30 by other methods, he warned that of all the factors analyzed in the treatment of frostbite that might influence the result, premature surgical intervention was the greatest contributor to a poor result.[83]

There have been few fundamental changes in the management of frostbite since the late 1960s. The work of Heggers and associates[94] improved our understanding of the underlying pathophysiology and altered treatment, but very little evidence has been produced that these changes have resulted in significantly fewer casualties.[95]

Recent developments in the understanding of frostbite as a reperfusion injury may, in the long run, change methods of treatment and even provide protection against injury. With an increase in knowledge of reperfusion injuries, inflammation, and immune responses, improved therapeutic methods will undoubtedly be developed that may improve the field treatment of both frostbite and immersion foot.[96]

The field management of hypothermia still remains difficult, because effective methods for rewarming safely in the field have yet to be developed.

Warm Water Immersion Foot Syndromes

The exact temperature at which true cold water immersion foot develops has never been precisely defined. During World War II in the Pacific theater, a medical officer named Frank Glenn, who later became Chairman of Surgery at Cornell Medical School, New York, New York, examined 120 men in Leyte, Philippines, within 12 hours of their admission to a medical treatment facility, all with a condition that he diagnosed as immersion foot.[97] In the Vietnam War similar injuries were found in men who had been slogging through paddy fields and jungles for protracted periods, their feet constantly wet. Although there were similarities between this condition and cold water immersion foot, it became apparent that the two conditions were not the same. The one seen in Vietnam, called tropical immersion foot (TIF), affected only the feet and ankles and was quickly cured by a few days in a dry environment. There were no serious complications, such as gangrene, that necessitated amputation.[98]

A similar syndrome, warm water immersion foot (WWIF), was also identified. The stratum corneum of the sole of the foot became waterlogged, turning the skin into a pale, soggy, painful layer. Walking became difficult and men had to be evacuated from the field because of it. Cure, however, only required 1 to 3 days in a dry place.[98]

LESSONS FROM THE PAST, IMPLICATIONS FOR THE FUTURE

Early historians related that many soldiers died or were frozen but did not describe how cold injuries were treated. Frozen feet were warmed at a fire. Soldiers with mild frostbite and hypothermia probably survived, but many others died. Modern armies are still being exposed to the risk of cold injury. No statistics are available from the fighting in Bosnia and Yugoslavia during the late 1990s, but the winter conditions, the nature of the battles, and the inadequacy of medical supplies must have resulted in large numbers of cold injuries and limbs lost.

In Northern India and the adjacent area of Pakistan, a high-altitude war has been fought intermittently for several years, extracting a heavy toll of frostbite and high-altitude illness. This war has been fought in the highest, most inhospitable environment faced by any military, with troops regularly stationed as high as 20,000 ft. It is likely that there have been more casualties from the environment than from enemy action.

General Carl Tiedman of the Norwegian Army analyzed the causes of the disaster that overtook Charles XII of Sweden,[99] and the lessons learned from the mistakes of that campaign are as relevant today as they were in 1719:

1. The campaign was started in the autumn, in the belief that it could not extend into the winter. Circumstances changed and disaster struck. Commanders should anticipate that a campaign will last longer than anticipated.
2. Provision of cold-weather clothing, tentage, and shelter was inadequate.
3. The supply lines were long, overextended, and easily attacked.
4. The troops, mostly Finns from flat country, were accustomed to cold weather but not to mountain warfare. Their training and experience were no match for the conditions of battle. Special training is necessary for troops to fight successfully in winter conditions. They must be trained psychologically and physically, know how to prevent cold injuries, and how to maintain their weapons.
5. The campaign had not been going well and the troops, cold and ill-fed, had to find their own food by raiding the local villages. The death of the king caused a catastrophic collapse of troop discipline and was the final blow to morale. Low morale, despair,

and defeat are certain precursors for cold injuries. Victory, discipline, and high morale are the best protection against frozen limbs.

6. While the army was retreating it was constantly attacked from the flanks, causing further disorganization, increased casualties, and loss of supplies. A retreating, chaotic army inevitably sustains heavy casualties.
7. The retreat was over high, bare passes without trees (and, therefore, without fuel for making fires) and shelter. The combination of cold, snow, and wind; hostile terrain; poor leadership; inadequate supplies; and defeat was a prescription for disaster.

Commanders of every era have made the same mistakes. Sometimes those mistakes have been unavoidable, sometimes they could have been prevented; but the lessons of the past cannot be ignored. The commander of today, however, has to consider medical factors that never troubled the army of Charles XII. The modern serviceman expects to be supported by a quick, efficient medical service. In the harsh environments of winter warfare, special arrangements are necessary to maintain the integrity of the medical service. The means to rewarm casualties with frostbite or hypothermia should be close to the front line; the risks of damage and death are increased by delays in treatment. The casualty with a cold injury, possibly combined with a wound, must be transported to a medical treatment facility in a warmed vehicle, whether by land or air. Helicopter evacuation may not always be possible in winter conditions; special overland vehicles should be available.

Success in winter warfare requires special training and preparation, not only in the techniques of fighting in snow and ice but also in protection against the elements. The individual soldier must be psychologically prepared to cope with the unrelenting harshness of winter—cold, wind, blizzards, deep snow, or steel-hard ground that makes it impossible to dig a shelter for protection from either the weather or the enemy. Cold, even above the definitive level for hypothermia, has a peculiarly depressing and inhibiting effect on resolve and activity, an effect that can only be reversed by reaching warmth. If he is to maintain fighting efficiency and survive, a freezing soldier must draw on deeper wells of courage, determination, and discipline than one who is warm.

Officers at the highest levels of command must understand the logistical and medical problems of fighting in the cold, so as to avoid the unrealistically optimistic outlook that caught both Napoleon and Hitler in an icy trap. To ensure that equipment and clothing will be ready when needed, they must be available in quantities that in peacetime seem to be unreasonable. Special over-the-snow vehicles, for instance, may have to be designed and manufactured, yet they will seldom be used in peacetime. Medical facilities require heating and insulated shelter that are unsuitable for use in tropical climates. History has repeatedly shown that a failure to make preparations will, eventually, lead to defeat.

The history of cold in war is a tribute to the determination and endurance of the human body and spirit. Despite hostile environments, poor leadership, lack of food and shelter, the wrong clothes, frozen weapons, and the myriad tribulations of moment-to-moment survival, soldiers of all nations and in all eras have fought in storms and freezing cold. Sometimes they were victorious, sometimes defeated, but always winter was neutral, an impassive onlooker to be used by the prepared and ready to destroy the unprepared.

NOTE: On 3 July 1951, Colonel Tom F. Whayne delivered a lecture, entitled "Cold Injury," as part of the Medical Service Officer Basic Course at the Army Medical Service Graduate School, Army Medical Center (now Walter Reed Army Medical Center), Washington, DC. In his lecture, Colonel Whayne analyzed the available medical information on the effects of cold on military operations in selected wars from the Revolutionary War to the end of World War II, emphasizing those in which US fighting forces were involved. The text of the lecture has been available only as a mimeographed handout distributed to a relatively few students, or bound, with other lectures delivered during the course, in a volume housed in the main library at Walter Reed Army Institute of Research, Washington, DC.[100] The editors of this textbook believe that the Whayne lecture deserves a wider readership; it is reprinted in its entirety as Appendix 2 to Volume 3 of *Medical Aspects of Harsh Environments*.

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Chapter 11

HUMAN PHYSIOLOGICAL RESPONSES TO COLD STRESS AND HYPOTHERMIA

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INTRODUCTION

Conducting military training or combat missions in cold environments poses a dual challenge: protecting the personnel from hypothermia and other forms of cold injury while also realizing the objectives of the mission. The goals of this chapter are to describe the components of the thermoregulatory system that influence peripheral and core temperature in cold environments, how the components respond to a cold environment, and what subsequently occurs when they are unable to maintain core temperature. In this chapter, we discuss the workings of the thermoregulatory system and emphasize the effects of hypothermia from the perspective of field medical practice.

Three major categories of hypothermia are recognized, based on the environment in which the hypothermia occurs (eg, air, water, high altitude), and on the physiological status of the threatened individual. Regardless of its origin, hypothermia is defined as a 2°C decrease in core temperature.[1] Normal core temperature is usually considered to be 37°C for brain, heart, and lungs. Although the clinically defined value for hypothermia is 35°C, this must be considered a rough estimate. Some subjects who experience a rapid decrease in core temperature might have a 36°C core temperature and demonstrate signs of hypothermia that are usually associated with a much lower core temperature. Regardless of the precise core temperature, hypothermia eventually causes a decrease in metabolic rate; this may allow the entire body to survive very cold temperatures and hypoxic states, and be rewarmed without any long-lasting debilitation. The coldest core temperature from which a person has been successfully rewarmed is 15°C.[2] From a military perspective, hypothermia will cause a decrease in overall effectiveness of the casualty, but, paradoxically, the decreased metabolic rate will allow for a much greater time in which the casualty can be rescued.

Hypothermia is classified as primary, secondary, and clinically induced (iatrogenic).[3] *Primary* hypothermia refers to the condition in which the casualty has normal thermal regulatory responses, but these are ineffective against the environment (Figure 11-1). This condition may be seen in any cold weather training scenario (eg, US Navy SEALs undergoing cold water exercises). Although the participants are physically fit, the cold environment will eventually overwhelm their physiological defenses and they will develop hypothermia.

Secondary hypothermia is caused by impaired thermoregulation, the result of an altered physiological state that may be caused by illness, fatigue, or injury. Impaired thermoregulation causes a disproportionately greater effect on the casualty's ability to tolerate cold (ie, cold environments will induce a decrease in core temperature). Secondary hypothermia may explain many of the cold weather casualties in previous wars. In these situations, the troops were fatigued and had insufficient food, clothing, and fluid. Consequently, the cold environment overcame their physiological responses, and hypothermia ensued. For example, Hannibal, Napoleon, and Hitler all experienced major losses during campaigns in cold weather; a majority of their cold-induced losses probably had secondary hypothermia. An injured, fatigued, or sick soldier in a cold environment can easily develop secondary hypothermia. A soldier who has lost blood and is dehydrated will not be able to respond adequately to the temperature challenges of a cold environment. Any environment that is cooler than the body will promote body cooling (even 70°F or 80°F). Thus, hypothermia can occur in deserts or jungles if an individual is dehydrated, fatigued, or injured. The time for the onset of hypothermia depends on a large number of factors: clothing, body size, metabolic rate, physiological state, hydration, and nutritional status. In military situations, the onset of hypothermia is insidious. It occurs gradually and poses a major threat for completion of military operations.

Clinically induced hypothermia, on the other

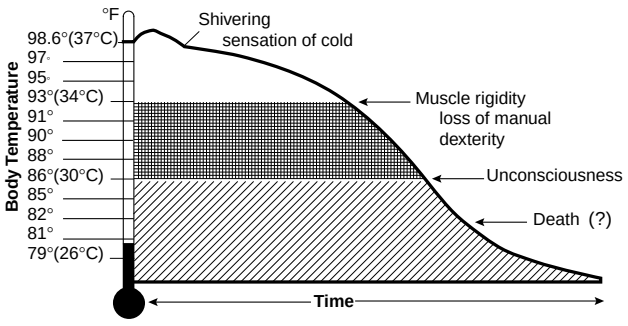

Fig. 11-1. Core temperature decrease leading to hypothermia. The diagram demonstrates the schematic representation of the fall of core temperature with both primary and secondary hypothermia. The question mark by the term "death" refers to the life-sparing property of hypothermia—in certain conditions.

EXHIBIT 11-1

HYPOTHERMIA-INDUCING ENVIRONMENTAL AND PATHOLOGICAL CONDITIONS

Increased Heat Loss
- Environmental
  - *Immersion
  - *Nonimmersion
- Induced Vasodilation
  - *Pharmacological
  - *Toxicological
- Erythrodermas
  - *Burns
  - Exfoliative dermatitis
  - Ichthyosis
  - Psoriasis
  - Iatrogenic factors
    - Cold infusions
    - Emergency childbirth
    - Heatstroke treatment

Decreased Heat Production
- Endocrinological Failure
  - Diabetic and alcoholic ketoacidosis
  - Hypoadrenalism
  - Hypopituitarism
  - Hypothyroidism
  - Lactic acidosis
- Insufficient Fuel
  - *Extreme physical exertion
  - *Hypoglycemia
  - *Malnutrition
  - Kwashiorkor
  - Marasmus
- Neuromuscular Physical Exertion
  - Lack of adaptation
  - Extremes of age
  - Impaired shivering
  - Inactivity

Impaired Thermoregulation
- Peripheral Failure
  - Neuropathies
  - *Acute spinal cord transection
  - Diabetes
- Central Neurological Failure
  - Cardiovascular accident
  - *Central nervous system trauma
  - Metabolic cause
  - Toxicological cause
  - Pharmacological cause
  - Anorexia nervosa
  - Cerebellar lesion
  - Congenital intracranial anomalies
  - Hyperkalemic periodic paralysis
  - Hypothalamic dysfunction
  - Multiple sclerosis
  - Neoplasm
  - Parkinsonism
  - Subarachnoid hemorrhage

Miscellaneous Associated Clinical States
- *Multisystem trauma
- *Recurrent hypothermia
- *Episodic hypothermia
- *Infections (bacterial, viral, parasitic)
- Carcinomatosis
- Cardiopulmonary disease
- Giant cell arteritis
- Hodgkin's disease
- Paget's disease
- Pancreatitis
- Sarcoidosis
- Shaken baby syndrome
- Shock
- Sickle-cell anemia
- Sudden infant death syndrome
- Systemic lupus erythematosus
- Uremia
- Vascular insufficiency
- Wernicke-Korsakoff syndrome

*Major cold weather challenges to military operations
Adapted with permission from Danzl D, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies.* 3rd ed. St Louis, Mo: Mosby–Year Book, Inc; 1995: 59.

hand, is defined as a decrease in core temperature that is induced for various surgical procedures such as coronary bypass. This chapter will not further discuss iatrogenic hypothermia but instead will emphasize primary and secondary hypothermia as it relates to military operations.

Because various environments and physiological conditions influence the development of hypothermia and the effectiveness of rewarming, there are various etiological factors for secondary hypothermia that are more commonly associated with military operations (eg, extreme physical exertion, multisystem trauma). Others are more frequently found in military rescue operations, in which American troops rescue civilians, particularly those at the extremes of age, who are in extreme hostile environments and are suffering from conditions such as dehydration and malnutrition (Exhibit 11-1).

Owing to the lessons of history, most modern military forces have adopted various measures to counter the development of hypothermia. Much of their research has focused on various clothing and energy-rich diets. Other research studies have been conducted to understand the interplay of the various components of the thermoregulatory system: the peripheral receptors, which communicate with the brain via the spinal cord; and the cardiovascular, respiratory, renal, circulatory, gastrointestinal, endocrine, and immune systems. One of the goals of this kind of research is to develop a "predictive model" that will help predict the onset of hypothermia, and more importantly, when a decrement in performance, or even death, will occur. Such a task is daunting because the model must include the physical factors that determine heat transfer as well as the effects of sleep deprivation, dehydration, the lack of food, and the like. Some of the challenges that are yet to be addressed by the model are the effects of psychological underpreparedness in a cold environment as well as any major gender differences. Studies by Hodgdon and colleagues[4] suggest that if a person is adequately prepared (eg, proper clothing, well hydrated, etc), then cold stress leading to hypothermia will not occur. Models are only validated to core temperatures that relate to mild hypothermia and as such cannot predict death with any scientific precision. It is ethically impossible to conduct such experiments. Thus, modeling is important but it cannot be a valid predictor of various stages of hypothermia.

In modern times, as in previous battle campaigns, hypothermia in military training or combat operations is not due to the underpreparedness of the operations but rather to unforeseen situations. During the Falkland War, the British suffered many cold-associated injuries because their timetable for capturing various strategic positions was completely altered, owing to the effectiveness of Argentine sharpshooters. The sharpshooters kept many British soldiers in very cold environmental conditions for much longer than the 1 hour that the timetable had called for.[5]

CORE AND PERIPHERAL TEMPERATURES

The best method to assess the degree of hypothermia and initiate appropriate medical intervention is by measuring the core temperature. Unfortunately, the technologies are not yet available for accurate measurements in the field. In most cases, the core temperature is controlled relative to the changes in peripheral temperature. Peripheral and core thermal receptors send important information to the central nervous system, which mediates all the cold-induced psychological and physiological responses.

Core Temperature Measurements

The measurement of core body temperature is necessary to assist the medical officer in the care and management of the hypothermic casualty, as well as to be the critical measurement in various scientific studies (eg, effectiveness of rewarming methods or different protective suits). In battlefield situations, measuring core temperatures is not practical. However, because the results of laboratory human studies are used to persuade military commands to make various decisions (eg, to purchase one form of heating systems or another), it is important that accuracy of the different core temperature monitoring systems be presented. Also, in the event that advances in technology allow for the monitoring of core temperatures in the field, the strengths and weaknesses of various anatomical sites to accurately reflect core temperature need to be recognized. Unfortunately, skin temperature cannot be used as a surrogate for core temperature. The sites that have been commonly used are oral, rectal, axillary, tympanic, and esophageal. The advent of easy-to-use tympanic temperature devices has sparked their widespread use for measuring core temperature. In thermally stressful environments, however, the tympanic temperature is not an adequate reflection of core temperature.[6] In a recent study, three different infrared detectors were com-

pared against esophageal temperature in subjects who were made hypothermic by cold water immersion. The temperatures registered by the three different infrared devices were 1.06°C lower than the other core values in subjects whose hypothermia ranged from 36.5°C to 33.3°C. The major reason for this discrepancy is that in all three devices, the cone of the infrared detector was too large to get an accurate reading of the tympanic membrane.[7]

Besides the technical problem, the tympanic temperature is influenced directly by the temperature of the venous blood of the face and indirectly by the temperature of the environment surrounding the head. In any situation in which the skin temperature of the face is being altered by the environment, the tympanic temperature reading will be false with respect to the core temperature.[8] Livingstone and colleagues[9] were able to show that cooling the face decreased tympanic temperature. In a defining experiment, McCaffrey and colleagues[10] showed that cooling or heating small areas of the face altered tympanic temperatures. Application of a bag of cold 3°C to 4°C water to an area of the right cheek and orbit results in a fall of tympanic temperature on the right side, while simultaneously, tympanic temperatures rose on the left as a bag of hot (45°C–50°C) water was held against the left cheek. Although the ease of use of tympanic temperature is enticing, its use in field situations is not recommended because environmental influences on the face can render the readings unreliable.

The best measurement of core temperature is taken at the esophagus, but it is extremely difficult to get compliance from subjects (for inserting a small-diameter cable down the nose and throat) at that site.[11] The axilla is not a good site for measuring core temperature because readings are variable, depending on the subcutaneous fat of the subject and the placement of the sensor. Oral temperature may be as accurate as rectal[12] but varying kinds of breathing patterns of subjects, as well as the potential for subjects to bite on the thermometer, preclude its use in the field. For most purposes, the rectal temperature is considered the most practical and accurate measurement; however, it lags behind esophageal temperature.[11] Measuring core temperature by monitoring urinary temperature, as an indirect measurement of urinary bladder temperature, is one way to get a reading on the core temperature of the body in a field situation. Urinary bladder temperature in certain situations closely correlates with pulmonary artery temperature, but in bypass operations it was below nasopharyngeal temperatures.[14]

Peripheral Temperature Sensors

The maintenance and control of core temperature depends on the interplay of two different temperature-sensing systems: the peripheral and the core. The peripheral sensors provide the body its first line of physiological information. Depending on the differences in temperature that the central nervous system (CNS) differentiates between the periphery and the core, various physiological responses will occur (Figure 11-2). Many of the initial responses to a cold environment are triggered by the peripheral thermal receptors of the skin. The sensation of cold is initially triggered by stimulation of specialized nerve endings called cold receptors. When these receptors are activated, they send electrical signals (ie, action potentials) to the brain that are then interpreted as a cold sensation.[15] In addition, these action potentials will trigger various psychological and physiological responses.

There are two groups of cold receptors, superficial and deep, of which approximately 60% are in the periphery. The arrangement of superficial and deep cold receptors suggests that cutaneous cold receptors measure the temperature gradient within the skin.[16] The response of humans to cold sensation is not purely due to the action of cold receptors. Warm and cold receptors have a bell-shaped frequency-to-temperature relationship, and some overlap with each other between the temperatures of 30°C and 40°C.[17] Although both warm and cold receptors have a tonic firing pattern, they respond to their specific stimuli. In other words, a cold receptor will respond to a cold stimulation with a transient excitation and then stay constant, but it can be inhibited by warming. Thus, the practical solution of warming hypothermic victims with warm blankets can mislead both the victim and the medical practitioner. The warm blankets will inhibit the cold receptors from firing, which will lead to a decrease in various physiological responses triggered by the cold, such as vasoconstriction and shivering. Once these responses are abated the rescuers may mistakenly assume that the person is no longer hypothermic. Without measurement of the core temperature, this false impression may lead to the mismanagement of the hypothermic victim.

The cold receptors transmit information on small myelinated fibers at 5 to 15 m/s and on C fibers.[17] There is a constant rate of discharge between 25°C to 33°C. Interestingly, cold receptors also demonstrate a paradoxical discharge between 40°C and 45°C. This discharge is dependent on body temperature. At core temperatures of 37°C and 39°C, the

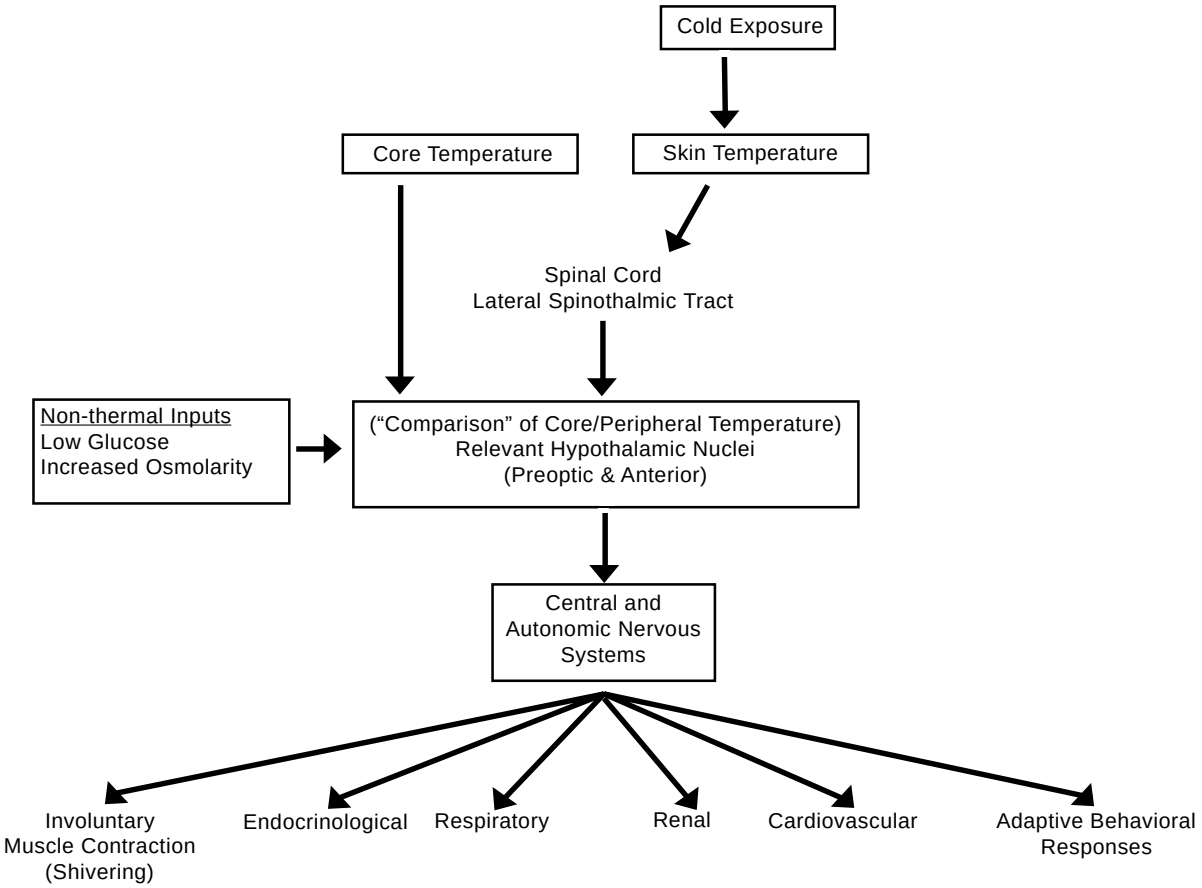

Fig. 11-2. Physiological schemata for thermoregulation in cold environments. The diagram illustrates the roles that peripheral and core temperatures play in driving various coordinated thermoregulatory responses.

cold receptors fire, respectively, at 55°C and 46°C. These data are used to explain the phenomenon of warm stimuli triggering a cold sensation. Once the cold receptor has fired, it rapidly adapts to a new static discharge.[15] Cold receptors, when activated, demonstrate a bursting pattern of doublets or triplets. The interburst interval, burst duration, and number of spikes within a burst all increase monotonically with decreasing temperatures.[16,17] This mechanism might explain how specific information from the cold receptive fields is interpreted in the brain. The mechanism of how the burst frequency is determined can be inferred from a number of studies in which sodium and potassium adenosine triphosphatase activity is inhibited, suggesting that there is an oscillating generator potential at the receptor site that triggers a burst of impulses when a certain threshold is exceeded.[17]

The signals from various afferent fibers enter the spinal cord at two levels: the first, the trigeminal (the face),[18] and the second, the superficial laminae of the dorsal horn (the rest of the body).[19] Where do these signals eventually terminate? The signals do not seem to traverse the spinothalamic pathway but probably ascend in the nucleus raphe, and then diverge to the sensory thalamic nuclei and the regulatory hypothalamic areas. Both sets of cold fibers project directly onto the thalamus, where the signals are initially interpreted.[20,21] (Interestingly, we are not able to accurately detect our own core temperature. Subjects can sense that they are getting cold, but there is no correlation between core temperature and perception of cold temperature.)

Certain descending pathways from the brain also influence the ascending signals from the cold receptors.[18,19] This area is not yet well studied. These descending pathways may be the key to our understanding of why certain individuals are not bothered by cold environments, because these descending pathways may influence the firing of the cold receptors.

THERMOREGULATION: THE BRAIN

The body's reaction to cold stress is controlled by the CNS, which can be likened to a central computer that controls all physiological systems. However, the brain itself can be cooled, which affects its own viability as well as its ability to control the various systems in terms of thermoregulation. The effects that cold environments have on the brain are multiple, but the area with the greatest interest deals with the hypoxic sparing effect that cold temperature has on brain function. The areas of brain thermoregulation covered in this chapter that compromise military operations are those dealing with motor control and circadian rhythms and sleep.

Hypothalamus

The incoming signals from the skin and visceral afferents will influence the hypothalamus (a major thermoregulatory control site), which will then trigger various thermoregulatory physiological responses. In engineering terms, certain parts of the hypothalamus are considered to be the thermostat of the body and will either increase or decrease core temperature by triggering behavioral and physiological responses. Also, the hypothalamus itself responds to temperature changes in the brain. When the temperature in the rostral part of the hypothalamus is changed, several thermoregulatory responses can be evoked. The preoptic-anterior hypothalamus contains neurons that (1) respond to the temperature in the brain and (2) receive input from the thermoreceptors from the skin and spinal cord.[20,21]

The hypothalamus contains three kinds of neurons: cold-sensitive, warm-sensitive, and temperature-insensitive. The thermosensitive neurons will increase their firing if the temperature changes.[22] All the responses that occur with the initial exposure to cold or a drop in core temperature, or both, are in many ways dictated by the hypothalamus and other CNS sites. That is to say, the hypothalamus is not the singular site that controls the thermoresponse to cold, because the spinal cord has been shown to be another site.[23,24]

The system is even more sensitively programmed, in that certain neurons in the hypothalamus

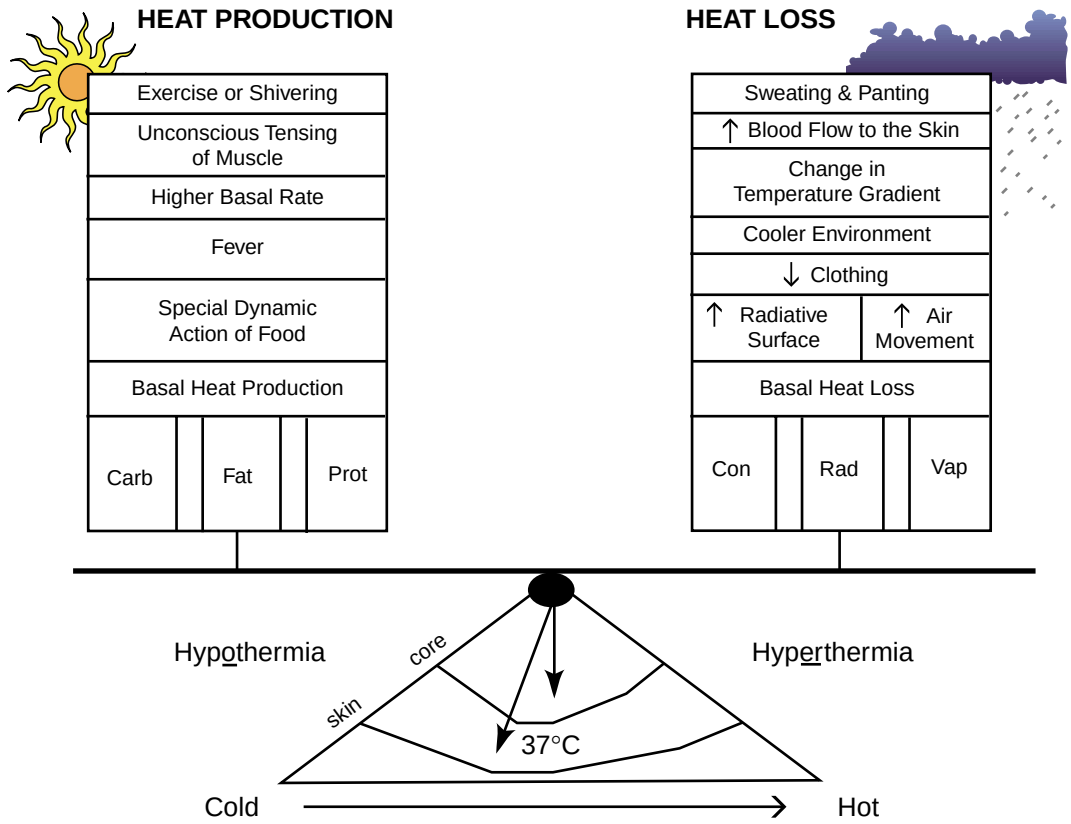

Fig. 11-3. Balance between heat production and heat loss mechanisms. The drawing illustrates the interaction of skin and core temperatures relative to internal and external factors. Carb: carbohydrates; Prot: proteins; Con: conduction; Rad: radiation; Vap: evaporation.

that respond to cold stimuli also respond to certain chemical changes. For example, when the hypothalamic neurons are exposed to low glucose or increased osmolality,[25,26] the cold-sensitive neurons fire. These nonthermal signals may partially explain the observations, recorded during training operations, of personnel complaining of being cold when they are actually dehydrated or hungry.

When the thermoregulatory system is activated, there will be a number of efferent responses such as an increase in heart rate, peripheral vasoconstriction, tensing of muscles, and higher metabolic rate caused by the release of various hormones. The metabolism is also regulated by the hormones and neural systems that regulate core temperature. The intake of food will also play a major role in maintaining and enhancing the metabolic rate of the hypothermic subject. Figure 11-3 demonstrates the interrelationships between those physiological systems that will produce heat and those environmental and physiological systems that will cause a decrease in heat loss. The diagram demonstrates that skin and core temperatures are independent of each other and that to maintain core temperature requires a balancing act between heat loss and heat production. It also demonstrates the interrelationships among core temperature, skin temperature, and the various physical and physiological factors associated with thermoregulation. This diagram presents a simplistic version of thermoregulation, because the physical processes of conduction and radiation can be used either to heat or to cool subjects.

Cold-induced peripheral vasoconstriction is the first major physiological response to a cold environment. This response is mediated by the autonomic nervous system and elicits the sensation of cold. Vasoconstriction proceeds in a distinct physiological manner, from the tips of the digits to the central part of the hand (Figure 11-4). Over 4 minutes, the tips of the fingers become more vasoconstricted and, therefore, cold, until eventually even the palm is cold. The exquisite control of the autonomic nervous system in controlling blood flow is shown in Figure 11-5, in which one hand has just been removed from a glove, whereas the other has been in a cold room. Notice also that the face is cool except for the forehead and area adjacent to the nose. The same phenomenon occurs in the feet (Figure 11-6) and the rest of the body. Many physiological and pharmacologi-

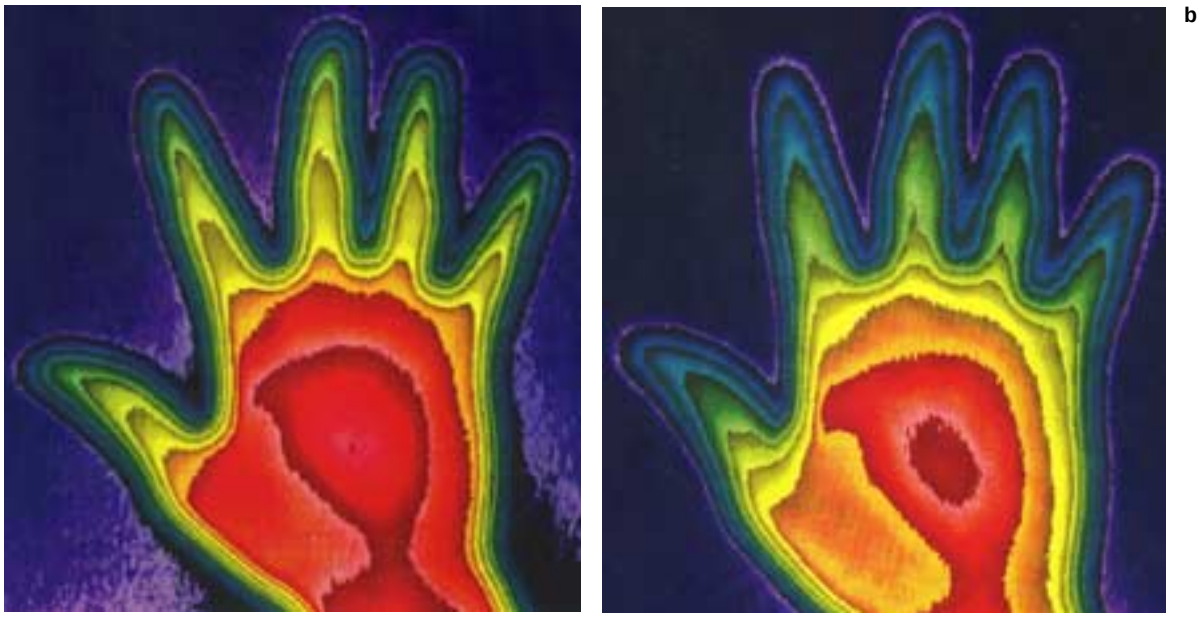

Fig. 11-4. These infrared images of a left hand in a cold environment demonstrate (**a**) the initial vasoconstriction of the hand exposed to a cold air environment (9°C). To the left of the image, the thermal scale ranges from 9.43°C to 35.68°C. Note the segmental nature of the vasoconstriction: the hotter parts are in the center. (**b**) Four minutes later at the same temperature, the digits of the same hand of the same subject have become very cold, with the palm becoming colder than it was initially. NOTE: infrared images record peripheral, not deep, temperatures.

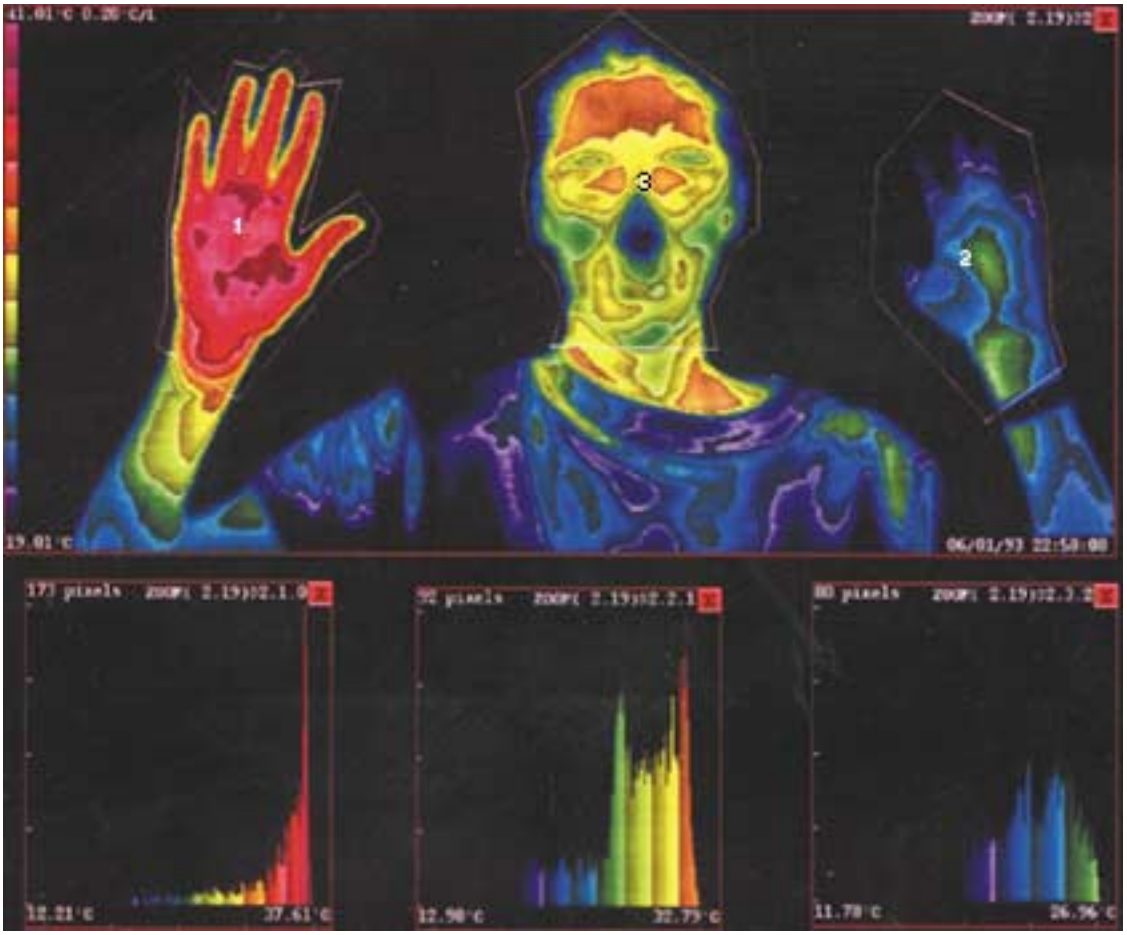

Fig. 11-5. This infrared image is of the upper body of a person in an air environment of 19°C. The right hand (1) was placed in a glove until immediately before the picture was taken. The left hand (2) was not gloved. Note the extreme difference in vasoconstriction in the two hands. Selective vasoconstriction can also be seen in the neck and face, with the nose (3) vasoconstricting the most, followed by the cheeks, with the paranasal areas and the forehead staying warmer. NOTE: infrared images record peripheral, not deep, temperatures.

cal agents influence peripheral vasoconstriction. Most military operations in cold weather are negatively affected by the pronounced discomfort associated with vasoconstriction of the extremities.

This representation is different from the classic one in that it demonstrates the power of the peripheral temperature in influencing core temperature.[27] From a practical point of view, in many cold weather operations, emphasis is placed on adequate hydration, sleep, nutrition, and clothing. Participants sometimes overdress, which leads to sweating and vasodilatation, which promote heat loss in a cold environment. Just as the physiological systems must constantly be increasing or decreasing various heat-producing mechanisms, so should the soldier who is in a cold environment. The major point is not to overdress, and at the same time to be aware of the insidious onset of hypothermia. This simple advice is difficult to implement, because the soldier must be able to withstand a wide range of temperatures. Thus, a bulky coat is usually issued.

The following discussion describes the effects of cold stress leading to hypothermia on major physiological systems (Table 11-1). Each physiological system has its own response to a decrease in core temperature, which create the signs of hypothermia (Exhibit 11-2). The challenge is understand that each of these systems interacts with every other. *Cold stress* refers to the body's response to cold, which, if not effective, will lead to hypothermia. In most field situations, the effects of cold are first felt in the extremities (feet, hands) and lead to frostbite; a more detailed presentation of the body's response to this stressor is found in Chapter 14, Clini-

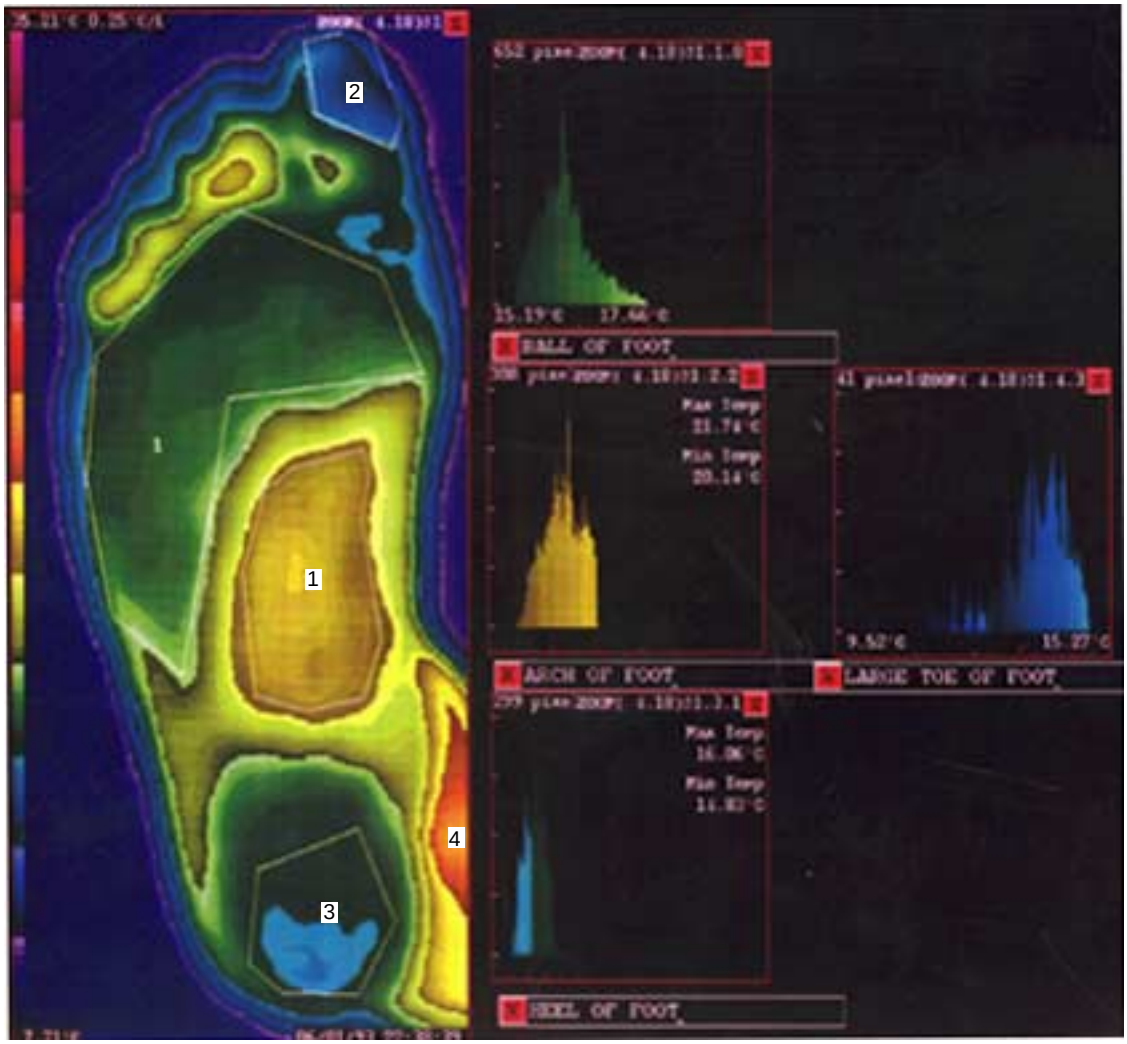

Fig. 11-6. This infrared image is of a subject's left foot, which was exposed to an environmental temperature of 7.0°C. The warmest part of the foot is the arch (1), whereas the toes (2) and heel (3) are vasoconstricted. The ankle (4) demonstrates a warm spot. NOTE: infrared images record peripheral, not deep, temperatures.

cal Aspects of Freezing Cold Injury. Hypothermia in the field represents a complete breakdown of logistical and medical support for field operations. The cardiovascular system is the most important physiological system concerning hypothermia, because the cold will eventually cause this system to break down. Although core temperature is important, the emphasis during rescue operations must be on evaluating and, if necessary, correcting cardiovascular and respiratory system function. Maintenance of adequate circulation and ventilation have a higher priority than thermal stabilization (Exhibit 11-3).

Central Nervous System

Many different anecdotal and field studies indicate that the first signs of hypothermia are disruption of higher functions such as visual and auditory hallucinations, and as the core temperature drops further, slurring of speech, decreased consciousness, and impairment of short-term memory occur. In one study,[28] local cooling of the inferior parietal lobe in a patient caused the patient to believe that his speech was being uttered by a stranger. It should be emphasized that these early signs might be the most critical for field operations, because they occur with mild hypothermia. In the field, monitoring an individual's behavior (by whomever is in charge, or a buddy) is more effective than attempting to measure the core temperature. Changes in an individual's behavior such as becoming withdrawn or silent may indicate the early stages of hypothermia. Consciousness is usually lost at a body temperature of 28°C to 30°C, but there are isolated

instances of persons still being able to talk when the core temperature was as low as 24°C.[29]

Although individual CNS neurons may be excited by a drop in temperature of one Centigrade degree, not all neurons are uniformly activated because the brain does not cool uniformly. This is demonstrated by a significant nonuniformity in temperatures in various areas of the brain in dogs, sheep, monkeys, and cats.[30] In addition, the anatomical organization of the cortical neurons may partially explain CNS changes associated with hypothermia. Some neurons have lengthy axons that extend to the periphery of the brain, and therefore cold temperatures will interfere with their electrical activity. Thus, neurons in the cortex with vertical extensions from the nerve cell body may have cold-induced multiple spikes.[31]

Eventually, hypothermia will cause a decrease in

TABLE 11-1

ALTERATION OF HUMAN PHYSIOLOGICAL SYSTEMS WITH DECREASING CORE TEMPERATURE

	Core Temperature		
Stage	**°C**	**°F**	**Characteristics**
	37.6	99.6 ± 1	Normal rectal temperature
	37.0	98.6 ± 1	Normal oral temperature
	36.0	96.8	Increase in metabolic rate, blood pressure, and preshivering muscle tone
Mild	35.0	95.0	Urine temperature 34.8°C; maximum shivering thermogenesis
	34.0	93.2	Amnesia, dysarthria, and poor judgment develop; maladaptive behavior, normal blood pressure; maximum respiratory stimulation; tachycardia, then progressive bradycardia
	33.0	91.4	Ataxia and apathy develop; linear depression of cerebral metabolism; tachypnea, then progressive decrease in respiratory minute volume; cold diuresis
Moderate	32.0	89.6	Stupor; 25% decrease in oxygen consumption
	31.0	87.8	Extinguished shivering thermogenesis
	30.0	86.0	Atrial fibrillation and other arrhythmias develop; poikilothermia; pupils and cardiac output 67% of normal; insulin ineffective
	29.0	85.2	Progressive decrease in level of consciousness; pulse, and respiration; pupils dilated; paradoxical undressing
	28.0	82.4	Decreased ventricular fibrillation threshold; 50% decrease in oxygen consumption and pulse; hypoventilation
	27.0	80.6	Loss of reflexes and voluntary motion
Severe	26.0	78.8	Major acid–base disturbances; no reflexes or response to pain
	25.0	77.0	Cerebral blood flow 33% of normal; loss of cerebrovascular autoregulation; cardiac output 45% of normal; pulmonary edema may develop
	24.0	75.2	Significant hypotension and bradycardia
	23.0	73.4	No corneal or oculocephalic reflexes; areflexia
	22.0	71.6	Maximum risk of ventricular fibrillation; 75% decrease in oxygen consumption
	20.0	68.0	Lowest resumption of cardiac electromechanical activity; pulse 20% of normal
	19.0	66.2	Electroencephalographic silencing
	18.0	64.4	Asystole
	16.0	60.8	Lowest adult survival from accidental hypothermia[1]
	15.2	59.2	Lowest infant survival from accidental hypothermia[2]
	10.0	50.0	92% decrease in oxygen consumption
	9.0	48.2	Lowest survival from therapeutic hypothermia[3]

(1) DaVee TS, Reinberg EJ. Extreme hypothermia and ventricular fibrillation. *Ann Emerg Med*. 1980;9:100–110. (2) Nozaki RN, Ishabashi K, Adachi N. Accidental profound hypothermia. *N Engl J Med.* 1986;315:1680. Letter. (3) Niazi SA, Lewis FJ. Profound hypothermia in man: Report of case. *Ann Surg.* 1958;147:254–266.

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nerve conduction. For example, in human peripheral nerves, conduction velocity decreases from 30 m/s at 35°C to 12 m/s at 21°C. These decreases partially explain the observed motor incoordination and decrease in manual dexterity.[32] There will also be some cold-induced muscle stiffness and decrease in blood flow to the limbs, which contribute to the incoordination and loss of strength at that core temperature.

As core temperature continues to fall, cerebral metabolism decreases linearly from 6% to 10% for

EXHIBIT 11-2

SIGNS OF HYPOTHERMIA

Psychiatric
- *Impaired judgment
- *Perseveration
- *Peculiar "flat" affect
- *Altered mental status
- Paradoxical undressing
- Neuroses
- Psychoses
- Suicide
- Organic brain syndrome
- Anorexia
- Depression
- Apathy
- Irritability

Cardiovascular
- *Peripheral vasoconstriction
- *Initial tachycardia
- Dysrhythmias
- Decreased heart tones
- Hepatojugular reflux
- Jugular venous distension
- Hypotension

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

Musculoskeletal
- *Increased muscle tone
- *Shivering
- Rigidity or pseudo rigor mortis
- Paravertebral spasm
- Opisthotonos
- Compartment syndrome

Gastrointestinal
- Ileus
- Constipation
- Abdominal distension or rigidity
- Poor rectal tone
- Gastric dilation in neonates or in adults with myxedema
- Vomiting

Genitourinary
- *Polyuria
- Anuria
- Oliguria
- Testicular torsion

Neurological
- Depressed level of consciousness
- Ataxia
- Dysarthria
- Amnesia
- Anesthesia
- Areflexia
- Poor suck reflex
- Hypoesthesia
- Antinociception
- Initial hyperreflexia
- Hyporeflexia
- Central pontine myelinolysis

Dermatological
- *Erythema
- *Pallor
- *Cyanosis
- Icterus
- Scleral edema
- Ecchymosis
- Edema
- Perniosis
- Frostnip
- Frostbite
- Panniculitis
- Cold urticaria
- Necrosis
- Gangrene

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

*Usually occurs during the initial exposure to cold stress and hypothermia

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

each one Centigrade degree decrease in temperature from 35°C to 25°C.[31] Significant attenuation and frequency alterations in the brain's electrical activity can be observed at temperatures below 34°C.[33–35] Most importantly, prolonged hypothermia of the brain affects cerebral functioning in a descending manner, so that cerebral cortex function is initially impaired, followed by subcortical structures. When medullary cellular activity is suppressed, cessation of respiration follows. This hypothermia-induced apnea can be reversed by warming the fourth ventricle. Complete absence of electrical activity (a flatline electroencephalogram) normally occurs at temperatures below 20°C.

One of the challenges for medical officers involved in field operations in the cold is the fact that hypothermia will negatively affect an individual's performance but, paradoxically, once the person is hypothermic, the cold will transiently protect the brain from hypoxia. Fundamentally, it is still not clearly understood how hypothermia protects the brain from various hypoxic environments. Cooling the brain nonuniformly affects neural function, localized blood flow, and the integrity of the blood–brain barrier. Relative to other organ systems, a disproportionately high redistribution of blood flow is directed to the brain when profound hypothermia has occurred. Autoregulation of cerebral blood flow is maintained until brain temperature falls below 25°C. Part of the explanation of the cold-protective effect that hypothermia has on the brain is that it reduces vascular permeability in cerebrally nonischemic rats.[36] Decreasing cerebral temperatures minimized hypoxia-induced abnormalities in the blood–brain barrier in ischemic animals, whereas raising the temperature to 39°C exacerbated the abnormalities.[37] In addition, mild hypothermia reduced the degree of postischemic edema in gerbils after 40 minutes of bilateral carotid occlusion.[38] Overall, these studies suggest that hypothermia reverses the destabilizing effects of hypoxia on cell membranes.

Clinically, profound hypothermia is induced to minimize or prevent cerebral ischemic injury during certain types of cardiac and cerebrovascular surgeries.[39–41] The beneficial effects of clinically induced hypothermia classically have been attributed to a temperature-dependent reduction in metabolism.[35,42,43] As a result, whole-body circulation can be arrested for prolonged periods, exceeding 30 minutes, without incurring severe cerebral injury.[39,42,44]

The mechanism of neural protection associated with hypothermia is not clear. Profound hypothermia is not a strict prerequisite for neuronal protection, because significant cerebral protection may occur at mildly cold temperatures (33°C–34°C). Improved postischemic neurological function has been reported in animals in which mild hypothermia was instituted[45–48]; however, the neural sparing effect of hypothermia may not be related to the timing of the ischemic insult. Improved outcomes following ischemia were apparent even when the hypothermia was induced either during or immediately after the occurrence of the ischemic event.[45,46,49] Further complicating this area are the observations that mild hypothermia induces cerebral protection, which has not been correlated to a reduced production of lactate (ie, reduced anaerobic metabolism).[48,49] Hence, a hypothermia-induced reduction in global cerebral metabolism, per se, does not appear to be the complete explanation for the protective effects of mild hypothermia. Experimentally, the beneficial effects of mild hypothermia may be due to the following conditions[45,47,49]:

- a reduced metabolism,
- temperature-induced alterations in ion-channel function, which promotes calcium homeostasis (a major determinant of metabolism),

EXHIBIT 11-3

PRIORITIZED RESUSCITATION REQUIREMENTS

1. Maintenance of tissue oxygenation:
   - Adequate circulation
   - Adequate ventilation
2. Identification of primary versus secondary hypothermia
3. Thermal stabilization:
   - Conduction
   - Convection
   - Radiation
   - Evaporation
   - Respiration
4. Rewarming options:
   - Passive external rewarming
   - Active external rewarming
   - Active core rewarming

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- increased membrane lipid stability,
- alterations in the release and reuptake of neurotransmitters (eg, excitatory amino acids and dopamine),
- preservation of the blood–brain barrier, and
- the release of various substances that have a protective effect on cellular membrane function.

However, these results have to be considered relative to the experimental animal used, as species vary in their ability to withstand cerebral hypoxia and hypothermia. This area of research is at present one of the most active, because hypothermia in some form may act to protect the hypoxic, physically traumatized brain.

Motor Activity

From a military point of view, the effects of a cold environment have their greatest overt effect on the motor system. Troops are not able to move as fast, and fine coordination is impaired.[50] Cold hands make it difficult to pull a trigger or operate a keyboard. Cold stress and hypothermia influence motor function by way of the neural and cardiovascular systems and on the muscle cell itself. As a person is initially cold-stressed and then becomes hypothermic, muscle tension leads to shivering, which continues until core temperature reaches 29°C to 31°C. Preshivering tone, of which cold-stressed subjects are usually unaware, normally precedes shivering. In part, this tonic muscle activity is the basis for the feeling of stiffness that most people experience when they get cold.[51] Increased motor tone has been reported to appear first in extensor and proximal muscles, which are the same muscles in which the amplitude of shiver is largest.[52] However, humans vary greatly in their shivering patterns, with some human subjects shivering first in their chest muscles.

Shiver has been defined as involuntary rhythmic waxing and waning muscular contractions that are used to maintain a normal body temperature.[53] These oscillations are modulated by myotatic reflex loops, because deafferentation will cause the frequency characteristics of shiver to become irregular.[54,55] However, shivering can be influenced by cerebral cortex. A subject can temporarily turn off shivering by relaxing, doing exercises,[56] or modifying the breathing pattern. These techniques are invaluable for field operations, because they allow troops to conduct certain aspects of their mission even when they are cold-stressed.[52,57]

From a thermogenic point of view, shivering increases heat production 2- to 5-fold more than is necessary for normal body heat production. During different phases of shivering, both agonist and antagonist muscles contract periodically but not necessarily reciprocally. Thus there will be an increase in muscle tension, but the limbs do not move effectively. The frequency of shiver varies from muscle to muscle but is considered fairly low, between 5 and 10 Hz. In laboratory experiments, cold-stressed subjects will demonstrate synchronized muscle contraction of all muscles monitored. If the antagonistic muscles were to be coactivated at higher rates or to elicit contracture (ie, sustained force production without associated electrical activity), then the heat that could be generated would be proportionally greater. However, a major drawback to this type of activation would be the high degree of resultant limb stiffness that would limit one's ability to make superimposed voluntary movements.

The control mechanisms for shiver have both central and peripheral nervous system components. CNS shivering was produced by localized cooling of the hypothalamus.[58,59] Demonstrating the effect of peripheral temperatures on inducing shivering, Lim[60] reported that reducing subcutaneous temperature from 33°C to 30°C, while maintaining a brain temperature of 38°C, evoked a shivering response. Further supporting the role of peripheral regulation of the triggering of shivering are the observations that humans placed in a 10°C environmental chamber for 15 to 40 minutes demonstrate intense shiver—even though their core temperatures have not changed or are slightly increased.[61]

In a field situation, shivering is an important sign that all physiological systems are functioning (eg, cold receptors, hypothalamus, muscles), and also that hypothermia may eventually occur. If shivering persons are able to complain about the environment, more than likely they are cold-stressed or mildly hypothermic. Their ability to complain is an important sign that the troops may be in a critical situation, but they are not severely hypothermic—yet.

Respiratory changes have been documented to alter increases and decreases in shiver amplitude or changes in duration, or both. Inspiration of cold air causes an increase in rhythmic and tonic muscle activity, whereas inspiration of warm, humidified air can attenuate or stop spontaneous shivering.[62] When soldiers wish to minimize shivering, they should not inspire deeply. Although shivering generates heat and assists in minimizing a decrease in core temperature, it is not always desirable and may, paradoxically, influence a person's performance.

In many environmental situations, such as when a deep sea diver is trying to perform a fine-motor task, shivering is clearly undesirable. Attempts have been made to minimize the occurrence of such tremors during diving by employing specialized (ie, warmed) oxygen tanks to avoid hypothermia and prevent shiver. Although this technique minimizes shivering, it will not prevent the onset of hypothermia.

Cold environmental temperatures affect the muscles directly, protecting them when frozen. There are clinical case reports (discussed in greater detail Chapter 14, Clinical Aspects of Freezing Cold Injury) of individuals with frozen limbs who have been successfully rewarmed with no apparent long-term effects.

Circadian Rhythms and Sleep

Sleep deprivation is common in military operations. Because the sleep cycle and other circadian cycles are intimately linked, sleep deprivation might affect thermoregulation, as it does other neural systems, so as to cause visual hallucinations and impaired balance.[63] Although the sleep cycle influences thermoregulation by altering fundamental mechanisms in the CNS, the ambient temperature also influences these cycles.[64] An ideal situation for inducing hypothermia would be having troops with minimum food and water, isolated in a hostile, cold environment—such as a mountain—in which they cannot sleep.

THERMOREGULATION IN MAJOR PHYSIOLOGICAL SYSTEMS

Cold stress triggers changes in all physiological systems. As the drop in core temperature continues, all these systems demonstrate the effect of cold on the cellular metabolism of the organ, the blood flow, and neural activation. Understanding the effect of hypothermia on each system will allow the medical officer to be better prepared to assist the victim of hypothermia.

Cardiovascular System

The cardiovascular system has received the most attention in clinical studies of hypothermia because various surgical techniques, such as cardiac bypass surgery, have successfully employed low body temperatures. The reversibility of cold-induced ventricular fibrillation (cardiac arrest, or standstill) is one of the major determinants of survivability from hypothermia. A drop in core temperature will induce ionic alterations in cardiac muscle, such as hyperkalemia, which may induce cardiac standstill or fibrillation.[56,65]

Cold stress induces sympathetically mediated peripheral vasoconstriction, an increase in cardiac afterload on the heart, and elevated myocardial oxygen consumption. These changes are often associated with an initial tachycardia. As the core temperature continues to fall, bradycardia and myocardial depression occur, resulting in a decreased cardiac output and hypotension. In mild hypothermia, the variability in circadian heart rate is greater than during normothermia, possibly due to an imbalance between the parasympathetic and sympathetic nervous systems.[66]

A decrease in heart rate by 50% can be recorded from individuals with core temperatures near 28°C.[67] The lowered heart rate results from a decrease in the spontaneous depolarization of pacemaker cells and is refractory to atropine.[68] At core temperatures below 32°C, atrial dysrhythmia occurs, secondary to atrial distension.[69] Ventricular arrhythmias are commonly observed below 32.2°C, but primary ventricular fibrillation is rare at 32.2°C, with maximal susceptibility occurring between 28°C and 30°C.[70] At core temperatures lower than 30°C, the heart is very sensitive to mechanical stimulation, and cardiopulmonary resuscitation efforts may convert a very slow sinus bradycardia to ventricular fibrillation. As the core temperature approaches 25°C, fluid shifts out of the vascular space, which may increase the hematocrit by 150%.[71] The ensuing hypovolemia and increased blood viscosity further compromise the cardiac output.

An electrocardiogram demonstrates significant changes with hypothermia. These electrical changes are indicative of specific myocardial ionic activities that are influenced by the cold. Membrane currents are controlled by multiple processes that control the membrane channels, which are composed of lipoprotein and other chemicals whose activities are temperature-dependent. Thus, low temperatures result in both a slower activation and inactivation of different membrane currents, and they contribute to various electrophysiological changes. During hypothermia there is a prolongation of the PR and Q-T intervals and widening of the QRS complex. A significant drop in core temperature results in the reduction of the rate of depolarization, which in turn results in a widening of the QRS complex. The explanation for this phenomenon is that during hypothermia, the rate of the opening and closing of the sodium channels is decreased, and so-

dium-channel conduction is decreased as well, causing a reduction in the maximal rate of membrane depolarization. This phenomenon involves an interplay between various ions, such as sodium and potassium, because these ions have common transport mechanisms.

Hypothermia also influences the repolarization phase of the cardiac action potential. Due to alterations in various potassium currents, a drop of one Centigrade degree in myocardial temperature lengthens the cardiac action potential and refractory period by 15 to 20 milliseconds. During phase I of repolarization, there is an early transient outward potassium current. During phase III, there are two simultaneous temperature-sensitive currents: a time-dependent, delayed rectifying, potassium current and a time-independent, inwardly rectifying potassium current. When both of these repolarizing currents are reduced, a consequent lengthening of the action potential duration and refractory period occurs. Other inward currents, such as sodium and calcium, are also affected by hypothermia and contribute to the lengthening of the action potential.

Following depolarization, there is an opening of the voltage-dependent calcium channels, causing an influx of calcium ions, which in turn activates the release of calcium from internal storage in the sarcoplasmic reticulum. Subsequently, intracellular free calcium binds to contractile proteins, resulting in muscle contraction.[72] As a result, during the initial stages of hypothermia, systolic contractile force and intracellular calcium increase. This is due either to increased levels of free cytosolic calcium or to the increased sensitivity of the contractile proteins to calcium. Some investigators contend that cardiovascular collapse during hypothermia is not due to the irregularities of myocardial contraction but to reduced contractility or arrhythmia.[73]

The explanation for hypothermia-induced cardiac arrhythmias is not settled. The circus theory proposes that either a nonhomogeneous conduction or refractoriness, or both, may exist. As a result, there is a greater increase in conduction time than in the refractory period. Such an increase in the ratio of conduction time to refractory period makes reentry currents possible, resulting in ventricular fibrillation.[74] Another explanation is that nonhomogeneous thermal profiles result in disproportionate changes in refractory periods and conduction times. These cold-induced changes could easily produce multiple ectopic sites, eventually resulting in ventricular fibrillation.

Hypothermia affects the atria and ventricles differently. Because the speed of conduction is greater in the atria than in the ventricles, the pacemakers of the atrium will maintain normal synchronized muscle contraction at much lower temperatures. In contrast, because the conduction velocity in Purkinje's fibers is slower even at normal temperatures, the ventricles are more susceptible to being inhibited by the cold. This allows the ventricular myocardium to contract irregularly, promoting multifocal ventricular tachycardiac sites, and leading eventually to fibrillation or cardiac standstill. This difference in susceptibility is seen in rat hearts that have been stored at 4°C for 0, 12, and 24 hours.

Owing to the importance of hypothermia-induced ventricular fibrillation, much research has focused on the effect of cold on cardiac muscle and the conducting system in the heart, but interestingly, the effect of hypothermia on the coronary circulation has not received the same degree of study. There is little evidence that suggests that cold stress influences the responsiveness of the coronary arteries.[75] However, it is well recognized that angina pectoris (constriction of coronary arteries) can be either precipitated or worsened just by exposing the skin to cold[41]—a decrease in core temperature (à la hypothermia) is not required. Although current understanding has presumed that cold increases the metabolism of cardiac tissues by activating the sympathetic nervous system, this hypothesis has not been rigorously substantiated. In summary, the coronary circulation appears to respond to a cold stress as it would whenever cardiac output and systemic pressure increase through activation of the sympathetic nervous system.

Respiratory System

The initial respiratory response to cold stress is a significant increase in rate (ie, hyperventilation), followed by a decrease (ie, hypoventilation) (see Exhibit 11-2). Skin temperature afferents can influence respiratory function dramatically. Certain individuals will hyperventilate when they are exposed to a cold stress and others will not. The cold-stressed hyperventilation is followed by a progressive decrease in the respiratory minute volume that is proportional to the decreasing metabolism. The control of respiration becomes compromised as the function of the brain stem is impaired by severe hypothermia. Respiratory rate falls from 15 to 7 breaths per minute at 30°C to 7 to 4 breaths per minute at temperatures in the mid 20s.[76] Eventually, retention of carbon dioxide by the tissue leads to respiratory acidosis. In most cases of severe hypothermia, respiration diminishes and the heart continues to contract for some time.[77]

In field situations, the evaluation of respiration in victims of hypothermia is extremely challenging, as their slow breathing rate might be masked by environmental conditions (eg, wind, machine noise). Hypoxia can accelerate the decrease in core temperature. In a moderate cold-stress situation, hypercapnia lowered the threshold for shivering by 0.13°C and increased the core cooling rate by approximately 25%.[78] This decrease in core temperature may be due to the hypercapnic hyperventilation. This observation is important, because it demonstrates that in moderately cold environments, hypercapnia influences thermoregulation, whereas at very cold temperatures the body's response is so vigorous that it swamps the hypercapnic effect.

Stimulation of the respiratory drive by both carbon dioxide and hypoxia is absent at 20°C.[79] During moderate hypothermia, in an absence of shiver, a reduction in oxygen consumption is associated with a parallel reduction in carbon dioxide production. Thus, what would be considered low levels of oxygen pressure in normothermic environments would be adequate at hypothermic levels. Although in hypothermia the arterial content of carbon dioxide is low, the solubility of carbon dioxide has increased.

As an individual becomes hypothermic, several other physiological factors associated with respiratory function are influenced[80]:

- ciliary motility decreases,
- bronchorrhea is present,
- the potential for noncardiogenic pulmonary edema increases as fluid shifts occur,
- the contractile function of the diaphragm and intercostal muscles alters,
- lung compliance decreases,
- the elasticity of the thorax decreases, and
- anatomical and functional physiological respiratory dead spaces are increased, whereas individual alveolar dead spaces are unchanged.

Pulmonary circulation time is usually prolonged unless there is intrapulmonary shunting.

Although hyperventilation is associated with cold stress, cold-induced respiratory arrest also occurs. This reflex may be important in victims of submersion hypothermia. Such a response causes the person who is submerged to aspirate water and consequently drown. Simultaneously, the cold water aspirate rapidly cools the brain and heart, because the heart continues to beat effectively while pumping cold blood, for 5 minutes after aspiration. The blood is rapidly cooled because it is circulated in the pulmonary cold water environment, dropping cerebral and cardiac temperatures. This rapid internal cooling is considered to be the explanation for the complete recovery of victims who experience cold water near-drowning. When a person nearly drowns in cold water, there are approximately 45 minutes during which the victim may be successfully revived. The rapid internal cooling of the internal organs, such as the brain and heart, allows the victim to survive hypoxia for approximately 45 minutes. This form of cooling is more effective in children than adults because they have a smaller mass and a large surface area–to-volume ratio. Not all victims who suffer from submersion hypothermia are successfully revived, however. Many remain comatose after heroic rescue and clinical attempts. This wide range of response may be due to a large number of variables, including the temperature of the water, the rate of cooling, the nature and quantity of the aspirate, and the clinical treatment.[81,82]

Renal System

Cold-induced diuresis is one of the early consequences of exposure to the cold, and it becomes prominent even before core temperature has decreased. The mechanisms for this cold-induced diuresis remain controversial.[83] One school of thought suggests that cold-induced diuresis is an autoregulatory response of the kidney to a relative central hypervolemia induced by peripheral vasoconstriction. Owing to a volume overload, the release of antidiuretic hormone is suppressed. The subsequent cold-induced diuresis decreases the blood volume so that progressive hemoconcentration develops.

The other explanation is that cold-induced diuresis may be due to osmotic alteration in the renal tubules. Renal function is eventually depressed during hypothermia owing to a fall in systemic blood pressure and the indirect effect of the cold on organ metabolism itself. As the renal blood flow decreases, renal vascular resistance rises, promoting a further decrease in renal flow and a subsequent decrease in glomerular filtration. During hypothermia, renal oxygen consumption is more rapidly reduced relative to other organs such as the liver, heart, brain, skeletal muscle, and skin. Serum sodium, calcium, chloride, and potassium concentrations remain in the normal range until core temperature is 25°C, but owing to the cold-induced depression of the renal tubular function, sodium and water reabsorption are reduced, promoting a pronounced osmotic diuresis.[84]

Faced with continuous hypothermia, an additional large shift of body water will occur. Whether the cold-induced diuresis is explained on the basis of volume overload or ionic imbalances, it is a major concern. For example, cold water immersion has been shown to increase urinary output by 3.5-fold, and this decrease in body water may be a factor contributing to the "rewarming shock" that occurs following active vasodilation induced by rewarming treatments.[85] Potassium ion regulation may be impaired in hypothermia. Hyperkalemia, one of the leading causes of cardiac dysrhythmia,[80] is usually an ominous sign of tissue hypoxia.[86]

From the practical standpoint in the field, one of the only ways to assess hydration is to examine the color of the urine. Most military units insist on visually inspecting the degree of darkness of each individual's urine. The more hydrated the individual, the less dark the urine (see Figure 5-5 in Chapter 5, Pathophysiology of Heatstroke).

Blood

As hypothermia decreases cellular function, the amount of oxygen available remains constant because the oxyhemoglobin dissociation curve shifts to the left. This shift is physiologically very important, as it dictates that the partial pressure of oxygen must fall to lower values before hemoglobin gives up its oxygen.[87] Thus, hypothermia induces a physiological bank of oxygen. In the face of hypoxia, cells shift to anaerobic metabolism, resulting in a metabolic acidosis. As hydrogen ions enter the blood, they shift the oxygen dissociation curve to the right, which promotes the unloading of oxygen. Thus we can say, simplistically, that hypothermia protects various organs because hypothermic organs have decreased oxygen demands, while simultaneously, adequate oxygen is available to meet those reduced metabolic demands.

Contributing to the therapeutic effects of hypothermia is the fact that both oxygen and carbon dioxide are more soluble in cold blood. Compared with normothermic values, the solubility of oxygen is increased by 33% at 25°C. Although this increase in solubility cannot be considered an added benefit until the temperature of the tissue falls to 16°C,[88] it nevertheless allows for oxygen to be available to the hypothermic cells.

Another important but deleterious consequence of hypothermia, particularly for combat casualty care, is that clotting time is prolonged. This is because enzyme reaction times are reduced, which slows clotting time, and because the platelets are sequestered in both the portal circulation and the liver. In addition, an elevation in hematocrit and viscosity occur.[88] Patients with clinically induced hypothermia, as measured by tympanic probes, experienced blood loss 0.5 L greater than that of normothermic patients, leading some investigators to argue for minimizing mild hypothermia during surgery.[89]

Acid–Base Balance

The most important, yet controversial, area of hypothermia is the clinical treatment of acid–base imbalance. Although the decrease in core temperature is considered the important physiological *consequence* of a cold stress, the key physiological *element* is the control of hydrogen ion concentration. Acid–base balance in hypothermic situations differs from that in normothermia. Owing to the variety of underlying causes of hypothermia, clinical prediction of acid-base status is not possible. In one series of 135 cases, 30% of patients were acidotic and 25% were alkalotic.[90] After an initial respiratory alkalosis from cold-induced hyperventilation, the more common underlying disturbance is a relative acidosis. Acidosis has both respiratory and metabolic components. From a respiratory perspective, as the temperature decreases, the solubility of carbon dioxide in blood increases. Metabolic acidosis is produced by impaired hepatic metabolism and acid excretion, lactate generation from shivering, and decreased tissue perfusion.

How should a medical officer correct a hypothermia-induced pH profile? Confusion persists regarding arterial blood gas pH correction relative to the reduction in core temperature. Initially, to aid the clinician's interpretation of the pathophysiology involved in hypothermic arterial oxygenation and acid–base balance, the pH was corrected to normal values for body temperature.[91] This approach created problems. If a pH electrode was used at the casualty's current core temperature, an uncorrected but exact pH value would be obtained. However, arterial blood samples are always warmed to 37°C before electrode measurements are obtained and are not measured at the patient's subnormal temperature.

Optimal clinical strategy to maintain acid–base homeostasis during treatment of accidental hypothermia is still evolving.[92] The practical clinical problem is of some importance in cardiac surgery, however, where there is considerable experience with hypothermia during cardiopulmonary bypass. The assumption that was accepted earlier was that 7.42 is the ideal, "corrected" patient pH at all tem-

peratures, and that therapy should be directed at maintenance of the corrected arterial pH at 7.42. The approach for maintaining this pH level, termed "endothermic," has been questioned.[89] A better intracellular pH reference may be electrochemical neutrality, in which pH = pOH. Because the neutral point of water at 37°C is pH 6.8, Rahn and colleagues[93] hypothesized that this normal 0.6-unit pH offset (7.4 – 6.8) in body fluids should be maintained at all temperatures. Because the neutral pH rises with cooling, so should blood pH. This pH approach, termed "ectothermic," is commonly followed.

Previously, Rahn[94] had observed that Antarctic codfish survive far below the freezing point of water (owing to a presence of glycoprotein that minimizes formation of ice crystals [antifreeze]), and they continue to function in an extremely alkalotic state. This same blood pH variation (ie, a rise in pH with a decline in temperature) is found in other cold-blooded vertebrates and invertebrates. Several experimental and clinical studies support Rahn's hypothesis. In one study,[95] a set of puppies with pH maintained at 7.4 had a 50% drop in cardiac performance after bypass. The control group, left alkalotic, had normal cardiac indices and increased cerebral blood flow. In another study[96] with canines during systemic deep hypothermia, constraining the correct pH to 7.4 caused myocardial damage, whereas relative alkalinity afforded myocardial protection. Other advantages of relative alkalinity include improved electrical stability of the heart. The fibrillation threshold of dogs markedly decreased when arterial pH was held at 7.4 but was unchanged with alkalosis. In contrast, maintaining the pH at 7.4 during hypothermia in a rat model did not affect cardiac work response.[97] These data suggest that the optimal range of extracellular pH is large in some species.

Supporting the view that the alkalotic state is beneficial to hypothermic patients is a study by Kroncke and associates,[98] in which they studied 181 patients who had cardiac bypass surgery, 121 consecutive cases of whom were "endothermically" managed with corrected normal pH and P_{CO_2} (partial pressure of carbon dioxide) values. Ventricular fibrillation occurred in 49 (40%). The remaining 60 patients were left "ectothermically" alkalotic; of these, only 12 (20%) developed spontaneous ventricular fibrillation.

These observations provide some evidence in support of Rahn's hypothesis, that the advantage that ectotherms obtain with a constant relative degree of alkalinity also applies to warm-blooded endotherms during hypothermic conditions. Potentially deleterious effects of alkalosis on other systems have yet to be identified. However, on the acidotic side, 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. Correction of pH and P_{CO_2} in patients with hypothermia is unnecessary and potentially deleterious. This last statement is pertinent to field rescue operations. Owing to the complexity of the interaction of the causes of hypothermia, as well the acid–base stabilization, it is always advisable to minimize heroic efforts in the field to rewarm hypothermic casualties and correct their pH, unless they can be properly evaluated and medically managed. Raising the body temperature or attempting to correct the pH level in blood, or both, might cause potentially deleterious changes in blood pH, leading to ventricular fibrillation.

Fluid and Electrolyte Balance

Dehydration is usually associated with hypothermia, with free-water depletion elevating serum sodium and osmolality. Because hypothermia produces natriuresis, saline depletion may be present.[69]

Blood viscosity increases 2% per degree Centigrade drop in temperature, and hematocrits higher than 50% are seen. During rewarming, low circulatory plasma volume is often coupled with elevated total plasma volume.[99]

Infusion of fluid does not always reverse hypothermia-induced fluid shifts. In one set of experiments,[98] normal saline had minimal lasting effects and did not hasten cardiovascular recovery from hypothermia. In another study,[100] 10% low molecular weight dextran solution increased plasma volume and decreased blood sludging.

In some patients with hypothermia, rapid volume expansion is critical.[101] In neonates, adequate fluid resuscitation markedly decreases mortality.[65]

Gastrointestinal System

Gastrointestinal smooth muscle motility decreases as core temperature falls, resulting in acute gastric dilation, paralytic ileus, and distension of the colon. In addition, all gastrointestinal secretions and free acid production are depressed.[69] The pancreas and the gastric mucosa are major sites of the cold-associated hemorrhages called Wischnevsky's lesions,[102] which are seen in 80% of victims of hypothermia and are of greater severity in younger

individuals. These lesions may be the result of reperfusion after cold-induced collapse of the microvasculature. Hypothermia causes a catecho---lamine-induced vasoconstriction of blood vessels and release of corticosterone, which can be ulcerogenic. Eventually catecholamine secretion is decreased, promoting a vasodilatation that results in significant reperfusion and eventual extravasation of blood. The reperfusion and associated changes alter the gastric mucosa's protective mechanism, resulting in cellular damage induced by hydrochloric acid.

Hypothermia causes a decrease in splanchnic blood flow, which may be greater than the proportional fall in cardiac output.[103] Liver cells continue to metabolize but are not able to utilize glucose. Associated with the depression of liver function will be a significant decease in its ability to rid the body of metabolites, drugs, or conjugate steroids. Simultaneously, the other cells in the body are also inactivated by the cold. Thus, the drugs' target cells will not metabolize the drugs. This fact explains why various drugs have a reduced effect in hypothermic individuals, and explains the failed attempts of drug-induced suicide in hypothermic victims.

Endocrine System

Cold stress and hypothermia are major stressors and evoke a widespread hormonal response. Exposure to cold will stimulate the release of catecholamines, which will stimulate thermogenesis.[104]

Corticosteroids also become elevated. There is an inverse relationship between the concentration of 11-hydroxy-corticosteroids in plasma and the depth of hypothermia. In one study,[105] the highest concentrations of corticosteroids (96.4 μg/dL) were measured in hypothermic individuals who died, whereas those who died 3 days later had corticosteroid values of 87.1 μg/dL, and those who survived had the lowest levels, 62.9 μg/dL. However, in another study, Stoner and colleagues[106] did not find any correlation between plasma cortisol concentration and core temperature with respect to survivability. Thyroid-stimulating hormone (TSH) and thyroid hormone concentrations have been recorded as normal in hypothermic patients. With rewarming, concentrations of thyroxine (T_4) and triiodothyronine (T_3) concentrations decreased: T_4 concentrations were 8.2 μg/dL and decreased to 7.0 μg/100 dL, and T_3 concentrations decreased from 155 μg/dL to 138 μg/dL.[107]

Insulin concentrations in hypothermic patients vary. Insulin's role in facilitating the transport of glucose into cells becomes inactive below 31°C, and yet at these temperatures blood glucose concentrations are noted to be variable. Prescott and colleagues[108] reported that some hypothermic patients were actually hyperglycemic, but these patients had diabetes and severe ketoacidosis. In general, the blood glucose concentration depends primarily on the metabolic state of the patient and not on the degree of hypothermia. The control of glucose levels in hypothermic states is far from understood because pancreatitis is a common finding at autopsy of hypothermic individuals.[109] The extent of hyperglycemia is proportional to the degree of body cooling. Depending on the degree of hypothermia, the hyperglycemia may be due to (1) an increase in catecholamine secretion, (2) a decrease in insulin activity, (3) a decrease in renal clearance of glucose, (4) a decrease in liver enzyme function, and (5) an increase in catecholamine-induced glycogenolysis. Information concerning protein and fat metabolism during various levels of hypothermia is lacking.[41]

Ethanol ingestion inhibits glucose-induced insulin secretion and stimulates pancreatic glucagon secretion. Overall, ethanol will lower blood glucose concentration and impair gluconeogenesis. Hypoglycemia associated with exercise will promote a faster rate of hypothermia.[110] Thus, military attention to proper diet in cold weather operations is critical. Giving alcoholic drinks to victims of hypothermia may make them feel better, owing to the anesthetizing effects of the alcohol, but will inhibit their natural heat-generating mechanisms.

Immune System

The effect of hypothermia on the immune system is rarely considered in reviews. In a real-life scenario, hypothermia is usually associated with infections that might compromise the tolerance of the victim. In controlled cold stress or hypothermic studies—in either Department of Defense laboratory or military field experiments in which the subjects were previously screened for illness—the hypothermic subjects rarely became sick. In an extensive number of hypothermic studies conducted at the University of Minnesota in which more than 250 medical students were made mildly hypothermic, none became ill following a 3-week period of evaluation. However, in both hospitals and field operations, in which various stressors interact to compromise the immune system, hypothermia and infection go hand in hand.

Everyday experiences demonstrate that decreased ambient temperature inhibits immune function. When a soldier injures a joint, for instance, ice

is used to prevent the infiltration of immune cells and the subsequent release of inflammatory cytokines. Conversely, heat can be applied to abscesses to speed healing. A more dramatic example would be the high propensity of leukopenia and bacterial infections in children kept hypothermic for clinical reasons.[111] Although advances in immunology have not yet been integrated with existing knowledge of hypothermic sequelae, unanticipated nonthermal positive effects may be seen.[112]

Fever augments immune function[113,114] because hyperthermia of two Centigrade degrees above normal core temperature temporarily raises the mononuclear cell count in patients with cancer and increases the mitogenic response.[115] Thus, an increase in body temperature, whether induced or spontaneous, can confer an advantage to the immune response.

On the other hand, decreases in core temperature are detrimental to immune function, as opposed to having merely a neutral effect.[114] Sessler and colleagues[116] demonstrated that wounds are larger in guinea pigs that are infected under hypothermic conditions than in those infected under control conditions. Because more than half of the body volume is 1 inch from the surface and significantly cooler than the core body temperature of 37°C,[117] local skin temperatures may influence the growth of infections. Vasoconstriction lowers resistance to infection by decreasing the partial pressure of oxygen in tissues.[118] This decrease in oxygen pressure decreases oxygen- and nitrogen-containing free radicals, both of which play major roles in microbial killing.

One explanation for cold-induced immunosuppression is that the immune cells are specifically inhibited by decreased temperature. In cases of secondary hypothermia, when thermal compensatory mechanisms become inadequate, certain observations can be made about the effect of cold on specific populations of immune cells. Histamine release from type I mast cells is decreased at low temperatures,[119] and Biggar and colleagues[120] showed that neutrophils were impaired in their migration, both in vivo and in vitro, at reduced temperature. When the cooled cells were rewarmed, they exhibited optimal activity. In a clinical study,[121] hypothermic patients (as assessed by tympanic temperature) who had undergone colorectal surgery had more surgical wound infections and their sutures were removed 1 day later than patients who were given additional warming. Peripheral vasoconstriction was seen in 78% of the hypothermic patients versus 22% for the normothermic group.[121] From a military perspective, there was another interesting finding: three times as many infections were found among smokers in both groups. Minimizing smoking among troops might do as much to minimize infections postoperatively as efforts to rewarm patients who are mildly hypothermic.

Wang-Yang and colleagues[122] reported that some in vitro responses of helper T cells in mice are inhibited by cold, but that B cells were not similarly suppressed. Cold interfered with interleukin (IL) production in virgin helper T cells, implying an early block in the activation of these cells. However, the responses of these cells to IL-2 and IL-4 were not affected by cold.

One of the most compelling, yet challenging, aspects of immunology is to understand the mechanisms by which individual parts integrate into a functional whole. Limited studies have addressed this important issue. Corticosteroids, which are released during cold stress, hypothermia, or both, have a well-documented immunosuppressive effect.[123] When cold stress is applied to an animal, specific changes in the cellular components can be observed. Sundaresan and colleagues[124] showed that when albino rats were subacutely stressed with cold water immersion, the total number of immune cells was initially expanded. Total white cell count was increased, as were total numbers of eosinophils and basophils. Phagocytic and avidity indices were also increased in phagocytic cells. However, Cheng and colleagues[125] showed that prolonged cold water stress actually has an immunosuppressive effect: they reported a decreased number of thymocytes and splenocytes, as well as diminished blastogenesis of T cells and lowered activity of natural killer cells. Macrophages were found to be less responsive to interferon gamma, and because these antigen-presenting cells are crucial for initiating immune cascades, the impairment of macrophage function could be a significant cause of a dampened immune response. While the mice in the Cheng experiment were obviously also stressed by anxiety and exercise, these results have implications for many settings of human accidental hypothermia.

Aarstad[126] reconfirmed the results of Cheng, in that an absolute value of cluster of differentiation 4+ (CD4+) cells, which are most commonly considered to be helper T cells, was affected by cold stress, but not that of CD8+ cells, which are most commonly considered to be killer T cells. In the Aarstad experiments, the number of stressors per day, as well as the duration of the trial, were varied and had an effect on the various populations of cells. For example, mice stressed once a day actually showed an increase in the percentage of CD4+ cells,

while the mice stressed twice a day showed a decrease.

Current data suggest that the immune system is significantly impaired in hypothermic settings. Some studies, however, indicate the contrary: that antibody–antigen interactions may actually be stronger at colder temperatures. Further, the optimal working temperature of complement is said to be 20°C to 25°C.[127] However, as was previously reiterated, many of the cell-mediated responses and the microenvironmental conditions that are critical to an active immune response are made defective by cold. To emphasize what has been presented, two of the more important players for initiating an immune cascade, helper T lymphocytes and macrophages, are specifically inhibited by cold.

Finally, it is also important to consider the microvasculature changes to cold, both local and throughout the body. Viscosity of the blood increases with cold, due in part to the aggregation of red blood cells and the increased adhesion of white blood cells to the endothelium.[128] Capillary occlusion is possible, leading to hypoxic damage. Endrich and colleagues[128] report a result different from many others; namely, an observed increase in the permeability of the chilled vessels to macromolecules, leading to some leukocyte extravasation before the increased adherence of these cells. Overall, cold affects the immune response not only by inhibition of specific cells but also through blood cell and vascular changes, such as alterations in viscosity of the blood and permeability of the vessels.

Acclimation may play a major role in attenuating a response to an acute cold stress. Kizaki and colleagues[129] reported that in response to an acute cold stress, cold-acclimated mice exhibited a significant attenuation of the increases in serum cortico-sterone levels and the expression of the GC-receptor messenger RNA on peritoneal exudate cells. If one can extrapolate from these studies to humans, it is conceivable that humans who are acclimated to cold may be able to withstand a cold stress and minimize any major alterations in their immune response.

MILITARILY RELEVANT ISSUES

There are several militarily relevant aspects of managing the cold casualty in the field, including resuscitation, rewarming, and human tolerance to cold environments. Medical officers should keep in mind that the time available to resuscitate hypothermic casualties is prolonged because of their slowed metabolism. As the familiar saying implies, "You are not dead until you are warm and dead."[130] In addition, we should never underestimate the difficulty of carrying out seemingly simple interventions in a combat zone. It is clear that still-unresolved problems of field resuscitation are areas in which the military medical research establishment can play an important role.

Resuscitation and Rewarming

Chapter 14, Clinical Aspects of Freezing Cold Injury, contains an extensive discussion concerning various rewarming modalities in the field and in the hospital. In many situations, military personnel may be faced with rewarming a person in the field. For mild hypothermia, having the person drink warm fluids and removing him or her from a cold environment should be more than adequate. After approximately 30 minutes of mild hypothermia, mild exercise is a very effective way to rewarm a victim of hypothermia. However, in certain situations, rewarming involving external methods may be implemented. Exhibits 11-4 and 11-5 list major rewarming techniques and contraindications to cardiopulmonary resuscitation (CPR) that any rescue group needs to consider. In a field operation, the options for rewarming are limited, and many methods of rewarming that have been proposed over the years may not be effective.

One point should be emphasized: body-to-body rewarming is *not* an effective technique. Giesbrecht and colleagues[131] reported that in humans who were made mildly hypothermic by immersion, shivering in a sleeping bag was just as effective as body-to-body rewarming. In fact, the hypothermic subject's shivering was blunted by body-to-body rewarming. In their conclusions, the authors recommend that subjects who are mildly hypothermic should be removed from their environment and rewarmed. In the field, when logistical considerations prevent evacuation, they recommend any form of external heat, including direct body-to-body contact. Such a recommendation is fraught with a number of problems. Over the years, victims of hypothermia have been found together in a sleeping bag, dead. More than likely, these deaths were a consequence of the mistaken assumption that one person can adequately rewarm another who is hypothermic. If the hypothermic individual is shivering, the addition of a warm body will suppress shivering. If the victim is severely hypothermic and is not shivering,

EXHIBIT 11-4

REWARMING TECHNIQUES*

- Endogenous Rewarming
  - Basal metabolism
  - Shivering
  - Exercise
- Passive External
  - Thermal stabilization
  - Insulation
- Active External
  - Radiant heat*
  - Hot water bottles*
  - Plumbed garments*
  - Electric heating pads and blankets*
  - Forced circulated hot air*
  - Immersion in warm water*
- Active Internal (Core)
  - Inhalational rewarming
  - Heated infusions
  - Lavage
    - Gastric and colonic
    - Mediastinal
    - Thoracic
    - Peritoneal
  - Extracorporeal blood rewarming
  - Diathermy

*Many methods of rewarming that have been proposed over the years may not be effective in the field, using core temperature change as the key criterion. Logistical ease of use and physiological effectiveness need to be evaluated before the techniques are fielded.

Adapted with permission from Danzl D, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies.* 3rd ed. St Louis, Mo: Mosby–Year Book, Inc; 1995: 72.

the addition of one warm body will not be adequate to rewarm the subject. Also, rewarming may induce rewarming-induced core temperature afterdrop, leading to rewarming collapse (discussed below). Unsubstantiated studies of body-to-body rewarming practices suggest that three normothermic, seminude subjects be placed around the seminude victim of hypothermia, all four in interconnected sleeping bags. Such a solution is neither practical nor recommended, but it does emphasize the fact that one person, no matter how warm, cannot warm a victim of severe hypothermia.[132]

Core Temperature Afterdrop and Rewarming Collapse

The major problem facing the transport of victims of hypothermia is the fact that any form of rewarming may induce major pathological responses of the cardiovascular system, leading to what is called *rewarming collapse*. This problem is so controversial and difficult to control in the field that some have advocated that (1) any hypothermic victim should simply be removed as quickly as possible to a hospital site and (2) minimal efforts should be taken to rewarm the subject in the field. *Core afterdrop* refers to the additional decrease in core temperature that can occur when a hypothermic individual is removed from the cold exposure. The importance of core afterdrop is that, if severe enough, it will trigger syncope and even ventricular fibrillation. Core afterdrop and its effects may be the major explanation for the deaths of victims of hypothermia after they have been rescued and rewarmed.

Core afterdrop has two major mechanisms of action: conductive and convective. As a person becomes hypothermic, a temperature gradient is established between the cooler periphery and the warmer core. Each layer from the core to the periphery is cooler than its immediately superficial layer. When a person is rewarmed, the temperature gradient is reversed, but the temperature of each layer from the core out to the periphery will continue to fall until the layer just superficial to it is warm. This phenomenon has been seen in both inanimate and animate objects.[133] However, afterdrop has another component. As victims of hypothermia are warmed, the process causes their peripheral blood vessels to

EXHIBIT 11-5

CONTRAINDICATIONS TO CARDIOPULMONARY RESUSCITATION IN ACCIDENTAL HYPOTHERMIA

1. Rescuers are endangered by evacuation delays.
2. Obviously lethal injuries are present.
3. Chest-wall depression is impossible.
4. Any signs of life are present.

Source: Danzl DF, Pozos RS, Auerbach PS, et al. Multicenter hypothermia survey. *Ann Emerg Med*.

dilate; the dilated blood vessels then act as conduits for relatively warm core blood to be carried to and cooled by the periphery. A greater afterdrop has been seen in studies[134] in which hypothermic subjects have been gradually rewarmed with increasingly warmer water, which causes greater vasodilation. Thus, from a practical point of view, the faster a hypothermic victim is warmed, the greater will be the afterdrop.[134] Because the number of unknown factors is large, medical officers need to keep in mind that the rewarming of severely hypothermic casualties in the field might induce rewarming collapse.

Another controversial area is the appropriateness of CPR in the field. As previously suggested (see Exhibit 11-5), there are times when CPR may not easily be implemented in the field. The first point listed, that the rescuers themselves should not be endangered by evacuation delays, should be emphasized.

Predictions of Human Tolerance in Cold Environments

As was previously mentioned, attempts have been made to model human thermoregulatory responses in cold environments and to arrive at times for various stages of hypothermia to begin. The complex interaction of various environmental, clothing, and physiological factors allows for the generation of such cooling curves to be extremely conservative. The physiology of the hypothermic individual varies with the degree of core cooling, which does not permit simplistic modeling. For example, individuals whose core temperature falls at a rate of 1.5°C per hour and who breathe 4% carbon dioxide lose core temperature faster than controls.[78] However, if the rate of cooling is three Centigrade degrees per hour, the effect of carbon dioxide is not noticed. The use of various agents (eg, herbs, drugs) has not been extensively studied for effectiveness in enhancing metabolic rate or resistance to cold stress. Nevertheless, in military communities such agents are routinely rumored to be effective—but without a shred of scientific data.

Because it is unethical to mimic various combined stressors (which may be lethal) in human subjects, anecdotal evidence and clinical case histories are the sole sources of survival and endurance data. Although lacking rigorous scientific controls, these sources may suggest various levels of human endurance and may give insight into various other mechanisms that might be at work as a person becomes hypothermic.[130] Furthermore, single case histories are valuable because they present issues that may never appear in a controlled laboratory situation. A specific example of physiological insight gained from field experiences is paradoxical undressing, which is associated with many dead victims of hypothermia. The cause of the undressing is unknown, but it indicates an area of additional research. A note of caution is warranted, however. Rescuers should be wary of undocumented anecdotal stories of persons who can withstand extremely cold environments for prolonged periods. Most of these cases, when studied thoroughly, suggest that the stories are fraught with contradictions or outright falsification.

Challenges for the Military in Future Cold Weather Operations

Although the basic mechanisms of thermoregulation in a cold environment are well documented, there are a number of unanswered physiological questions and challenges that military medical science will have to address. From a military perspective, with the possibility of chemical–biological warfare ever present, the greatest threat to military personnel will be to protect themselves from these agents. Thus, soldiers in cold weather operations who are enclosed in mission-oriented protective posture (MOPP 4) gear face a number of hazards: (1) the toxic environment, (2) the dangers on the battlefield, (3) the build-up of core temperature as they are encapsulated in impermeable protective clothing, and (4) decreased core temperature as their peripheral and core temperatures fall when they remove the MOPP 4 ensemble.

A simple, lightweight microclimate cooling system may be required for soldiers conducting cold weather operations. In the future, many of the soldier's physiological systems (eg, heart rate) will be monitored in the field, and the data will be transmitted to remote sites. A technical system that allows for the monitoring of core temperature in the field is required. Although some systems are available to perform this function, most do not give a robust or consistent recording and are not field hardened. Because most troops must be prepared for 24-hour deployment worldwide, they will not be acclimatized to either hot or cold temperatures. (For a thorough review of human cold acclimatization, interested readers may consult Young's chapter in the *Handbook of Physiology*.)[135] Constant exercises in the cold are required for maintaining combat effectiveness for cold weather operations. Although more "hot spots" are thought to be in hot climates, and hyperthermia may be considered a greater

problem than hypothermia, history teaches us that wars or peacekeeping operations occur in unexpected places (eg, Bosnia, Serbia, North Korea).

Possibly the greatest challenge for cold weather military operations will be to adequately train and teach personnel the straightforward facts about the body's robust response to cold stress. Ignorance about cold stress may be as lethal as any toxic material in the cold environments in which the military conducts its operations.

SUMMARY

Cold environments have proven to be the nemesis of many a well-planned military campaign. The insidious nature of the decrease in core temperature is the fundamental underpinning of the induction of mild to moderate hypothermia for supposedly well-trained and well-prepared military troops. Because the military has personnel who come from various geographical regions, the ability to use only specifically designated troops for cold weather operations is not practical. The military has researched various ways to minimize the onset of hypothermia by evaluating various kinds of cold weather gear and cold weather rations, as well as by understanding the physiology of cold response and the pathophysiology of hypothermia. Although it is often stated that there is nothing to research in the area of thermoregulation in the cold, many practical observations indicate that there are areas of research that are critical for effectively performing military operations in cold environments. The military has pioneered the use of models to predict the onset of hypothermia. However, the variability in human response based on the physiology of the troops makes this a laudable but not achievable goal in the real world.

The control of the core temperature is dependent on the peripheral and central thermal information sent to the hypothalamus and other areas of the brain. The peripheral thermoreceptors are extremely powerful in driving the initial physiological responses to cold stress. Peripheral vasoconstriction, shivering, and tachycardia are all induced by the cold stimulus. Interestingly, cold receptors adapt to the cold stimuli, thus decreasing their effect on increasing the activity of the somatic and autonomic nervous systems. The range of human response to cold stress suggests that humans have different thresholds for peripheral cold receptor activation. Simplistically, it is thought that various areas of the brain, especially the hypothalamus, compare the peripheral temperatures with the core temperature. By methods still not understood, the CNS is able to activate various responses based on the difference, or the rate of difference, between the periphery and the core. The deleterious effects of mild hypothermia on higher brain function (eg, impaired memory, slurred speech) are commonly reported. However, the first response to cold stress is the behavioral one in which the affected person attempts to minimize the cold by altering the immediate environment. Such activities as huddling, walking faster, and attempting to get out of the wind are all manifestations of the attempt by the CNS to maintain core temperature.

In military situations in which personnel cannot escape the cold environment, tents and sleeping bags are the first line of defense after the personnel gear that the subjects wear. In the event of a breakdown of logistics so that personnel cannot sleep, eat, and drink water in a cold environment, the beginnings of secondary hypothermia will soon show their effect. In the battlefield, the signs of hypothermia will be evident in individuals working with computers or other skills requiring fine motor control and good decision-making skills.

A decrease in core temperature will affect every component of a physiological system: neural innervation to the organ, blood flow to the organ, receptors and other chemicals on the surface of the cells that compose the organ, and hormonal control of the cells and the organ itself, which will then affect other organ systems. All physiological systems are affected by a decrease in core temperature, some more so than others.

Once the physiological systems cannot maintain adequate core temperature, various systems begin to shut down. Of all systems, the most important is the cardiovascular, because cold core temperatures will eventually cause asystole or ventricular fibrillation and ultimately death. Many of the effects of the cold on the heart and vasculature and blood would be of only academic interest were it not for the fact that a hypothermic person is capable of being rewarmed and revitalized. Understanding the pathophysiology of hypothermia is key toward maintaining the well-being of a rewarmed cold casualty. Owing to the life-sparing qualities of hypothermia, the focus in many rescue attempts has been on returning the core temperature to normal values. This approach has its pitfalls.

Although rescue attempts usually concern themselves with thermal stability, the key systems that need to be stabilized in a hypothermic state are the same as in a normothermic state: namely, the cardiovascular and the respiratory systems. Even assuming that these systems have been stabilized, the threat of core afterdrop leading to rewarming collapse remains ever present, especially in field situations. The critical importance of rewarming must be addressed simultaneously with stabilizing the pH level.

Although it has not been vigorously studied, the effect of a decrease in either peripheral or core temperature on the immune response is very important in a field situation. Limited studies suggest that cold inhibits many of the immune responses.

In the real world of men and women being involved in field operations, hypothermia is an ever-present nemesis that attacks the weak and weary. Models that predict human cooling curves and hence survivability cannot ethically be tested and therefore can give only crude estimates of time in terms of human survivability. Each branch of the military service will expose its personnel to cold environments that can insidiously lead to hypothermia. The incidence of hypothermia can be minimized only by scrupulous attention to the state of each individual by the officers in charge. In this era in which technology promises mastery over the environment, it is important to be watchful for the breakdown in logistics that would eventually lead to major casualties due to hypothermia.

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Chapter 12

HUMAN PSYCHOLOGICAL PERFORMANCE IN COLD ENVIRONMENTS

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INTRODUCTION

Military personnel are expected to perform at optimal levels in all varieties of hostile environments, including cold and extreme cold. The cold can be a potent stressor in field conditions, causing both a deterioration in morale and decrements in performance, including the performance of mission-sensitive duties. This chapter reviews those decrements in human psychological performance that are expectable in mild, moderate, and extreme cold environments in an attempt to provide guidance in framing reasonable performance expectations. In addition, this chapter highlights areas where additional training or pretraining may prove beneficial, including the possible role of acclimatization as a method of attenuating performance decrements. Finally, some attention is drawn to possible central nervous system correlates or determinants of decreased performance in the cold.

An important distinction should be kept in mind in the discussion of the differences in magnitude between exposure to cold air and exposure to cold water. Cold water immersion causes a convective heat loss that is 25 times greater than exposure to cold air at an equivalent temperature, and it places substantially heavier thermoregulatory demands on the body.[1] The thermal conductivity of water has been estimated to be 1,000 times greater than cold air at similar temperatures.[2] In general, the largest performance decrements on psychological measures are seen with individuals immersed in cold water, or wet or partially wet individuals who are also exposed to cold air. Duration and severity of the cold stress are modifiers that will affect performance, as will the presence or absence of turbulence in cold water or wind in cold air.

PROLONGED SEVERE COLD STRESS, HYPOTHERMIA, AND BEHAVIOR

Fig. 12-1. This experimental subject, dressed in full arctic clothing, was exposed to prolonged, severe, cold air stress (–30°F for 3 h) in the Hypothermia and Water Safety Laboratory, School of Medicine, University of Minnesota, Duluth, Minn. Subjects in these conditions demonstrate significant decreases in both core body and peripheral temperatures. Photograph: Mr Dan Schlies, Educational Resources Department, School of Medicine, University of Minnesota, Duluth, Minn.

Cold stress causes a complex set of physiological and psychological responses, often before there is a drop in deep body temperature (ie, rectal or other core body temperature). These responses are dependent on the nature and severity of the cold thermal stress applied. The drop in deep body temperature that follows prolonged cold stress is typically gradual and may exacerbate previous performance decrements or create new ones. When deep body temperature falls below the normal resting range of body temperature (around 37°C), it can be regarded as a beginning stage of hypothermia, but hypothermia is generally defined clinically as a deep body temperature below 35°C (or about 95°F) (Figure 12-1).

The effects of deep body cooling on overt behavior are similar to the effects of general anesthesia. Levels of consciousness and alertness gradually decrease as body temperature drops toward 30°C.[3] Responses become slow and reflexes sluggish; speech becomes slurred and increasingly difficult. Mobility is impaired, and individuals often become drowsy or apathetic.

As will be noted in detail later, there is evidence to suggest some impairment in memory registration, beginning at a core temperature of 36.7°C and progressing to a point at about 34°C to 35°C, at which 70% of information normally retained is lost.[4] Concentration becomes increasingly impaired, although at early stages of hypothermia (34°C–35°C) the impairment appears to be in speed of mental operations rather than accuracy. Although lowered

body core temperature also slows the performance of complex calculation tasks, and probably slows performance of reasoning tasks as well, such slowing is not accompanied by any loss of accuracy—provided that adequate time is allowed for the task to be completed. The slowing observed, however, is substantial, with calculations being about 175% slower at a core temperature of 34.2°C.[4]

As the body cools further, casualties of cold stress may become increasingly confused and even incoherent as hypothermia progresses below 34°C to 35°C. Cold stress-induced or hypothermia-induced auditory and visual hallucinations are a not uncommon occurrence as core temperature drops below 35°C and are occasionally reported by individuals exercising in the cold as well.[5,6] Voluntary movements gradually become slower and a simple movement such as touching the nose (normally accomplished within 1 sec when warm) may take a casualty 15 to 30 seconds as his core temperature approaches 30°C. Muscle rigidity, sometimes accompanied by neck stiffness, is often striking at this point, making it difficult to extend the limbs. Gait may become ataxic and deep tendon reflexes may decrease. At a core temperature of 30°C to 31°C the pupils of the eyes may react so slowly that the light reflex may be wrongly assumed to be absent, although it *will* essentially disappear at lower temperature.[3] Consciousness is usually lost between 32°C and 30°C, although there are exceptions to this. At a core temperature of 27°C a casualty may sometimes grunt when questioned, but below about 26°C he usually fails to respond to any stimulus whatsoever, including deep pain. If temperature continues to drop the casualty will lapse into coma.

HYPOTHERMIA AND BEHAVIOR: POSSIBLE MECHANISMS OF ACTION

One factor that might account for slower performance by extremely cold or hypothermic individuals is a direct slowing of synaptic transmission. A fall in central (core) temperature from 37°C to 22°C has been shown to increase the rise time of the end-plate potential in cat muscle to 200% and to increase the fall time to 260% of control values.[4]

The mechanism responsible for cold stress-induced auditory and visual hallucinations is not clear, although there has been some speculation that this may relate to dopaminergic supersensitivity.[5] The hypothesis has been advanced[5] that all hallucinations, and other disturbances of perception, involve the dopamine/5-hydroxytryptamine (5-HT, or serotonin) system and are a result of an overload at a rate-limited step in the degradation pathway of 5-HT. In prolonged heat stress or fever, in which hallucinations can also occur, there is an excessive production of dopamine, which effects a lower body temperature. Dopamine release activates dopaminergic receptors, which cause lowering of the body temperature either directly or via intermediate 5-HT and receptors.[7] During prolonged exposure to cold, dopamine receptors could therefore become supersensitive.

It is possible that at least some of the observed decrements in human performance that occur following prolonged extreme cold stress and during hypothermia may be determined by (*a*) direct or indirect effects of brain cooling, (*b*) changes in brain cerebral blood flow, or (*c*) changes in the electrical activity of the brain[8]; therefore, each of these areas will be reviewed briefly.

Hypothermia and Brain Cooling

Local brain cooling first increases and then decreases neural excitability. The effects of cooling appear to be reversible, and the changes in temperature needed for reported changes in brain function are one Centigrade degree or less in various areas of the nervous system.[9] Spinal and cerebral neurons are reported to become hyperresponsive when only one or two Centigrade degrees below normal.[10] In a series of studies, Hayward and Baker[11] showed a significant amount of nonuniformity in the temperatures in various areas of the brain in dogs, sheep, monkeys, and cats. It is possible that localized brain cooling could occur in humans as well, a possibility of great significance to the military, for localized brain cooling can greatly influence the selective responses of certain areas of the brain, and thus human performance.

As the arterial blood passes through the preoptic-anterior hypothalamus, the temperature of the blood determines the temperature of the brain.[12] In mammalian species that possess internal carotids, cerebral arterial blood temperature oscillations mirror those found in systemic arterial blood. In the monkey, the prototype of the internal carotid species (because monkeys can be subjected to experimental manipulations), accelerated cutaneous and upper respiratory heat losses lead to a sequential and parallel cooling of local venous blood, central venous blood, aortic arterial blood, cerebral arterial blood at the circle of Willis, and various brain sites.[11] We have recently demonstrated similar results in our labo-

ratory using the bypass-cooled dog.[8] From this and other evidence we can conclude that

- the arterial blood removes heat from the brain during hyperthermia,
- venous blood circulation buffers rapid brain cooling during hypothermia, and
- brain–blood temperature gradients are the major determinant of fluctuations in brain temperature.

Effects of Hypothermia on Cerebral Blood Flow

The few studies conducted to date on cerebral circulation and oxidative metabolism during hypothermia indicate that cerebral blood flow (CBF) and cerebral metabolic rate for oxygen ($CMRO^2$) decrease with body temperature.

We might conclude that progressive reductions in body temperature produce progressively greater decreases in cerebral metabolism. Bering and colleagues,[13] measuring the cerebral metabolic response of monkeys to hypothermia, have demonstrated that this reaction does not occur. Stone, Donnelly, and Frobese[14] confirmed Bering's findings and discovered that in the anesthetized human, the sharpest drop in cerebral metabolism occurs with body core temperature reductions down to 28°C, and that further temperature reductions could not produce a correspondingly greater depression of cerebral oxygen utilization.

During hypothermia, cerebral vascular resistance increases despite elevated arterial carbon dioxide tensions. This increase may be due to two factors:

1. Hemoconcentration in response to hypothermia, reported previously by Prec and colleagues[15] and confirmed by Stone and colleagues,[14] increases blood viscosity and cerebral vascular resistance.
2. Cerebral vessels may constrict in much the same manner as peripheral vessels in a response to hypothermia.

In the absence of shivering, cerebral oxygen consumption is sharply reduced at body temperature of 28°C to 29°C. Ehrmantraut, Ticktin, and Fazekas[16] studied the effects of marked hypothermia on CBF and $CMRO^2$ in two unanesthetized human adult male victims of accidental hypothermia, one with a core temperature of 30°C and one with a core temperature of 23°C (this patient required bypass rewarming). In both cases, CBF and $CMRO^2$ decreased when the subjects were hypothermic. When the men were rewarmed, both values returned to normal levels. In light of what is now known of the delicate coupling of CBF and cerebral metabolism,[17] it is likely that CBF was decreased to match the lowered metabolic rate imposed by hypothermia.[12]

Similar results were also reported in humans[14] and in dogs,[18,19] the latter investigators using the radioactive microsphere technique. Anzi and colleagues[20] also utilized this technique to measure CBF in more discrete regions of the brain. A remarkably uniform decrease in regional CBF was noted throughout the brain, ranging from 62% in the brainstem to 82% in the posterior lobe. In another, more-recent study, Steen and Milde[21] studied the effects of prolonged, 24-hour hypothermia and subsequent rewarming in dogs. When the dogs were rewarmed, $CMRO^2$ increased but CBF increased to only 30% to 40% of normal levels, suggesting some possible metabolic problems in cases of prolonged hypothermia.

The effects of central core cooling on cerebral $CMRO^2$, CBF, and glucose substrates may also at least partially account for the observations of slowed mental processing in hypothermic individuals. Most neuroscientists would agree that information treatment in the brain is handled by neurons that transmit the information in the form of action potentials. This in turn leads to potential changes in the target neurons. The synaptic activity, ionic transfers, transmitter synthesis, release and reuptake, formation of second and third messengers, and protein phosphorylation are all energy-demanding processes that deplete energy-rich phosphates. Thus it could be expected that the regional neuronal metabolism should increase during and immediately after regional information processing. Because the local CBF is adjusted to the local metabolic demands, one would also assume that the blood flow would increase. Mental activity—initiated, produced, and evaluated by the brain itself in normothermic individuals—generally also gives rise to $CMRO^2$ increases in multiple cortical fields and subcortical structures. These short-term metabolic changes are almost exclusively the consequence of increased synaptic activity in locations where the information transformation takes place. The amount of metabolic increase in an active field during mental activity is fully equivalent to the increases in motor or sensory fields during intense voluntary movements or during intense perception.[22] The reduced CBF and decreased metabolic rate that occurs with profound core body cooling, however, may not allow efficient information processing by hypothermic individuals.

Effects of Hypothermia on Evoked Responses

In general, the effects of hypothermia on nerve conduction can be summarized as follows[9]:

1. progressive slowing of axonal conduction,
2. abolition of synaptic transmission prior to conduction failure (postsynaptic activity is abolished at approximately 20°C, whereas axonal conduction does not fail until temperatures lower than 10°C are achieved), and
3. an increased excitability at mild degrees of hypothermia, which precedes the subsequent synaptic depression at lower levels of hypothermia.

Hypothermia also produces several neurophysiological changes such as decreased resting potential, decreased amplitude but increased duration of the action potential, a reduction of nerve conduction velocity, and depression of synaptic transmission due to impaired transmitter release. The changes in evoked responses with hypothermia can be explained by two basic hypothermic effects on neural activity: slowing of the conduction along the axons, and increased synaptic delay. The effect on synaptic transmission appears to be more profound than on the axonal conduction.

Several investigators[23–25] have found that latencies of each component wave of the brainstem auditory evoked response (BAER) progressively increased as a function of decreasing temperature, and that the effect was more profound on the later than on the earlier components. Individual BAER components show a slower rise time and become longer in duration under hypothermic conditions. The latency of BAER component waves and the interpeak latency (IPL) increase exponentially rather than linearly, as a function of decreasing temperature over the entire range of hypothermia (36°C–20°C). We have seen the same pattern of increased latency of the BAER in our laboratory, and it appears to be present with drops in core temperature as modest as one to two Centigrade degrees.

Fitzgibbon and colleagues[26] found a 10% to 20% increase in BAER latency when unanesthetized human subjects are cooled about four Centigrade degrees, which is consistent (in terms of temperature sensitivity) with the data of Marshall and Donchin.[27] These investigators showed latency increases of the auditory evoked response of 6% to 7% during the normal sleep phase of the circadian rhythm when core temperature was about two Centigrade degrees below daytime normothermia.[27] We have observed similar latency increases, again often beginning at one to two Centigrade degrees of core temperature cooling.[8] These studies indicate that evoked responses in humans are slowed during cooling by about 3% to 4% per Centigrade degree. The 10% to 20% increases in latency of peaks in the visual evoked potentials that were observed in some subjects are of the same magnitude as those described by others for evoked responses in a variety of mammals at approximately the same temperature of 33°C to 34°C.

A number of laboratories, including our own, have studied changes in somatosensory evoked responses (SERs) following core body cooling and cold exposure (Figure 12-2). Benita and Conde[28] locally cooled

Fig. 12-2. This experimental subject is exposed to mild cold stress in evoked potential experiments measuring the effect of mild external cold stress (90-min exposure to 7°C air) versus mild internal cold stress (ingestion of ice slurry). Subjects in the mild cold air exposure condition demonstrate increased central nervous system arousal, resulting in decreased latencies in visual evoked potentials and decreased reactions times, in the absence of significant decreases in core temperature.
Subjects in the ice slurry condition demonstrate significant decreases in core body temperature and significant reductions in nerve conduction velocity, accompanied by increased latencies in auditory and visual evoked potentials. They do not demonstrate changes in reaction time. Experiments conducted at the Hypothermia and Water Safety Laboratory, School of Medicine, University of Minnesota, Duluth, Minn. Photograph: Mr Dan Schlies, Educational Resources Department, School of Medicine, University of Minnesota, Duluth, Minn.

a small nuclear region in the brain and found that the latencies of the presynaptic and postsynaptic responses were delayed. The SER in response to induced hypothermia has been studied by several investigators,[29,30] whose results suggest that peripheral and brainstem nerves seem to have comparable behaviors during hypothermia. Our studies with nonanesthetized humans who were internally cooled by ingesting an ice slurry drink also suggest increases in the latency of the SEP response, even at modest levels of core body cooling.

In relation to observations of considerable behavioral alteration in persons who are mildly hypothermic (34°C–36°C) in accidental or experimental situations, these findings suggest that if such behavior is a direct result of brain cooling, then the subtle alterations of electroencephalograms and visual evoked potentials that have been observed here may be correlates of this phenomenon. Minor electrical changes may be associated with detectable impairment of cognitive processes.

Central nervous system mechanisms, in tandem with peripheral physiological effects and more purely psychological effects, may well have an additive effect and produce many of the impairments in human performance that have been observed. Further empirical investigation is needed in this area to delineate the components that are determinants of performance decrements for a given cold stress and a given psychological or psychomotor task.

EFFECTS OF MILD-TO-MODERATE COLD STRESS AND BEHAVIOR

Manual Performance

A number of studies in both cold air and cold water have demonstrated substantial performance decrements in manual performance following cold stress, the magnitude of which appears to be a function of both surface cooling and, with prolonged exposures, deep body cooling (Figure 12-3). When the entire body is cooled, there is likely a local effect at the periphery involving direct interference of sensory-motor functioning, and a general and central effect influencing higher centers that serve to control, direct, and coordinate action.[31] For example, if the hands are preferentially cooled in air to 13°C and the rest of the body is kept warm, then reliable decrements in manual performance are observed, which tend to increase for the first 40 minutes of exposure and change little if at all during the remainder of the first hour of exposure.[32,33] Cooling of the remaining body surface as low as 26°C does not cause further measurable decrement.[34] Alternatively, if the hands are kept warm, then impairments in manual performance are observable when the rest of the body surface area is cooled to 21°C, although this impairment is less pronounced than when the hands alone are cooled to 13°C.[35] If body surface temperatures and hand surface temperatures are simultaneously reduced to 21°C and 13°C, respectively, a greater level of impairment occurs, and if body core temperatures are also dropping, the greatest manual performance decrements occur.

Fig. 12-3. This experimental subject, exposed to 0°C air for 120 minutes, is shown completing a standardized dexterity measure, the Grooved Pegboard Test. Following 120 minutes of exposure, the average time to complete this task with the dominant hand increases from 55 to 78 seconds. Experiments conducted at the Hypothermia and Water Safety Laboratory, School of Medicine, University of Minnesota, Duluth, Minn. Photograph: Mr Dan Schlies, Educational Resources Department, School of Medicine, University of Minnesota, Duluth, Minn.

At extreme cold temperatures, losses of sensitivity and manipulative ability are amplified by heavy protective garments worn to buffer heat loss. This decrease in manual performance is further compromised by loss of flexibility in the muscles of the forearm and finger, increase in muscle viscosity of the extensors and flexors of the fingers, and difficulty

bending joints owing to an increase in joint synovial fluid viscosity.

Tactile Sensitivity

Perhaps the most common noninjurious effect of cold exposure is numbing of the extremities. Possible causes of numbness and the degree of numbness occurring under given ambient conditions have been extensively examined for more than 50 years. Fox[36] and Weitz[37] lowered subjects' forearm skin temperatures 12°C and reported impairment of sensitivity to vibratory stimuli in proportion to the magnitude of cooling. Mackworth[38] reported preliminary data for what he called a "biological index of numbness" related to two components of windchill-air temperature and wind velocity-and much of the later work in this area included the examination of windchill effects (Figure 12-4).

To assess finger numbness, Mackworth developed the "V-test," which has become a standard in numbness assessment. The V-test apparatus consists of a flat wooden ruler cut in half, the two halves laid side by side and joined at one end with a bolt and permanently separated by a one-half inch gap at the other end, forming a V shape with a "gap" that varies in size from 0 to 13 mm. Finger numbness is assessed by laying a finger across the two sides of the V and asking subjects to report when they feel two edges. Tactile discrimination is estimated by the gap size (in millimeters) necessary for the subject to report the presence of two edges. Mackworth[38] reported data on changes in index finger numbness of 35 subjects examined in subarctic Canada using the V-test across two temperature ranges (–25.1°C to –30°C vs –30.1°C to –35.0°C) with varying wind velocities. Each subject was tested while wearing a thick glove with the index finger portion removed. It was found that the rate of increase of numbness in these experiments was largely a function of wind velocity rather than temperature, although this may be an artifact of the narrow temperature ranges used. Higher wind velocities also produced a more prolonged effect with a longer required recovery period. At the coldest temperature sampled and a wind velocity of 6.1 to 10.0 mph, numbness began in 1.2 minutes, versus 2 minutes at the same temperature in winds below 6.0 mph. Later, Mackworth[39] observed a similar pattern of numbness onset in both 4 mph wind conditions and no wind conditions, at ambient temperatures of –16°C and –25°C.

Mills[40] examined pressure sensitivity of bare fingers at –21°C and reported a 4-fold pressure threshold increase in the cold, measured in grams of pressure necessary for subjects to feel the contact from a slowly lowering rod. Morton and Provins[41] examined subjects whose hands were locally exposed to cold but whose bodies were otherwise kept warm (the hand was isolated in a miniature cold chamber). As air temperature to the hand was reduced from 32.5°C to 2.5°C, finger numbness increased in an accelerated fashion. A second study by the same authors[42] investigated isolated cold water stress and finger numbness during a 40-minute immersion of the hand in cold water. Finger sensitivity was reported to be significantly-and essentially equally-impaired in water baths at 6°C, 8°C, 15°C, and 30°C, with some further decrease in the sensitivity only when water baths of 2°C and 4°C were used. Bowen[31] demonstrated substantial impairment of hand tactile sensitivity following a 2-minute exposure to 8°C water, and an enormous 336% decrease in hand tactile sensitivity following a 24-minute exposure.

A number of studies have identified skin or extremity temperature as the relevant variable that predicts impairment in manual dexterity and tactile sensitivity (Table 12-1). Mackworth[38] reported very little systematic change in V-test performance until the measured skin temperature reached 10°C to 15°C or below. Morton and Provins[41] were in general agreement with this finding, but noted measurable V-test impairments beginning at hand skin temperatures of 20°C to 25°C. They suggested that the relationship between skin temperature and numbness followed an L-shaped distribution, with relatively little impairment in sensitivity and performance at skin temperatures above 8°C, but a very rapid drop-off in performance below an approximate threshold temperature of 6°C to 8°C. They hypothesized that nerve fibers, skin receptors, or some combination of the two are subject to "cold block," or loss of neural activity below 6°C and 8°C, and therefore are no longer capable of excitation by a new stimulus. Available evidence suggests that the pain threshold for a cold stimulus is also in this range—about 10°C—in all but the most highly acclimatized subjects.[43]

Dexterity

Several researchers have also investigated cold stress and its effects on dexterity (Table 12-2). Springbett[44] was the first experimenter to systematically investigate the effect of local cooling and overall skin cooling on dexterity. Using the Minnesota Manual Dexterity Test, Springbett demonstrated significant impairment in 16 subjects exposed to –4°C for 70 minutes, irrespective of the

Estimated wind speed* (km/h mile/h)	Actual Thermometer Reading (°C/°F)											
	10 50	4.4 40	-1.1 30	-6.7 20	-12.2 10	-17.8 0	-23.3 -10	-28.9 -20	-34.4 -30	-40 -40	-45.6 -50	-51.1 -60
	Equivalent Chill Temperature (°C/°F)											
Calm	10 50	4.4 40	1.1 30	6.7 20	12.2 10	17.8 0	23.3 -10	28.9 -20	34.4 -30	40 -40	45.6 -50	51.1 -60
8 5	8.9 48	2.8 37	2.8 27	8.9 16	14.4 6	20.6 -5	26.1 -15	32.2 -26	37.8 -36	43.9 -47	55.6 -68	55.6 -68
16 10	4.4 40	2.2 28	8.9 16	15.6 4	22.8 -9	31.1 -24	36.1 -33	43.3 -46	50 -58	56.7 -70	63.9 -83	70.6 -85
24 15	2.2 36	5.6 22	12.8 9	20.6 -5	27.8 -18	35.6 -32	42.8 -45	50 -58	57.8 -72	-102.8 -85	72.8 -99	-80 -112
32 20	0.0 32	7.8 18	15.6 4	23.3 -10	31.7 -25	39.5 -39	47.2 -53	-55 -67	63.3 -82	71.1 -96	73.3 -110	86.7 -124
40 25	1.1 30	8.9 16	17.8 0	26.1 -15	33.9 -29	42.2 -44	50.6 -59	58.9 -74	67.7 -88	75.6 -104	83.3 -118	91.7 -113
48 30	2.2 28	10.6 13	18.9 12	27.8 -18	35.2 -33	44.4 -48	52.8 -63	61.7 -79	70 -84	78.3 -108	87.2 -125	95.6 -140
56 35	2.8 27	11.7 11	20.0 4	29.4 -21	37.3 -35	46.1 -51	55.0 -67	63.3 -82	72.2 -88	80.6 -113	88.4 -129	98.3 -145
64 40	3.3 26	12.2 10	21.1 -6	29.4 -21	38.4 -37	47.2 -53	56.1 -69	102.8 -85	73.3 -100	82.2 -116	91.1 -132	100 -148
	Little Danger (in 5 h with dry skin) Maximum danger is from the false sense of security				**Increasing Danger** Danger from freezing of exposed flesh within 1 min			**GREAT DANGER** Flesh may freeze within 30 sec				
Nonfreezing cold injuries may occur at any point on this chart												

Fig. 12-4. Cooling power of wind on exposed flesh, expressed as an eqivalent temperature (under calm conditions)
*Wind speeds > 64 km/h (40 mph) have little additive effect.
Adapted from US Army Research Institute of Environmental Medicine. *Sustaining Health and Performance in the Cold: Environmental Medicine Guidance for Cold-Weather Operations.* Natick, Mass: USARIEM; July 1922: 37. Technical Note 92-2.

TABLE 12-1

COLD STRESS AND TACTILE SENSITIVITY: SUMMARY OF SEVEN STUDIES

Study	Temperature (°C)	Exposure Time	Results
Weitz (1941)[1]	12°C	*Varied (by forearm skin temp)	Impairment to vibratory stimuli
Mackworth (1953)[2]	–25.1°C to –30°C; –30.1°C to –35°C; plus varied wind	*Varied by hand temp, numbness occurred as early as 1.2 min following exposure at –35°C	Rate of numbness on V-test a function of wind velocity primarily; no impairment until skin temp 2 10°C–15°C
Mackworth (1955)[3]	–16°C; –25°C; + 0 mph; or 4 mph wind	*Varied by hand temp	Similar pattern of numbness both conditions
Mills (1956)[4]	–21°C	21 min	4-Fold pressure threshold increase in cold
Morton and Provins (1960)[5]	Hand temp reduced from 32.5°C to 2.5°C	*Varied by hand temp; numbness typically observed following 3–4 min of exposure	Numbness increased in accelerated fashion when hands cooled but body kept warm. V-test impairments at hand temps of 20°C–25°C, sharp dropoffs at 6°C–8°C
Provins and Morton (1960)[6]	Hand exposed to 2°C, 4°C, 6°C, 8°C, 15°C, or 30°C water temps	40 min	Equal numbness at 6°C, 8°C, 15°C, and 30°C; increased numbness at 2°C and 4°C
Bowen (1968)[7]	Hand exposed to 8°C water temp	2–24 min	Substantial impairment at 2 min; 336% decrease in sensitivity at 24 min

*The criterion was a targeted skin temperature rather than a fixed exposure time.
Temp: temperature
Data sources: (1) Weitz J. Vibratory sensitivity as a function of skin temperature. *J Exp Psychol.* 1941;28:21–36. (2) Mackworth NH. Finger numbness in very cold winds. *J Appl Physiol*. 1953;5:533–543. (3) Mackworth NH. Cold acclimatization and finger numbness. *Proc Royal Soc*. 1955;143:392–407. (4) Mills AW. Finger numbness and skin temperature. *J Appl Physiol*. 1956;9:447–450. (5) Morton R, Provins KA. Finger numbness after acute local exposure to cold. *J Appl Physiol.* 1960;15:149–154. (6) Provins KA, Morton R. Tactile discrimination and skin temperature. *J Appl Physiol.* 1960;15:155–160. (7) Bowen HM. Diver performance and the effects of cold. *Hum Factors*. 1968;10(5):445–464.

temperatures of the rest of their bodies. Leblanc[45] discovered that cooling the structures of the forearm caused an equivalent or even greater decrement in dexterity than local cooling of either the fingers or the hand, a finding later replicated by Clarke, Hellon, and Lind.[46] Gaydos and Dusek[34] replicated the Springbett[44] findings and observed that significant impairments in block stringing and knot tying occurred when hand skin temperature was lowered to approximately 11.5°C, but no decrement was observed when hand temperatures were maintained at 27°C or higher, even when the rest of the body surface was at a lower temperature (25.5°C).

Clark and Cohen[47] identified a criterion hand skin temperature of 16°C as the minimum temperature at which no significant decrements in dexterity performance on a complex knot tying task occurred, whereas substantial errors occurred at hand skin temperatures at and below 13°C. They also reported that a greater decrement of performance occurred when the subjects' hands were cooled slowly as opposed to quickly, that a sizable decrement in performance persisted even after rewarming in subjects who had been cooled slowly, and that the rate of rewarming was directly related to the rate of cooling.

Bowen[31] demonstrated 20% decrements in four measures of hand manipulative functioning following 20-minute exposure to 22°C water, 25% decrements following 20-minute exposure to 17°C water, and 45% decrements following 20-minute exposure to 8°C water. Kiess and Lockhart[48] demonstrated that local hand warming is capable of preserving some dexterity, despite moderate lowering of skin body

TABLE 12-2

COLD STRESS AND DEXTERITY: SUMMARY OF EIGHT STUDIES

Study	Temperature (°C)	Exposure Time	Results
Springbett (1951)[1]	–4°C air	70 min	Significant impairment on Minnesota Manual Dexterity Test, whether body was warm or cooled
Leblanc (1956)[2]	Forearm and/or hand cooled	Varied, up to 10 min	Cooling forearm caused equal or greater impairment than cooling fingers or hand
Gaydos and Dusek (l958)[3]	11.5°C to 27°C skin temps	Varied according to skin temp	Dexterity (knot tying/block stringing) impaired at low temperature; effect related to hand-skin temperatures
Clark and Cohen (1960)[4]	Hand in water; hand temps from < 13°C and > 16°C	—?	Hand-skin temperature of 16°C identified as minimum temp below which impairments in dexterity occur
Bowen (1968)[5]	Hand in water at 8°C, 17°C, or 22°C	20 min	20% decrements in 22°C water; 25% decrements in 17°C water; 45% decrements in 8°C water
Bensel and Lockhart (1974)[6]	–7°C; 16°C	180 min; 180 min	Decrements in six manual tasks at –7°C; greatest decrement with fastest rate of cooling
Davis et al. (1975)[7]	5°C water (whole body)	35–50 min	17% decrement in dexterity immediately, but not progressive
Enander (1987)[8]	5°C	90 min	13% decrement in hand dexterity

—?: Unknown

Data sources: (1) Springbett BM. The effects of exposure to cold on motor performance. Toronto, Ontario, Canada: Defense Research Board of Canada; 1951. (2) Leblanc JS. Impairment of manual dexterity in the cold. *J Appl Physiol*. 1956;9:62–64. (3) Gaydos HF, Dusek ER. Effects of localized hand cooling vs total body cooling on manual performance. *J Appl Physiol.* 1958;12:377–380. (4) Clark RE, Cohen A. Manual performance as a function of rate of change in hand temperature. *J Appl Physiol.* 1960;15:496–498. (5) Bowen HM. Diver performance and the effects of cold. *Hum Factors*. 1968;10(5):445-464. (6) Bensel CK, Lockhart JM. Cold-induced vasodilatation onset and manual performance in the cold. *Ergonomics*. 1974;17:717–730. (7) Davis FM, Baddeley AD, Hancock TR. Diver performance: The effect of cold. *Undersea Biomed Res.* l975;2(3):l95–2l3. (8) Enander A. Effects of moderate cold on performance of psychomotor and cognitive tasks. *Ergonomics.* l987;30(l0):l43l–l445.

temperature. Bensel and Lockhart[49] examined performance on six different manual dexterity tasks and—in contrast to Clark and Cohen[47] — concluded that the greatest rate of performance decrement in their sample occurred with the fastest rate of cooling, independent of the absolute final temperatures achieved. In contrast to the Bowen results, Davis, Baddeley, and Hancock[50] reported a 17% deterioration in dexterity in wet suit-clad scuba divers, who were tested following 35 to 50 minutes of exposure to 5°C water; the deterioration was not progressive but rapid, following initial cold water immersion. This disparity in findings may be caused by the nature of the tasks sampled in these two studies, with knot tying requiring a higher degree of very fine motor movements that may deteriorate faster in the cold. Enander (1987)[51] reported a 13% decrement in manual dexterity in a sample of 24 subjects clad in lightly quilted jackets and pants exposed to 5°C air for 55 to 90 minutes.

Strength

The available evidence suggests that cold exposure-particularly cold water exposure-causes substantial decrements in hand strength and impairs the ability to sustain a submaximal upper extremity muscle contraction (Table 12-3). Craik and Macpherson[52] reported in 1943 that immersing the hand in 7°C water for 15 minutes reduced mean grip strength 21%, and reduced the strength of finger-thumb opposition 44%. In 1947, Horvath and Freedman[53] reported an average decrease in grip strength of 28% in heavily clothed subjects who were

TABLE 12-3

COLD STRESS AND STRENGTH: SUMMARY OF FOUR STUDIES

Study	Temperature (°C)	Exposure Time	Results
Craik and Macpherson (1943)[1]	7°C water (hand)	15 min	21% decrease in mean grip strength; 44% decrease in finger-thumb opposition
Horvath and Freedman (1947)[2]	–25°C air (whole body)	180 min	28% decrease in mean grip strength
Clarke et al. (1958)[3]	27°C water (forearm)	Varied by skin temp, 30–120 min total exposure	60% decrease in grip strength at 2°C forearm skin temp
Bowen (1968)[4]	8°C water	24 min	14% decrease in hand strength

Temp: temperature
Data sources: (1) Craik KJW, Macpherson SJ. *Effects of Cold Upon Hand Movements and Reaction Times*. London, England: Medical Research Council Military Personnel Research Committee; 1943. Report BPC. 43/196. (2) Horvath SM, Freedman A. The influence of cold on the efficiency of man. *J Aviation Med*. 1947;18:158–164. (3) Clarke RSJ, Hellon RF, Lind AR. The duration of sustained contractions of the human forearm at different muscle temperatures. *J Physiol*. 1958;143:454–473. (4) Bowen HM. Diver performance and the effects of cold. *Hum Factors*. 1968;10(5):445–464.

exposed to –25°C air for 3 hours without exercising. In 1958, Clarke, Hellon, and Lind[46] isolated forearm muscle temperature as a critical determinant of hand strength decrements by cooling forearm muscle to 27°C via a water bath and observing a progressive deterioration in hand grip, which approached a 60% decrement at 2°C. Bowen[31] demonstrated a 14% impairment in hand strength following a 24-minute exposure to 8°C water. Similar results have been reported in sustaining a submaximal contraction by Coppin, Livingstone, and Kuehn,[54] and by Clarke, Hellon, and Lind.[46] The available evidence to date suggests that local cooling of the muscles, particularly forearm muscles, to a muscle temperature at or below 27°C is likely to impair limb strength and decrease the length of time that a submaximal muscle contraction can be maintained.

Motor Speed

Relatively little work has been done to investigate the effects of cold stress on motor speed, in part because most motor speed measures have a confounding component of manual dexterity as well. The available evidence, however, suggests some likely decrements in motor speed with significant cold exposure (Table 12-4). In 1970, Stang and Weiner[55] reported increased time to complete underwater work tasks in their sample of 12 experienced wet suit-clad scuba divers exposed to 16°C and 10°C water for 90 minutes, although some of the reported difficulty may be attributable to fine motor task demands in their simple assembly tasks (eg, attaching a plate to a work bench frame with wing nuts, attaching two plates together with wing nuts, transferring small nuts and screws

TABLE 12-4

COLD STRESS AND MOTOR SPEED: SUMMARY OF TWO STUDIES

Study	Temperature (°C)	Exposure Time	Results
Stang and Weiner (1970)[1]	10°C to 16°C water (whole body)	90 min total	Increased time to complete underwater assembly tasks, although some tasks had a dexterity or strength component
Enander (1987)[2]	5°C air (whole body)	Varied, 55–90 min	Decrements in tapping speed beginning after 20 min of exposure and increasing in severity with longer durations of exposure

Data sources: (1) Stang PR, Wiener EL. Diver performance in cold water. *Hum Factors*. 1970;12(4):391–399. (2) Enander A. Effects of moderate cold on performance of psychomotor and cognitive tasks. *Ergonomics*. 1987;30(10):1431–1445.

TABLE 12-5

COLD STRESS AND VIGILANCE: SUMMARY OF SEVEN STUDIES

Study	Temperature (°C)	Exposure Time	Results
Mackworth (1950)[1]	21°C to 36°C	Varied by skin temp	Optimal vigilance efficiency and minimal signal omissions at 26°C
Kissen et al. (1964)[2]	4°C	60 min	Progressive deterioration in vigilance to visual matching display
Poulton et al. (1965)[3]	–2.2°C to 1.7°C	30 min	Decreased vigilance of shipboard lookouts at decreased oral temperatures, but some uncontrolled variance due to rain and wind
Teichner (l966)[4]	12.8°C; 26.7°C	40 min; 40 min	No difference in visual detection between two temperatures sampled
Baddeley et al. (1975)[5]	4.4°C water (whole body)	60 min	No decrements in an underwater visual vigilance task
Vaughan (1977)[6]	4.5°C water; 15.5°C water	180 min	Increase in average visual detection times at both temperatures for immersed scuba divers.
Angus et al. (1979)[7]	0° to 5°C	16 d	Decrements in vigilance task following outdoor sleeping, further decrements due to REM sleep deprivation

REM: rapid eye movement

Data sources: (1) Mackworth NH. *Researches on the Measurement of Human Performance*. London, England: Medical Research Council; 1950. Report Series 268. (2) Kissen AT, Reifler CB, Thaler VH. Modification of thermoregulatory responses to cold by hypnosis. *J Appl Physiol*. 1964;19:1043–1050. (3) Poulton EC, Hitchings NB, Brooke RB. Effect of cold and rain upon the vigilance of lookouts. *Ergonomics*. 1965;8:163–168. (4) Teichner WH. Individual thermal and behavioral factors in cold-induced vasodilatation. *Psychophysiology*. 1966;2:295–304. (5) Baddeley AD, Cuccaro WJ, Egstrom GH, Weltman G, Willis MA. Cognitive efficiency of divers working in cold water. *Hum Factors*. 1975;17(5):446-454. (6) Vaughan WS Jr. Distraction effect of cold water on performance of higher-order tasks. *Undersea Biomed Res*. l977;4(2):103–116. (7) Angus RG, Pearce DG, Buguet GC, Olsen L. Vigilance performance of men sleeping under arctic conditions. *Aviat Space Environ Med*. 1979;50(7):692–696.

from one side of a plate to the other) or decrements in strength (eg, loosening torqued nuts and bolts with wrenches, removing bolts with a speed wrench). Using a purer measure of motor speed, Enander[51] reported in 1987 substantial decrements in tapping speed using a simple, handheld counter in a sample of 24 subjects exposed to 5°C air, which occurred following approximately 20 minutes of cold exposure and increased in severity with longer durations of cold exposure.

Vigilance

Since 1950, several investigators have studied the effects of cold exposure on vigilance tasks, but the body of literature in this area remains markedly smaller than companion studies of heat stress effects on vigilance, attention, and concentration (Table 12-5). Mackworth[56] reported optimum vigilance efficiency and minimal signal omissions for artificially acclimatized subjects at 26°C air temperatures versus either 31°C, 36°C, or 21°C. Pepler,[57] using naturally acclimatized subjects in Singapore, was able to confirm Mackworth's findings in part, and the results of these two studies led to speculation that there is an optimal level for vigilance in the range of 27°C to 32°C,[58,59] and that vigilance exhibits an inverted U-shaped distribution with lowered vigilance at both higher and lower temperatures.

Kissen, Reifler, and Thaler[60] reported a progressive deterioration in visual vigilance in lightly clad subjects exposed to 4°C air for 1 hour, who were required to distinguish randomly displayed matched pairs of a visual pattern from unmatched pairs. They observed a decrease in the number of correct identifications as cooling progressed, coincident with decreases in body core temperature, although commission errors did not vary with increased exposure time. Poulton, Hitchings, and

Brooke[61] examined vigilance of shipboard lookouts at –2.2°C and 1.7°C cold exposures and reported decrements in lookout vigilance coincident with decreased oral temperatures, although there were methodological problems in these studies owing to the uncontrolled variance attributable to wind and rain during some watch periods. Teichner[62] investigated prolonged visual detection at 12.8°C and 26.7°C air temperatures, which were accompanied by minimal decreases in body core temperature. There were no observed consistent differences between these two temperature conditions in mean percentage detection or average response speed.

In an early review paper, Grether[63] hypothesized that optimal vigilance is observed at 26.7°C on the Effective Temperature (E.T.) scale, an index of perceived warmth that combines dry and wet bulb temperatures with air velocity. In 1975, however, Baddeley and colleagues[64] reported no decrements in visual vigilance (detecting the onset of a faint peripheral light during the performance of a two-man pipe assembly task) in a sample of 14 acclimatized divers following a 60-minute exposure to 4.4°C water, despite a mean drop in rectal temperature of 0.72°C. In contrast, Vaughan[65] reported in 1977 an increase in the average time to detect peripherally displayed targets in a sample of US Navy–qualified, wet suit–clad scuba divers exposed for 180 minutes to 4.5°C and 15.5°C water. In 15.5°C water, detection speed slowed to 3.6 seconds in the first hour, to 4.2 seconds in the second hour, and to 7.2 seconds in the third hour. However, at 4.5°C water exposure, detection speed was initially much slower at 7.8 seconds, improved in the second hour to 4.8 seconds, and deteriorated again in the third hour to 8.4 seconds.

In 1979, Angus and colleagues[66] reported substantial decrements in performance on the Wilkinson visual vigilance task over the course of 16 days of exposure to 0°C to 5°C arctic temperatures, with a marked decrement in performance following the initial cold exposure. Although there was an observed gradual improvement in vigilance on subsequent exposure days, there was deterioration in performance on days following especially cold nights when rapid eye movement (REM) sleep was disrupted, suggesting partial adaptation of vigilance ability during prolonged cold exposure, modified somewhat by the effects of REM deprivation.

In an attempt to account for the disparity in findings from different laboratories in this area of investigation, Hancock[67] argued in 1984 that substantial vigilance decrements occur only during dynamic shifts in body core temperature and are not likely to occur at steady state temperatures, suggesting that fully acclimatized subjects would be unlikely to exhibit vigilance decrements even at very cold ambient temperatures.

Reaction Times

Investigations completed thus far suggest minimal decrements in simple reaction time (except in the most extreme conditions) but marked changes in more complex reaction-time tasks (Table 12-6).

Simple Reaction Time

In 1942, Williams and Kitching[68] tested both simple and choice visual reaction time (motor response to a visual stimulus) in three subjects following 60-minute exposures to environmental chamber temperatures of –18°C and –45.5°C The authors concluded that although body temperatures dropped, there was no direct relationship between reaction time and body temperatures, and that the observed variations in reaction time that occurred at temperatures of –45.5°C were explainable as due to discomfort-induced distraction. In 1947, Horvath and Freedman[53] reported no deleterious effects of cold exposure to visual choice reaction time in a sample of 22 subjects housed in temperatures of –29°C for 8 to 14 days and tested three times per day. Forlano, Barmack, and Coakley[69] in 1948 and Teichner[70] in 1954 reported that simple reaction time was unaffected by cold air exposures as severe as –45°C. In a follow-up study of 640 men exposed to cold air temperatures as low as –37°C, Teichner[71] reported in 1958 that linear decrements in reaction speed were observed in subjects beginning at an ambient temperature of –26°C with a wind speed of 10 mph or greater. Mild exercise caused a small recovery in reaction speed.

At wind speeds of 5 mph or less, no effect of temperature on reaction speed was observed. These results were apparently not related to measured skin temperatures and were instead believed to be due to increased distractibility and discomfort in windchill conditions, leading to the coining of the term "psychological cold tolerance" and the speculation that these effect of cold and windchill on simple reaction time might not be seen in highly acclimatized individuals. Since that time, Pease, Ludwig, and Green[72] in 1980 and Goodman, Hancock, Runnings, and Brown[73] in 1984 have demonstrated decrements in mean reaction time to moderate to severe cold stress, although not without extreme cooling of the responding arm. In 1987, Enander[51] reported no decrement in simple visual

TABLE 12-6

COLD STRESS AND REACTION TIME: SUMMARY OF EIGHT STUDIES

Study	Temperature (°C)	Exposure Time	Results
Williams and Kitching (1942)[1]	–18°C; –45.5°C air	60 min	Decrements in simple and choice RT at –45.5°C
Horvath and Freedman (l947)[2]	–29°C air	8–14 days	No difference in visual choice RT
Teichner (l954)[3]	–45.5°C air	45 min	Simple RT not affected in air temps as low as –45°C
Teichner (1958)[4]	–37°C air + wind	Varied, 45–63 min	Linear decrements in RT at –26°C when accompanied by 10 mph wind
Stang and Weiner (1970)[5]	10°C to 21°C (water)	90 min	0.5% slower visual choice RT in 16°C water; 23% slower visual choice RT in 10°C water
Ellis (1982)[6]	–12°C water	90 min	Increase in errors of 200%–300% in serial choice RT; no effect on simple RT
Ellis et al. (1985)[7]	–5°C air	Varied; fast cooling 60 min; slow cooling 180 min	Increase in errors on serial choice RT when rapidly cooled, but not if slowly cooled
Enander (1987)[8]	5°C air	Varied, 55–90 min	Replicated Ellis (1982)[6]

RT: reaction time

Data sources: (1) Williams CC, Kitching JA. The effects of cold on human performance, I: Reaction time. *Misc Canad Aviat Rep.* Toronto, Ontario, Canada: Banting and Best Department of Medical Research; 1942. (2) Horvath SM, Freedman A. The influence of cold on the efficiency of man. *J Aviation Med*. 1947;18:158–164. (3) Teichner WH. Recent studies of simple reaction time. *Psychol Bull.* 1954;51:128–149. (4) Teichner WH. Reaction time in the cold. *J Appl Psychol.* 1958;42(1):54–59. (5) Stang PR, Wiener EL. Diver performance in cold water. *Hum Factors*. 1970;12(4):391–399. (6) Ellis HD. The effects of cold on the performance of serial choice reaction time and various discrete tasks. *Hum Factors*. 1982;24(5):589–598. (7) Ellis HD, Wilcock SE, Zaman SA. Cold and performance: The effects of information load, analgesics, and the rate of cooling. *Aviat Space Environ Med.* l985;56(1):233–237. (8) Enander A. Effects of moderate cold on performance of psychomotor and cognitive tasks. *Ergonomics.* 1987;30(10):1431–1445.

reaction time in a sample of 12 men exposed to air temperature of 5°C for 90 minutes.

Choice Reaction Time

In 1970, Stang and Wiener[55] reported decrements in visual choice reaction time in their sample of 12 experienced scuba divers exposed to water at temperatures of 16°C and 10°C. When compared with their task performance in 21°C water, subjects responded 10.5% slower when exposed to 16°C water, and 23% slower than that when exposed to 10°C water.

In 1982, Ellis[74] reported significant decrements in serial choice reaction time in subjects exposed to air temperatures of –12°C for 90 minutes. The serial choice reaction time task used in these experiments consisted of the serial visual presentation of a series of digits between 1 and 8 that were to be classified by the subject as either odd or even by depressing one of two control buttons. Immediately after either button was pressed, a next digit was randomly selected and presented, and the cycle continued for 500 trials. Despite an increase in serial choice reaction time errors of 200% to 300% for these cold-stressed subjects, there was no accompanying decrement in simple reaction time in the cold (immediate pressing of a button when the number "0" appeared on the screen). These errors were directly proportional to observed reductions in mean skin temperatures and reported to be largely independent of any fall in rectal (core) temperatures.

Ellis, Wilcock, and Zaman[75] demonstrated in 1985 reliable decrements on the 8-choice visual choice reaction-time task in eight lightly clad male subjects rapidly cooled by exposure to –5°C air for 60 minutes, but decrements on the same 8-choice visual choice reaction-time task were not observed with a subsample of six male subjects cooled slowly by a 180-minute exposure to 8°C air. This has led to speculation that rapid cooling via mild cold air stress may affect complex reaction-time primarily by increasing discomfort and distraction rather than by a direct effect due to changes in body surface or core temperature. In 1987, Enander,[51] using a very similar visual choice reaction time task, was able to replicate Ellis's[74] 1982 results in a sample of 12 lightly clad women exposed to 5°C air for 55 to 90 minutes. There were increases in the number of errors, speed of incorrect responses, and num-

ber of false alarms, leading to speculation that for more complex tasks reaction time may be compromised by a decreased ability to inhibit incorrect responses.

Target Tracking

The programmatic study of the effect of cold stress on manual pursuit tracking began more than 50 years ago (Table 12-7). In 1947, Blair and Gottschalk[76] found that manipulation of a metal tracking control by thumb and forefinger was impaired during cold air exposure. The initial exposure of men fully dressed in arctic clothing (including arctic mittens) to air temperature of –25°C produced a 19% reduction in performance from that found at 23°C, and a further reduction in temperature to –41°C produced an additional 21% decrement, although some of the observed decrement may have been attributable to numbness and surface cooling of the fingers on the bare metal apparatus. In 1954, Teichner and Wehrkamp[77] reported results of multiple trials of pursuit rotor tracking at air temperatures of 13°C, 21°C, 29°C, and 38°C. Performance of their subjects deteriorated both above and below 21°C, but that performance appeared to fall off more rapidly at 13°C than it did at 29°C or 38°C. A follow-up study of the effects of longer-term cold exposure on pursuit rotor performance was reported by Teichner and Kobrick in 1955.[78] Their six subjects lived in a constant temperature environmental chamber for 41 days, and their pursuit rotor tracking ability was tested daily (15 trials per day). For the first 16 days the chamber temperature was held at 24°C, the next 12 at 13°C, and at 24°C for the remaining 13 days. Visual-motor tracking performance was markedly and immediately impaired in the cold and recovered gradually, but only to the approximate level of performance obtained at the beginning of these experiments, essentially eliminating the beneficial effects of practice on this task that had been observed over the course of the first 16 days.

In 1957, Russell[79] investigated a manual tracking task in six ambient temperatures ranging from –10°C to 40°C. The test task involved using a manipulator control to actuate a stylus, which was used to track a laterally moving ink line on a moving chart paper. The manipulator was configured to respond to either slight pressure or to actual movement. Russell found that duration of exposure had no significant effect on performance, but that performance on both types of tracking tasks declined steeply when

TABLE 12-7

COLD STRESS AND TARGET TRACKING: SUMMARY OF SIX STUDIES

Study	Temperature (°C)	Exposure Time	Results
Blair and Gottschalk (1947)[1]	23°C; –25°C; –41°C	Varied with skin temp	19% reduction in manipulation of a metal tracking control at –25°C; additional 21% reduction at –41°C
Teichner and Wehrkamp (1954)[2]	13°C to 38°C	30 min	Pursuit rotor tracking deteriorated above and below 21°C, but more rapid falloff at 13°C
Teichner and Kobrick (1955)[3]	13°C to 24°C	41 days	Visual motor tracking markedly and immediately impaired at 13°F
Russell (l957)[4]	–10°C to 40°C	73 min	Decrements in tracking at 10°C; pressure tracking deteriorated faster than movement tracking
Payne (l959)[5]	4°C; 13°C; 21°C	200 min; 200 min; 200 min	Decrement in complex tracking task at 4°C
Enander (1987)[6]	4°C to 20°C	60 min	Significant increase in errors, speed of incorrect response, and number of false alarms on two computerized tracking tests at 4°C

Temp: temperature

Data sources: (1) Blair EA, Gottschalk CW. *Efficiency of Signal Corps Operators in Extreme Cold.* Fort Knox, Ky**:** US Army Medical Research Laboratory; 1947. AMRL Report 2. (2) Teichner WH, Wehrkamp RF. Visual–motor performance as a function of short-duration ambient temperature. *J Exp Psychol.* 1954;47:447–450. (3) Teichner WH, Kobrick JL. Effects of prolonged exposure to low temperature on visual–motor performance. *J Exp Psychol.* 1955;49(2):122–126. (4) Russell RW. Effects of Variations in Ambient Temperature on Certain Measures of Tracking Skill and Sensory Sensitivity. Fort Knox, Ky**:** US Army Medical Research Laboratory; 1957. AMRL Report 300. (5) Payne RB. Tracking proficiency as a function of thermal balance. *J Appl Physiol*. 1959;14:387–389. (6) Enander A. Effects of moderate cold on performance of psychomotor and cognitive tasks. *Ergonomics.* 1987;30(10):1431–1445.

subjects were exposed to ambient temperatures below 10°C, and that pressure tracking deteriorated sooner than movement tracking, suggesting that subtleties of motor control might deteriorate faster in the cold than more pronounced motor movements.

In 1959, Payne[80] examined tracking performance of lightly dressed subjects at 21°C, 13°C, and 4°C on a complex task requiring attention to several controls at once for accurate performance. Payne reported that performance on this task was directly related to ambient temperature, with the poorest performance occurring at 4°C, a condition also reported by subjects to be quite stressful. Recent work by Enander[51] (1987) has demonstrated significant increases in number of errors, speed of incorrect response, and number of false alarms on two computerized tracking tests completed by female subjects during mild cold exposure. These effects, however, were only obtained when tests were constructed that required continuous rapid accurate responding, with minimal available opportunity to inhibit error responses.

Memory and Recall

It has been known for some time that extreme cold stress can induce confusion and impaired consciousness, and a series of studies have examined the effects of cold water immersion and pronounced cold stress on memory and memory registration (Table 12-8).

In 1959, Keatinge[81] reported that two of his subjects experienced complete amnesia for the last few minutes of 20-minute immersions in 5°C water, during which time they experienced core temperature decreases to 34.2°C and 35.1°C. In 1968, Bowen[31] reported a 22% decrement in performance on the Clock Test, a measure of short-term memory, in subjects exposed to 8°C water. In 1970, Stang and Weiner[55] reported decrements on an arithmetic test

TABLE 12-8

COLD STRESS AND MEMORY: SUMMARY OF EIGHT STUDIES

Study	Temperature (°C)	Exposure Time	Results
Keatinge (1959)[1]	5°C water (whole body)	20 min	Reported complete amnesia in two subjects for the last few minutes of immersion, at body core temperatures of 34.2°C and 35.1°C
Bowen (1968)[2]	8°C water	Varied, typically less than 25 min	22% decrement in performance on the Clock Test, a measure of short-term memory
Stang and Weiner (1970)[3]	10°C water	90 min	Decrements reported on an arithmetic test, attributed to lapses in concentration
Egstrom et al. (1972)[4]	4.4°C water	50 min	Impairment in divers' ability to recall material learned underwater
Baddeley et al. (1975)[5]	4.4°C water; 25.6°C water	50 min	Impairment of the recall of short paragraphs
Davis et al. (1975)[6]	5°C water; 20°C water	35–50 min	Memory recognition impaired after exposure to 5°C water
Coleshaw et al. (1983)[7]	15°C water	Varied, 45–60 min	70% decrement in memory registration at core body temp of 34°C–35°C
Thomas et al. (1989)[8]	5°C air	60 min	Decrement in delayed matching to sample visual memory

Temp: temperature

Data sources: (1) Keatinge WR. *The Effect of Work, Clothing and Adaptation on the Maintenance of the Body Temperature in Water and on Reflex Responses to Immersion*. Cambridge, England: University of Cambridge; 1959. Thesis. (2) Bowen HM. Diver performance and the effects of cold. *Hum Factors*. 1968;10(5):445–464. (3) Stang PR, Weiner EL. Diver performance in cold water. *Hum Factors*. 1970;12(4):391–399. (4) Egstrom GH, Weltman AD, Baddeley WJ, Cuccaro WJ, Willis MA. *Underwater Work Performance and Work Tolerance.* Los Angeles, Calif: University of California, School of Engineering and Applied Sciences; 1972. UCLA-ENG-7243. Biotechnologic Laboratory Technical Report 51. (5) Baddeley AD, Cuccaro WJ, Egstrom GH, Weltman G, Willis MA. Cognitive efficiency of divers working in cold water. *Hum Factors*. 1975;17(5):446–454. (6) Davis FM, Baddeley AD, Hancock TR. Diver performance: The effect of cold. *Undersea Biomed Res.* 1975;2(3):195–213. (7) Coleshaw SRK, Van Someren RNM, Wolff AH, Davis HM, Keatinge WR. Impaired memory registration and speed of reasoning caused by low body temperature. *J Appl Physiol.* 1983;Jul 55(1 Pt 1):27–31. (8) Thomas JR, Ahlers ST, House JF, Schrot J. Repeated exposure to moderate cold impairs matching-to-sample performance. *Aviat Space Environ Med.* 1989;60(11):1063–1067.

during exposure to 10°C water for 90 minutes, with errors attributable to momentary lapses in concentration.[65]

In 1972, Egstrom and colleagues[82] reported impairment in divers' abilities to recall material learned underwater when tested after 50 minutes of exposure to 4.4°C water, although no impairments were noted in vigilance, reaction time, or reasoning tasks. The authors speculated that this result might represent reduced attentional capacity. In 1975, Baddeley and colleagues[64] examined the performance of subjects exposed for 50 minutes to water at 4.4°C and 25.6°C. Although no impairment was found on reasoning tasks or vigilance tasks, there was impairment in the recall of short paragraphs. No decrement in recognition memory was observed. Also in 1975, Davis, Baddeley, and Hancock[50] compared performances on tasks involving simple arithmetic, logical reasoning, digit span, word recall, and memory recognition during 35- to 50-minute exposures to water at 5°C and 20°C. Only the memory tests were significantly affected by differences in exposure temperature. Although decrements in word recognition performance were highly correlated with body cooling, word recall scores were not, implying an effect not solely attributable to the result of direct central (core body) cooling.

In 1983, Coleshaw and colleagues[4] reported significant impairment of auditory memory registration following immersion in 15°C water. At core body temperatures of 34°C to 35°C, loss of about 70% of the data that could normally be retained was observed. There was no observed impairment in previously learned data, however, implying an effect of deep body cooling on new learning and concentration, or attention, or both.

In 1989, Thomas and colleagues[83] reported a clear decrement in delayed matching to sample visual memory performance in a sample of six subjects exposed to 5°C air for 60 minutes, and this impaired level of performance persisted at the same level in two subsequent exposures to 5°C air, each occurring 1 week apart. Each subject in this experiment was lightly dressed and seated in front of a computer terminal, which visually displayed a target matrix of 16 squares colored either red or yellow. Subjects responded to seeing the target matrix by pressing a button on the console; the target matrix was then immediately removed from the screen and after a 3-second delay, two comparison matrices were displayed on the screen. One matrix was identical in pattern to the sample matrix, the other differed by one cell only, and the subject was required to choose the identical matrix. During the exposure to 5°C air, the number of errors increased relative to pretest performance in 22°C ambient air, response times following the presentation of the target stimulus were shorter, and response times prior to selecting the comparison matrix were longer. These data were unrelated to central body cooling effects and generally were interpreted as a decrement in performance related to increased arousal rather than distraction.

Complex Cognitive Functioning

The available evidence in this area suggests that decrements in cognitive functioning due to cold stress are for the most part directly related to task complexity. In 1966, Baddeley[84] studied the effect of cold water immersion on the ability to estimate time by having 20 scuba divers count up to 60 at what they considered to be a 1-second rate while immersed in 4°C sea water. Subjects consistently counted at a slower rate while immersed, which was correlated with decreases in oral temperatures (median rank-correlation = 0.50). Rate of counting was not correlated with pulse rate, predive anxiety, or effects of the order of the tests. It should be noted that the face was directly exposed to cold water in these experiments, resulting in oral temperature measurements that are probably gross underestimates of deep body temperatures.

In 1968, Bowen[31] reported a 12% decrease in accuracy on a symbol processing task completed by subjects immersed in 8°C water. This task consisted of a subject's reading an entry that listed, in sequence, a code number and four colors. The subject was then required to visually scan a 10 X 10 table (10 colors by 10 numbers) to find a numerical value for each color. Each subject then selected a problem from a problem chart array, which provided four sequential numbers. The subject paired each of these sequential numbers with the previously derived numerical values for each of the four colors, multiplied each pair, summed the products, and checked off the correct number with a grease pencil on an answer slate. Subjects also experienced fewer successfully completed items on a problem-solving set—exceptions test when immersed in 8°C water. This test involved having the subjects visually scan five numbers presented horizontally and deciding which four of these numbers had the same arithmetic common denominator (ie, which numbers could all be evenly divided by the same number) and excluding the one that did not by checking it with a grease pencil. The increased performance decrement on both of these tasks was not believed to be related to

decrements in motor speed or dexterity.

In 1977, Vaughan[65] reported significant decrements in navigation problem-solving tasks completed by eight US Navy-qualified scuba divers during the first hour of immersion in 4.5°C water, but noted that performance on these same tasks during the second and third hours of immersion were not significantly different-whether immersed in 4.5°C water or 15.5°C water. The navigation problem-solving task involved a display console in an underwater simulator, which presented information about the vehicle's position with reference to its intended track. The console additionally displayed real time, across-track error, and distance traveled along-track. Given the displayed data (which were varied remotely for each navigation problem), divers were asked to plot successive positions of the vehicle, draw a vector triangle, and determine set and drift of the current, vehicle speed, vehicle course over the bottom, and a new heading for the vehicle that corrected for current vector effects. Observed decrement on this navigation task during the first hour of immersion was interpreted by Vaughan as due to distraction rather than as secondary to core or peripheral cooling.

In 1993, Giesbrecht and colleagues[85] reported decrements in the performance of complex tasks requiring mental manipulation (backward digit span) and mental processing and analysis (Stroop Color-Word Test) following the immersion of swimsuit-clad subjects in 8°C water once central cooling of 2°C to 4°C had occurred, suggesting a greater effect of cold on tasks that (*a*) are complex or perceptually demanding and (*b*) require significant concentration or short-term memory. In their sample of six subjects, cold water immersion had no significant effect on less cognitively demanding tasks such as auditory attention, visual recognition, and forward digit span.

Learning

Although several investigators have demonstrated deleterious effects of cold stress on components of the learning process (attention, concentration, vigilance, and memory), there has been relatively little work examining direct effects on learning. To date, the effect of high temperatures has been more extensively examined than low temperatures. In 1968, Pepler and Warner[86] examined the learning efficiency of 72 male and female college students who studied a programmed text once a week for 6 weeks (3 h/d) while lightly dressed and housed in an environmental chamber at ambient temperatures of 17°C, 20°C, 23°C, 27°C, 30°C, or 33°C. The lowest mean error rate and lowest mean rating of perceived effort occurred in the 27°C condition. The highest mean error rate occurred at 17°C: the students changed their learning style and worked more quickly, they experienced higher levels of effort, and they made errors at a faster rate. Although far more work is obviously needed in this area, these data are in general agreement with Enander's[51] report of significant increases in number of errors, speed of incorrect response, and number of false alarms during mild cold exposure.

Thermal Sensation Versus Thermal Perception

In cold environments, an individual's behavioral responses to the cold are often a function of his or her subjective assessment of cold and thermal discomfort rather than of actual exposure time, physiological status, prior acclimation to the cold, or deep body temperature.

Some evidence suggests that perceived thermal intensity (*a*) depends solely on signals from peripheral thermal receptors and (*b*) is independent of the stimulation of central thermal receptors. This is consistent with the observations of Benzinger[87,88] in a series of experiments published in 1970 and 1978 on the role of surface temperature on cold perception. These studies suggest that central thermoreception in the homeostatic range from 36.1°C to 37.8°C appears to make no demonstrable contribution to cold perception at its threshold. In contrast, the thermal comfort experiments using perfused gloves done by Cabanac and colleagues[89,90] and published in 1971 and 1972, suggest that the behavioral response to cold stress strongly depends on internal body temperature. A third position is represented by the work of Mowrer,[91] who concluded in 1976 that thermal intensity depends solely on signals from peripheral thermal receptors, and that thermal pleasantness or comfort is the result of an interaction of signals from both central and peripheral receptors. One explanation of this disparity may be that central cold receptors contribute to the conscious sensation of cold primarily at lower central temperatures. In 1970, Benzinger[87] demonstrated the apparent absence of a central component for temperatures in the range above 36°C central temperature.

In a series of experiments by Gagge and colleagues[92] published in 1967 and Hardy and colleagues[93] in 1971, a marked drop in skin temperature and heart rate was observed following exposure to 17.5°C air.

Initial reactions to these changes were an intense sensation of cold and discomfort, which were followed by both (1) a temporary reduction of the estimates after vasoconstriction had developed and (2) a slight increase in internal body temperature. During the following 2-hour period body cooling was essentially passive, and reports of discomfort and cold sensation were relatively stable and increased only slightly. During the final hour, there was little, if any, correlation between reports of discomfort and thermal sensations of cold with mean skin temperature. Similarly, Hardy[94] found in another series of cold air experiments in 1970 that neither mean skin temperature nor internal body temperature appeared to be related in any quantitative sense to sensory responses; like most of the experiments reported in this section, cold sensation was found to be related to discomfort or pain. These sensory responses increased on entering the cold—before any appreciable change in body temperature—and decreased rapidly on leaving the cold—before any significant recovery of the 2.8°C decrement in body temperature that had been incurred during the exposure. The sensors were thus "leading" the body temperature changes and their effects were anticipatory, suggesting that cold sensation may be a type of sensory response related to the rate of change of skin temperature.

In 1989, Hoffman and Pozos[95] examined subjective estimates of cold and temperature at multiple body sites in 12 subjects immersed in 10°C water for an average of 112 minutes while wearing flotation suits. Subjects were unable to reliably assess how cold they actually were, with .51 being the highest correlation observed between perceived temperature and actual temperature.

The results of these studies suggest that although subjects exposed to mild-to-moderate cold air may be able to reliably assess thermal intensity and body temperatures, individuals who are rapidly cooled in cold water may have considerable difficulty separating feelings of pain and discomfort from feelings of cold, which could have serious consequences in cold weather survival situations and demonstrates the somewhat subjective and variable nature of cold perception.

Arousal Versus Distraction Models of Complex Behavior

Two theoretical models have been generally proposed to explain the effect of cold stress on human performance, although neither has satisfactorily accounted for the observed patterns of performance decrement. The first, the theory of general arousal, predicts effects dependent on the degree of physiological stimulation in relation to task difficulty and subject experience.[51] This model is described as having a U-shaped distribution, such that the performance of a simple task may be facilitated by an increase in arousal (eg, a mild cold stress), whereas the optimal level of arousal needed to facilitate performance is lower if the nature of the task is more complex. Using such a model, therefore, an environmental stressor such as mild cold stress would be expected to facilitate performance on simple tasks such as vigilance and degrade performance on more complex tasks such as choice reaction time or delayed matching to sample tasks. To date, however, this model has been found to have serious limitations: it does not adequately explain some performance decrements in mild cold stress conditions despite attempts to build more complex arousal models; and it also does not well address the majority of performance decrements observed during moderate-to-severe cold stress.[96]

The second theory, that cold stress has a distraction effect, was first proposed in 1958 by Teichner[71] as a way to explain the apparent lack of consistent relation between measures of physiological cooling and human performance. According to the distraction model, cold stress is believed to cause momentary switches of attention from the primary task, resulting in performance decrement. Although this model would to some extent account for decrements in attention, concentration, and reaction time in some cases of mild cold stress, there is no room in this model to address observed facilitation of performance in modest cold stress, nor have investigators observed the pattern of missed signals that this model would predict.[51,75]

Training and Acclimatization

Although there have been several reports of both naturally occurring and experimentally induced acclimatization altering the cooling rates of fingers and hands, relatively few reports have included an examination of resultant changes in hand skills, dexterity, and strength. In 1952, Yoshimura and Iida[97] reported a significant attenuation of finger vasoconstriction and earlier cold-induced vasodilation in a sample of cold-acclimatized Chinese and Mongolian subjects versus non–cold-acclimatized Japanese subjects. In 1955, Meehan[98] described similar differences between Athabascan arctic Indians and caucasians. Krog and colleagues[99]

described similar differences in 1960 between Norwegian and Laplander fishermen and controls; Nelms and Soper[100] in 1962 between British fish filleters and laboratory technicians; and Hoffman and Wittmers[43] in 1990 between male arctic explorers and a comparison group matched for gender, age, and finger size. In addition, Enander, Skold-strom, and Holmer[101] noted in 1980 that a cold-acclimatized group of meat cutters experienced significantly less cold and discomfort (increased psychological cold tolerance) when exposed to 10°C air, compared with a sample of office workers, despite no significant differences in measured hand temperature.

In 1960, Krog and colleagues[99] additionally investigated the effect of cold habituation (acclimatization) on the motor responses of Norwegian and Laplander fishermen. They reported that their cold-acclimatized subjects had a faster finger-tapping rate and greater grip strength than a matched, nonacclimatized control sample. This result was reported to be correlated with an altered cold-induced vasodilation (CIVD) response in the acclimatized subjects' fingers, such that their fingers did not get as cold as the control samples' fingers before their fingers began to rewarm spontaneously. (For further information on CIVD, please see Chapter 13, Prevention of Cold Injuries, and Chapter 14, Clinical Aspects of Freezing Cold Injury.)

In 1962, Clark and Jones[33] trained subjects for 3 weeks on a standard manual task in either a hands-cold training condition or a hands-warm training condition, and later assessed the subjects' abilities to perform this task in the cold. One day of cold-hands training reduced significantly the size of the performance decrement usually associated with cold exposure, but continued cold experience did not. Skill level on the task was not reported to interact with the cold-induced performance decrement.

Much further work should be done in this area, although these preliminary studies appear to indicate at least some beneficial effects of cold-climate training on manual skills. Yet to be adequately addressed is the issue of cold-weather training on those tasks that appear to be vulnerable to distractions, effects that are attributable to the cold. If there is a substantial distraction effect due to cold stress, this distraction should be attenuated by previous cold exposures, acclimatization, or both. Also as yet inadequately addressed is whether phased training is beneficial to combined training in attempting to reduce performance decrements in the cold. Phased training refers to training on a task in the absence of a stressor (in this case, the cold) and retraining that same task in the presence of the stressor, versus combined training in which the task is initially learned in that stress environment in which it is likely to be performed. The available literature in the field of psychological stress effects suggests that there may be no measurable differences between the two types of training in terms of stress coping,[102] but this possibility should be empirically tested in cold-stress training environments. The only evidence in this regard was the observation by Clark and Jones[33] in 1962 that whether a subject initially practiced a manual task with cold hands or warm hands, a switch to the alternate condition led to an initial performance decrement, suggesting a component of state-dependent learning. This effect was not observed, however, in subsequent studies by Enander in 1986.[103]

PRACTICAL APPLICATIONS AND MILITARY CONSIDERATIONS

The performance decrements following cold exposure that have been demonstrated in controlled laboratory environments have important implications for personnel in the field as well. In some circumstances, cold-induced decreases in performance can lead to significant impairments in mission-critical tasks, putting both cold-affected individual and the group at risk. The following are practical suggestions for military operations in the cold.

- Loss of hand dexterity and loss of tactile sensitivity in extreme cold can lead to measurable loss of feedback as to what the hands are doing if tasks are done without adequate visual inspection (eg, tasks performed in the dark or in very low light conditions requiring the manipulation of switches, buttons, or component parts or repair tasks done primarily by feel). Individuals may need the provision of aids that may not have been needed in temperate environments, such as extra lighting via flashlights or penlights, additional inspection mirrors, the use of glow tape or luminescent dots, adaptation of unimanual tasks to become bimanual tasks, or the provision of special tools. Tasks traditionally done by touch, such as some calibration tasks, may need to be done by visual inspection or by the use of specialized tools.
- Attempting to keep the hands as warm as possible to minimize changes in hand dex-

terity and tactile sensitivity raises the problem of bulky gloves, which decrease dexterity but may retain hand heat, versus thinner gloves or bare hand performance, which may initially preserve dexterity until the fingers become numb, at which point dexterity decreases. This dual need for warmth and dexterity may require task-specific gloving or electrically heated gloving to decrease bulk, suggesting the need for specialized clothing issue for individuals with mission-critical cold weather tasks. Ergonomic redesign of controls may be required to allow gloves-on task performance, and metal surfaces of controls and tools that contact the hand may need to be covered with plastic or rubber to decrease heat conductivity and decrease heat loss from the hands.

- Ironically, problems also result from gloves that are too warm (or clothing that is too warm), especially following exercise or work that generates sweat, which creates faster recooling rates due to evaporation and increases the risk of freezing or nonfreezing cold injury. This may require a rethinking of customary work-rest cycles to avoid excess heat generation, combined with layered clothing or the use of "clothing systems" that will allow the rapid adding or subtracting of clothing layers in response to changes in ambient temperature and activity level. The military should pay close attention to gloving design, especially the use of absorbent inner-gloving liners to wick away moisture and breathable outer microfilament fabric to allow moisture to leave the interior of the glove but also accommodate a relative vapor barrier to impede moisture from the outside. There may also be a supply need for additional issue of multiple pairs of gloves for troops in the field to avoid having individuals with wet gloves. These same considerations would apply to socks, boots, and to a lesser extent, outerwear. Ideally, some provision would be made for the rapid drying of garments in the field as well, although some of the quicker-drying microfilament fabrics may make this concern moot.
- Weapons tracking systems that are hydraulically controlled will have increased damping in extreme cold and operate at different rates than those that operators are accustomed to, causing potential performance decrements. At extreme temperatures, oils and lubricants may become less efficient and more viscous, causing binding of component parts and consequently a lag between initiating a task and the response of the component equipment. The same is true for cabled control and flight surfaces, which may cause systems to respond at different (and sometimes varying) rates. Combined with potential manual dexterity decrements and decrements in vigilance, increased operator errors may ensue. These types of operator errors may be minimized with training in similarly cold environments or, when practical, local heating of component parts.
- Component mission tasks may need to be divided into either subtasks that can be performed by individuals working shorter periods of time and pausing to rewarm their hands, entire body, or both, or by assigning tasks to teams and consistently rotating warm troops from warm shelters to cold work sites. This requires a recognition that some tasks that may be easily accomplished in temperate environments by a single individual (such as equipment repair tasks or the operation of some weapons systems) may take much longer in extreme cold or be accomplished with greater errors and potential injury. This may require the sort of cross-training of individuals on multiple tasks familiar to those in special operations so that task-switching can occur in extreme cold environments, rather than training individuals on a small number of very specialized tasks.
- Decreases in hand strength in extreme cold environments, coupled with sometimes balky mechanical controls, may require the ergonomic redesign of specialized tools to increase leverage, specialized pretraining to increase hand strength, or the redesign of controls or tasks (eg, local heating of controls via circulating hot air, heated shelters for operators, and heat coils or heated shelters for equipment, if practical).
- Impairment in vigilance at extreme cold temperatures suggests the need for much more frequent changes of sentries and sailors on watch if not sheltered from the elements than would be expected in temperate climates. This would also hold true for

divers, and suggests the need for shortened dive times in cold water missions when practical. In like manner, the available evidence of possible decrements in memory recall following cold water immersion suggests the need for either briefer exposure times or the use of written notes taken at the time or direct, on-line communication.

SUMMARY

In an attempt to provide guidance in framing reasonable performance expectations for military personnel, this chapter reviews the scientific evidence for decrements in human performance that are expectable in mild, moderate, and extreme cold environments. In addition, areas are highlighted where additional training or pretraining may prove beneficial, including the possible role of acclimatization as a method of attenuating performance decrements. Finally, some attention is drawn to possible central nervous system correlates or determinants of decreased performance in the cold.

The effects of deep body cooling on overt behavior are similar to the effects of general anesthesia. Levels of consciousness and alertness gradually decrease as body temperature drops toward 30°C. Responses become slow and reflexes sluggish; speech becomes slurred and increasingly difficult. Mobility is impaired, and individuals often become drowsy or apathetic. Some evidence suggests a degree of impairment in memory registration beginning at a core temperature of 36.7°C. Concentration becomes increasingly impaired, although at early states of hypothermia (core temperatures of 34°C–35°C) the impairment appears to be in speed of mental operations rather than accuracy. Lowered core temperature also slows the performance of complex calculation tasks, and it probably slows performance of reasoning tasks, as well. As the body cools further, individuals may become increasingly confused and even incoherent as hypothermia progresses below 34°C or 35°C. Voluntary movements gradually become slower, and a simple movement such as touching the nose may take an individual 15 to 30 seconds as his core temperature decreases to near 30°C. Muscle rigidity is often striking at this point, making it difficult to extend the limbs, and is sometimes accompanied by neck stiffness. Gait may become ataxic, and deep tendon reflexes are decreased. Consciousness is usually lost at core temperatures between 32°C and 30°C, although there are some exceptions to this.

One factor that might account for slower performance by extremely cold or hypothermic individuals is a direct slowing of synaptic transmission. Local brain cooling first increases and then decreases neural excitability. The effects of cooling appear to be reversible, and the temperature changes needed for reported changes in brain function are one Centigrade degree or less in various areas of the nervous system. Venous blood circulation offers a buffering of rapid brain cooling during hypothermia, and brain–blood temperature gradients appear to be major determinants of fluctuations in brain temperature. The effects of core temperature cooling on cerebral $CMRO^2$, CBF, and glucose substrates may also at least partially account for the observations of slowed mental processing in hypothermic individuals. In general, the effects of hypothermia on nerve conduction are summarized as follows:

1. progressive slowing of axonal conduction,
2. abolition of synaptic transmission prior to conduction failure (postsynaptic activity is abolished at approximately 20°C, whereas axonal conduction does not fail until temperatures of < 10°C are achieved), and
3. an increased excitability at mild degrees of hypothermia that precedes the subsequent synaptic depression at lower levels of hypothermia.

A number of studies in both cold air and cold water have demonstrated substantial decrements in manual performance following cold stress, the magnitude of which appears to be a function of both surface cooling and, with prolonged exposures, deep body cooling. When the whole body is cooled, there is likely a local effect at the periphery involving direct interference of sensory-motor functioning, and a general effect influencing higher centers of the central nervous system that serve to control, direct, and coordinate action.

At extreme cold temperatures, loss of sensitivity and manipulative ability is amplified by heavy protective garments worn to buffer heat loss. This decrease in manual performance is further compromised by loss of flexibility in the muscles of the forearm and finger, increase in muscle viscosity of the extensors and flexors of the fingers, and difficulty in bending joints because of increased viscosity in the synovial fluid. A number of studies have identified skin or extremity temperature as the relevant variable that predicts impairment in manual dexterity and tactile

sensitivity. Available evidence suggests that the relationship between skin temperature and numbness follows and L-shaped distribution, with relatively little impairment in sensitivity and performance at skin temperatures above 8°C but a very rapid drop-off in performance below an approximate threshold temperature of 6°C to 8°C. The pain threshold for a cold stimulus is also in this range, about 10°C in all but the most highly acclimatized subjects. Substantial errors in dexterity occur at hand-skin temperatures at and below 13°C. A greater decrement of performance occurs when subjects' hands are cooled slowly, as opposed to quickly; a sizable decrement in performance persists even after rewarming in subjects who are cooled slowly; and the rate of rewarming is directly related to the rate of cooling.

The available evidence suggests that cold exposure—particularly cold water exposure—causes substantial decrements in hand strength and impairs the ability to sustain a submaximal upper-extremity muscle contraction. The available evidence, however, suggests some likely decrements in motor speed with significant cold exposure.

Although cold exposure clearly affects vigilance, substantial vigilance decrements are likely to occur only during dynamic shifts in core body temperature and are not likely to occur at steady state temperatures, suggesting that fully acclimatized subjects would be unlikely to exhibit vigilance decrements even at very cold ambient temperatures. Investigations completed thus far suggest (1) minimal decrements in simple reaction time except in the most extreme conditions, but (2) marked changes in more complex reaction time tasks. On computerized tracking tests completed during mild cold exposure, recent work has demonstrated significant increases in the number of errors, the speed of incorrect responses, and the number of false alarms. These effects, however, are only obtained when tests were constructed that required continuous rapid, accurate responding, with minimal opportunity available to inhibit error responses.

The fact that extreme cold stress can induce confusion and impaired consciousness has been known for some time, and a series of studies has examined the effects of both cold water immersion and pronounced cold stress on memory and memory registration. Decrements have been observed in divers' abilities to recall material learned underwater, impairment in the recall of short paragraphs, and delayed matching to sample visual memory. The available evidence in the area of complex cognitive functioning suggests that decrements in cognitive functioning owing to cold stress are, for the most part, directly related to task complexity.

Finally, individuals who are rapidly cooled in cold water may have considerable difficulty separating feelings of pain and discomfort from feelings of cold, which could have serious consequences in cold weather survival situations and demonstrates the somewhat subjective and variable nature of cold perception.

Preliminary studies appear to indicate at least some beneficial effects of cold climate training on manual skills. Yet to be adequately addressed is the issue of whether cold weather training has an effect on the tasks that appear to be vulnerable to distraction that is attributable to the cold. If there is a substantial distraction effect due to cold stress, then this distraction should be attenuated by previous cold exposures or acclimatization, or both. Also as yet inadequately addressed is whether phased training is beneficial to combined training in attempting to reduce performance decrements in the cold. Some practical advice is offered relative to minimizing the effects of cold in the field.

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Chapter 13

PREVENTION OF COLD INJURIES

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INTRODUCTION

Cold injuries have been recorded as a major problem for military operations in cold environments since Xenophon's march of the Ten Thousand in 401/400 BCE,[1] which involved severe frostbite problems in mercenaries crossing Armenia in winter, and Hannibal lost almost half his army of 47,000 while crossing the Alps in 218 BCE.[2] The problem of the identification and treatment of cold injuries continues to this day to plague modern military operations. The ability of warfighters to perform and survive in a cold environment requires knowledge and understanding of both the nature of the environmental threat and methods of coping with it.

The major cold injuries are frostbite; trench foot and immersion foot, which are now grouped in the new category, nonfreezing cold injury (NFCI); and hypothermia. Frostbite involves crystallization of tissue fluids in the skin or subcutaneous tissue after exposure to freezing temperatures, with the degree of injury depending on the speed of cooling. NFCI is the result of long-term exposure of the feet to cold, wet conditions. Trench foot can occur at temperatures above freezing and its development is time-dependent, with the injury severity increasing with the duration of exposure. Immersion foot is usually associated with chronic immobilization of the extremities or a static, upright position involving cold water exposure, or both. Hypothermia occurs when the core body temperature reaches 35°C (95°F).

Military operations in cold, snowy environments increase the chances for minor cold injuries that can limit unit effectiveness due to lost man-hours. Cold weather increases the difficulty of performing tasks related to eating, drinking, and normal hygiene. An increased solar load due to reflected light from snow or high-altitude operations increases the risk of exposure to ultraviolet (UV) radiation. Snow can also conceal hazards in the terrain and increase the risk of falls, resulting in injury.

Snow blindness (solar keratitis) is a temporary visual disturbance due to injury of the conjunctiva and superficial cells of the cornea caused by UV light. Increased UV radiation can also affect the skin, causing sunburn and chapped lips. Excessive sunburn can affect the wearing of cold-protective clothing, thus increasing the risk of cold injury. Sunburn is also an indicator of the role of UV radiation in producing oxidative stress, which can depress the body's immune function and increase the risk of systemic infection.[3,4]

Exposure to cold, dry air depresses mucociliary function, which compromises lower airway defense mechanisms. Edema and vasoconstriction of upper airway mucosa cause rhinitis.[5] Accumulation of mucus secretions may block drainage of the sinus resulting in sinusitis. Interior environmental conditions (eg, overheated dry air) inside heated living spaces (eg, tents) can compromise normal respiratory functions.

Personal hygiene is more difficult in cold environments, and lapses in hygiene can lead to gastrointestinal disturbances. Cold weather and dehydration exacerbate such problems as constipation (due to changes in diet, dehydration, or unwillingness to defecate), which may eventually lead to development of hemorrhoids. Diarrhea can exacerbate the problem of dehydration.

Dental hygiene can be another problem in cold-weather military operations, owing to lack of time and facilities for adequate preventive care. Field diets (operational rations) consist of refined carbohydrates ingested at frequent intervals, which increases the need for dental cleaning. Lack of preventive care can cause acute necrotizing ulcerative gingivitis. Toothache in normal teeth or teeth with extensive restoration is common with prolonged exposure to cold. Cold-induced contraction and expansion of dental fillings in teeth may allow for bacterial invasion and subsequent tooth decay.[6]

Complexity and Multiplicity of Environmental Threats

The environment is characterized by both meteorological and geographical considerations that affect the injury potential associated with operational activities. Conditions are termed cold-wet when the temperature is around freezing and the ground alternately freezes and thaws. On the other hand, cold-dry conditions occur when temperatures are below freezing and the ground is frozen. The ambient temperature is affected by both wind and humidity. Geographical considerations are the natural features of the terrain. Operations in mountainous terrain increase the severity of the effects of cold temperature and increased solar load, while operations in flatlands suffer from little cover and increased exposure to wind. Desert environments can create a potential for cold injuries due to the extreme day-to-night variation in temperature. The presence of waterways, either open or frozen, increases the risk of cold injury resulting from water immersion.

Fig. 13-1. These US Army soldiers from the 6th Infantry Division (Light), shown setting up camp in Alaska, illustrate the problems of working in a cold, dark environment.

The combination of increased hours of darkness, icy surfaces, and wearing heavy, bulky clothing makes movement and work more difficult (Figure 13-1). In these conditions, the possibility of slips and falls causing severe muscle and connective tissue damage or broken bones is increased. Any injury in a cold environment enhances the risk of circulatory shock. Protection of a field casualty who is receiving first aid is more difficult, owing to the increased risk of cold injury when cold-weather clothing is removed.

Military personnel serving on ships operating in cold environments and personnel serving on land will experience similar problems with safety and cold injuries. Shipboard personnel will experience greater risks associated with hypothermia because of the threat of falling overboard. The time that an unprotected person can exert any effort to survive (swimming, pulling oneself out of the water) is 2 to 5 minutes in –1.6°C (29°F) seawater.[7] And whether on land or at sea, the accumulation of snow and ice on exposed metal surfaces (eg, tanks, ships) enhances the possibility of falls.

Issues Relevant to Military Operations in Cold Environments

Civilians seldom put themselves in high-threat situations and consequently do not suffer large numbers of cold injuries. Military personnel, on the other hand, have to function in whatever environment they are placed. Mission requirements may require prolonged cold exposure, limited preparation time for unit movements, limited resupply, and inadequate equipment. Many different military occupations will increase the risk of cold injury during cold-weather military operations. Personnel who handle petroleum products, antifreeze, or alcohol are exposed to extreme risk of frostbite because these substances remain liquid at temperatures well below –17.7°C (0°F). At these temperatures, contact of these substances with skin or mucous membranes can cause an immediate freezing injury. Saturation of uniforms with these liquid substances will negate insulation properties and expose the wearer to increased risk of injury.

The effect of cold weather on nuclear, biological, and chemical warfare operations can be significant.[8] A nuclear explosion on a frozen area will have greater blast effects, due to reflection of the blast waves, which increases the danger zone. A frozen danger zone will also compromise the ability of military personnel to quickly entrench. Radioactive fallout can be concentrated because of wind and snow conditions. Effective chemical warfare relies on the vaporization of the chemical to cause damage. In a cold climate, agents with high freezing points (eg, hydrogen cyanide, cyanogen chloride, Lewisite, mustard agent, and ammonia) will not be effective, but many nerve and choking agents (eg, tabun, sarin, soman, VX, VR-55, SA, phosgene, DP, and nitrogen mustards) will be effective. Agents that lack volatility at cold temperatures may become persistent agents and be deposited on the snow. Contact with these agents can result in their penetrating the military uniform, causing direct skin contact; or the agents can be carried on the military uniform into a warm area, such as a tent, where the agent may then vaporize and cause damage.

There are problems in the use and care of chemical protective gear (ie, mission-oriented protective posture gear, MOPP) at freezing environmental temperatures (0°C, 32°F). There is no standard procedure for wearing MOPP gear in cold environments, but usually it will be worn over the cold-weather clothing. This will require that parts of the cold weather clothing be removed to prevent overheating, increasing the risk of cold injury. Oral fluid replacement will be difficult while military personnel are in MOPP 4 gear because the external drinking tube will freeze. The rubber used for the mask will become stiff and brittle and more likely to tear. The filters used with the mask will freeze. The mask has metal rivets that must be covered to protect the face from contact cold injury. If the mask is carried outside the uniform, it will be dangerous to don until it is warmed. Wearing MOPP gear can increase risk of physiological injury or fatigue if

worn correctly because sealing it requires tight straps, which restrict blood flow. Chemical detectors will be less effective and autoinjectors containing nerve agent antidotes will freeze unless protected. Decontamination procedures are based on using water solutions, which will freeze. The use of nonfreezing solutions for decontamination will increase the risk, because these solutions will instantly cause a cold injury at freezing ambient temperatures. (Medically relevant features of nuclear, biological, and chemical protective equipment are also addressed in *Medical Consequences of Nuclear Warfare*[9] and *Medical Aspects of Chemical and Biological Warfare*,[10] other volumes in the Textbook of Military Medicine series.)

On ships serving in cold environments, major problems for personnel will stem from the accumulation of ice on exposed surfaces. Icing will occur first on small items and will affect lifelines and railings. The primary effects of topside icing are the changes in displacement and center of gravity, both of which increase the ship's instability. The severity of ice accumulation will depend on the type of ship, the ship's stability status, and its ballasting capability. These problems compound the safety considerations for shipboard personnel on the weather decks.[7] The increased instability of the ship will increase the danger that personnel will fall overboard, and it will also increase the amount of cold water on deck, which, in turn, further increases the danger of cold injuries.

COLD INJURY POTENTIAL

Environmental conditions in combination with physical activity, the duration of exposure, amount of protection, level of fitness, and individual cold susceptibility all contribute to an individual's risk for cold injury. Temperature, precipitation, and wind combine to increase the rate of body heat loss (ie, the intensity of exposure) and increase the risk of cold injury, which can be expressed as

$$\text{Risk} = (\text{time} \bullet \text{intensity of exposure}) \div (\text{adaptability to cold exposure} \bullet \text{technology})$$

where *time • intensity of exposure* is a measure of the cold stress (ie, the probability of developing cold injury), and *adaptability to cold exposure • technology* is a measure of an individual's resistance to the cold stress. The term "adaptability to cold exposure" can also be defined as thermocompetence, and "technology" refers to such items as clothing.

Intensity of Cold Stress

Freezing temperatures and low humidity favor the development of frostbite. Near-freezing temperatures in conjunction with moisture favors the development of trench foot. The presence of wind accelerates body heat loss and increases the risk for hypothermia. Cold water immersion produces the largest gradient for body heat loss and poses a significant threat of hypothermia. There are gradations of severity within each injury that are determined by the intensity and duration of exposure. Predictions for survival under different conditions can be obtained from a curve that plots estimated survival times against immersion in water at different temperatures (Figure 13-2).

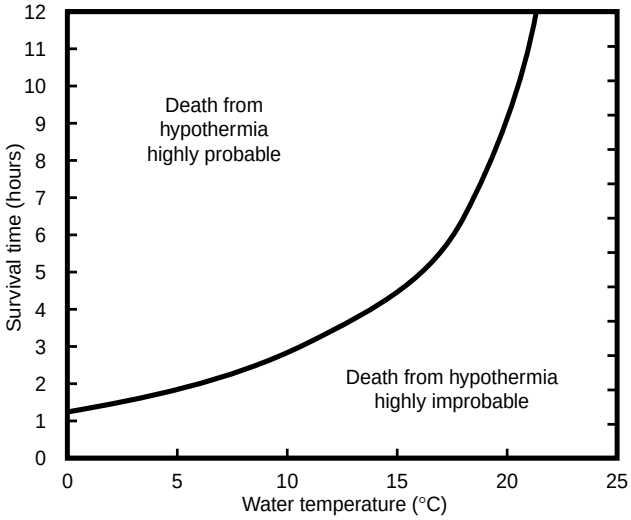

Fig. 13-2. This graph provides estimated survival times for humans immersed in different water temperatures. Survival times are based on an individual wearing a flotation device, so drowning is not possible. The survival time is based on minimal movement (swimming or treading water) and is based on the drop in core body temperature (ie, hypothermia). For example, a normal, uninjured, 70-kg man immersed (to the neck) in water at 0°C could be expected to survive (albeit in a hypothermic state) for 45 to 90 minutes. The *effective* survival time (not seen on this chart), on the other hand, is the time when a person can swim (without a flotation device) and could help himself out of the water. The speed of cooling is based on effective insulation, which can be due to an increased level of body fat or to protective clothing (antiexposure suit), or both.

Table 13-1 shows the relationship between core temperature and symptoms of hypothermia. The combination of wind and low temperatures creates a marked cooling effect on the body called the wind chill effect. Wind accelerates body heat loss under both cold-wet and cold-dry conditions. Wind increases convective heat loss from the surface of clothing because of its ability to penetrate loose-fitting clothing or openings, and wind removes the still, warm, layers of air trapped in the garments. If the clothing is wet, evaporative cooling is increased, which can account for up to 80% of body heat dissipation.

The addition of moisture to surfaces exposed to the environment greatly increases the rate of heat loss and increases the occurrence of cold injury. This moisture is usually from precipitation but can occur from perspiration soaking the insulation layers. In a cold environment with low humidity, perspiration contributes to body heat loss. Under cold-wet conditions, the combination of freezing and thawing causes wetting of the boots, which leads to cold injuries. The wetting of the skin of the feet will, over time, produce excessive hydration of cells and is a primary cause of trench foot.

The wind chill index, which establishes a risk of cold injury for different combinations of wind and temperature, has been developed and used as a tool for judging environmental risk (see Figure 12-4 in Chapter 12, Human Psychological Performance in Cold Environments). There are limitations for use of the wind chill index, in that it overestimates cooling power based on the effects of wind on the skin surface and underestimates cooling for clothed surfaces.

Adaptability to Cold Exposure

The ability of an individual to resist cooling involves many factors, including body composition, fitness level, fatigue, age, military rank, race, nutritional status, hydration status, tobacco use, legal and illegal drug use, and morale.

An increase in subcutaneous fat results in an increase in the insulation layer, which increases the resistance to cooling, during both exposure to cold air and immersion in cold water. However, military personnel tend to be more physically fit and consequently have lower levels of subcutaneous fat.

TABLE 13-1

CORE TEMPERATURE IN RELATION TO HYPOTHERMIA

°C	°F	Hypothermic Signs and Other Conditions
37	98.6	"Normal" oral temperature
36	96.8	Increased metabolic rate, in an attempt to balance heat loss
35	95.0	Maximal shivering
34	93.2	Individual usually responsive; normal blood pressure
33	91.4	—
32	89.6	Consciousness clouded; most shivering ceases
31	87.8	Pupils dilated; blood pressure difficult to obtain
30	86.0	Progressive loss of consciousness; increased muscular rigidity; slow pulse and respiration
29	85.2	Cardiac arrhythmia develops
28	82.4	Ventricular fibrillation may develop
27	80.6	Individual appears dead
26	78.8	—
25	77.0	Ventricular fibrillation may appear spontaneously
24	75.2	Pulmonary edema develops
23	73.4	—
22	71.6	Maximum risk of ventricular fibrillation
21	69.8	—
20	68.0	Heart standstill
19	66.2	—
18	64.4	Lowest accidental hypothermia patient with recovery

Adapted from Harnett RM, Sias FR, Pruitt JR. *Resuscitation From Hypothermia: A Literature Review for United States Coast Guard, US Coast Guard Headquarters*. Clemson, SC: Clemson University; 1979: 3. Contract DOT-CG-72074-A, Task 5.

Physical fitness has not been shown to correlate with decreased cold injury risk, but fitness levels are an important deterrent to fatigue, which is predisposing to cold injuries. Age has a significant effect on the incidence of cold injury, but military populations tend to be in the age range with the greatest resistance to cold injury. A low military rank correlates with an increased risk of cold injury because individuals of low rank have the greatest cold exposure for the longest duration.[11]

In a laboratory study,[12] black people were reported to have greater susceptibility to freezing injury than white people, which may be related to a stronger sympathetic nervous system response to cooling or a diminished cold-induced vasodilation response. Caution should be exercised in extrapolating this observation, however, because racial susceptibility to cold injuries has not been studied in battlefield situations.

Increased calorie intake is necessary to sustain the increased heat production and the increased work required to function in a cold environment.[13] Tobacco use is contraindicated in a cold environment because its vasoconstrictor action may increase the risk for peripheral cold injury.

Morale is a very important aspect of survival in a cold environment. Feelings of isolation, frustration, and depression can lead to fear for one's safety, which can negatively affect the will to live.

Technology

Humans have adapted to living in a cold environment by using protective clothing to survive. Clothing acts as insulation, which prevents body heat from escaping. Therefore, certain guidelines have evolved that should help in the selection of clothing. Dressing in layers, if possible, allows the soldiers to increase or decrease their insulation. Tight-fitting clothing that constricts blood flow to the extremities should be avoided. In a cold environment, a universal rule is to dress cool, because it is important to prevent the accumulation of perspiration. The accumulation of body oils and perspiration in fabrics reduces the insulation value of the fabric by reducing the air trapped in the fabric pores. The presence of perspiration in the clothing accentuates convective cooling and loss of body heat.

Tentage used in cold environments is designed on the same layering principle as cold weather clothing. The usual tent for army field operations is the Arctic 10-Man Tent. This tent has a strong, tightly woven outer shell, which is impervious to rain and snow. The inside liner is a lighter-weight fabric and is hung to provide an air space along the outer shell. This tent does not have a floor and suffers from the problem of moisture accumulation. After the first use, it becomes heavy and stiff and difficult to move. In contrast, the US Marine Corps uses a 4-man tent that consists of a floored tent and a rainfly that creates an anteroom for equipment storage. However, this tent does not have an insulative liner to create a dead-air space, which would make it more comfortable at colder temperatures. General purpose tents of various sizes may also be used for medical and command functions.

The larger tents are heated with the M1950 Yukon Stove, which is an effective heater but it may be the source of many injuries. The Yukon stove burns all fuels but is usually used with gasoline supplied from a 5-gal can mounted on a tripod outside the tent. When the Yukon stove is clean and set up properly, very little carbon monoxide (CO) accumulates inside the tent. There is a real danger of CO poisoning any time a stove (squad stove or individual stove) is used inside a tent without a vent pipe. CO is clear, odorless, and tasteless, which make it a very dangerous product of incomplete combustion and contaminant in unventilated quarters.

COLD INJURY PREVENTION

Function and performance in a cold environment are determined by maintenance of adequate whole body and local heat balances. Mathematical models have been developed for describing heat balance and simulation of the thermoregulatory processes involved, and have been used to develop guidelines for cold exposure (eg, work/rest cycles). The earlier models were descriptive; they were based on an analysis of heat exchange and physiological adjustments to thermal stress conditions.[14] Later models included a control component that responded to the effects of heat exchange and simulated human thermoregulatory adjustments.[15] These models may predict skin and core temperatures and allow an assessment of the risk of harsh environments on health and performance. One of the earliest predictors of response to cold was the wind chill index, which related to the cooling effect of wind on bare skin at different wind speeds. When cold weather clothing is worn correctly and all bare skin is covered, and all other logistical material (eg, food water, tents) are accounted for, there is no limit for

humans functioning in the cold. The most conservative method of estimating cold stress, the wind chill chart is divided into zones of little danger, moderate danger, and great danger. A safe method is to curtail all unnecessary training operations when environmental conditions fall in the red "great danger" area. This chart is still in widespread use for determining the danger associated with military operations. Later models have added values for ventilation, condensation, and water retention for clothing, and these models may be useful in prediction of survival in cold environments.[16]

However, there are many limitations to the use of the current models. The heat transfer in clothing under transient and dynamic conditions has been poorly described. The physiological components of the response are also poorly described, and the criteria for prediction of certain effects are unclear.[17] Perhaps the biggest problem is the inability of the models to incorporate individual variation. Because morale is such an important part of survival in the cold, behavioral components of the responses must be included if the models are to be useful. Models have been used to predict survival times in cold water and give general guidelines for safety. Models have also been used to develop work/rest cycles in cold environments.

Training

Individual training before deployment should stress the role of the individual in the prevention of cold injury. Fatigue, hydration, nutrition, lack of cold weather skills, tobacco use, and a nonawareness of weather factors are known to be very important precursors to cold injury. Physical conditioning should be maximized before deployment to a cold environment because more-fit individuals are better at resisting extremity cooling. Physical conditioning should continue in the cold environment. There is no limitation to exercise in cold weather if an individual is wearing proper layered clothing and using prudent techniques. Training in cold weather will reinforce the fact that military units cannot be beaten by the weather if they are adequately trained and prepared. Troops should have training exercises in which they wear the cold weather clothing before deployment and should perform the military maneuvers that will be expected in the cold environment.

Fatigue can occur even with superior physical conditioning. The combination of wearing heavy, bulky clothing and struggling against wind, ice, and cold will lower the threshold for fatigue. The wearing of the 18-lb Extended Cold Weather Clothing System (ECWCS) increases the amount of work being performed. The wearing of the vapor barrier boots (Type I or Type II) also increases the work demand, because weight (4-5 lb) carried on the feet has a much greater multiplier effect for increasing effort than weight carried on the body. Fatigue causes loss of attentiveness and diminishes physical coordination. Symptoms of fatigue are loss of coordination, dizziness, shortness of breath, and chills. One of the best preventives against fatigue is to always work within your capabilities. Working with a buddy is strongly recommended,[18] as neither the development of facial cold injuries nor the cold-induced personality changes associated with decreased core body temperature can be detected by the individual involved. The buddy system enables military personnel to look out for each other and recognize the added time and effort needed under the particular weather conditions.

Nutrition

Long-term deployment of a combat unit requires the use of operational rations (Meal, Ready-to-Eat [MRE]; Ration, Cold Weather [RCW]; Food Packet, Long Range Patrol Ration [LRPR]) until kitchen facilities can be established. The caloric requirement for cold environments is 4,500 kcal/d. Within 10 days, one hot meal will be provided. This meal will be made from canned ingredients (B rations) and will be served in addition to the hot fluids provided for consumption (a hot-wet meal). The second 10 days calls for one hot-wet meal per person per day, in addition to operational rations. The plan for the third 10 days is to furnish two hot-wet meals daily, in combination with one operational ration. Food supply is not a consideration on board ship, but personnel should be aware that exposure to cold will usually require greater effort, which demands an increased caloric intake (Figure 13-3).

Most operational rations require water for rehydrating and to improve palatability, but all rations require potable water to increase consumption. A method for heating the ration (MRE ration heater) or heating water (individual stove) is required. Studies[19] on the use of operational rations have shown that troops do not consume sufficient calories to maintain body weight, and it is suggested that food consumption should increase when time is set aside for hydration and food consumption on a regular basis. In addition, consumption is increased when meals are eaten in a group setting (so-

Fig. 13-3. Hot meals during cold weather operations are important, both to increase morale and to prevent cold injuries.

cial effect). Once hot-wet meals are available, it is important to serve them hot and to encourage immediate consumption.

Hydration

Supplying, purifying, and delivering potable water are difficult tasks in cold environments. Water intake is essential to maintain the body's chemical balance and has been shown to enhance food intake.[20] *Water intake is the most important survival requirement in a cold environment*. Each individual requires at least 4 qt (canteens) of water each day. Caffeinated beverages such as coffee and hot chocolate cause increased water loss, and increased water consumption is therefore required. An increased level of activity will also increase the requirement for water intake. Hydration problems are minimized when water is consumed on a schedule and consumption is verified. The symptoms of dehydration include an increased pulse rate, constipation, and dark urine in small quantities as opposed to the normal straw-colored urine.

During most field operations, water will be delivered to units in 5-gal containers or will be obtained by individuals using stoves to melt snow. All water sources should be considered unusable until purified by boiling (at least 12 min is required at sea level) or by iodine tablet disinfection. Effective disinfection is achieved in 4 hours, and flavor additives should not be added until after that amount of time has transpired. A completely frozen 5-gal can will require 8 hours in a heated environment to thaw. Direct heating of water cans (plastic or ceramic-lined) is contraindicated. If stoves are to be used, then a gallon of fuel will be sufficient to produce 13 gal of water (the amounts are altitude-dependent) from snow or ice, which then must be purified before being consumed. In cold environments, thirst is an insufficient stimulus to maintain hydration, so it may be necessary to engage in forced drinking to maintain hydration. On board ship, the danger of dehydration is no less of a problem and individuals should augment fluid intake. Additional water will be required for personal hygiene.

Clothing

The ECWCS is a layered insulating system designed to maintain adequate environmental protection between 4.4°C (40°F) and –51°C (–60°F) (Figure 13-4). The ECWCS has 23 components—a mixture of clothing items, handwear, headwear, and footwear—and uses moisture-management principles to move perspiration away from the skin. The ECWCS is issued by the unit, whose job determines the need for the field gear. Each individual service member in the designated unit should receive the complete clothing ensemble and be instructed on how to wear and care for the ensemble. There is a sequence for wearing the items and this sequence should be employed. *The fit of each item is critical*. Every item must be tried on in its correct sequence. If clothing is too tight, it will restrict blood flow and increase risk for a cold injury.

The current method for cold weather dressing is to layer the protection to trap air and also to allow some layers to be removed as needed. The concept of the clothing is to move perspiration away from the skin (using man-made fibers that do not absorb moisture) and to prevent outside moisture from penetrating the clothing (by means of a directional-flow nylon and polytetrafluorethylene laminate material). Even with this system, it is still neces-

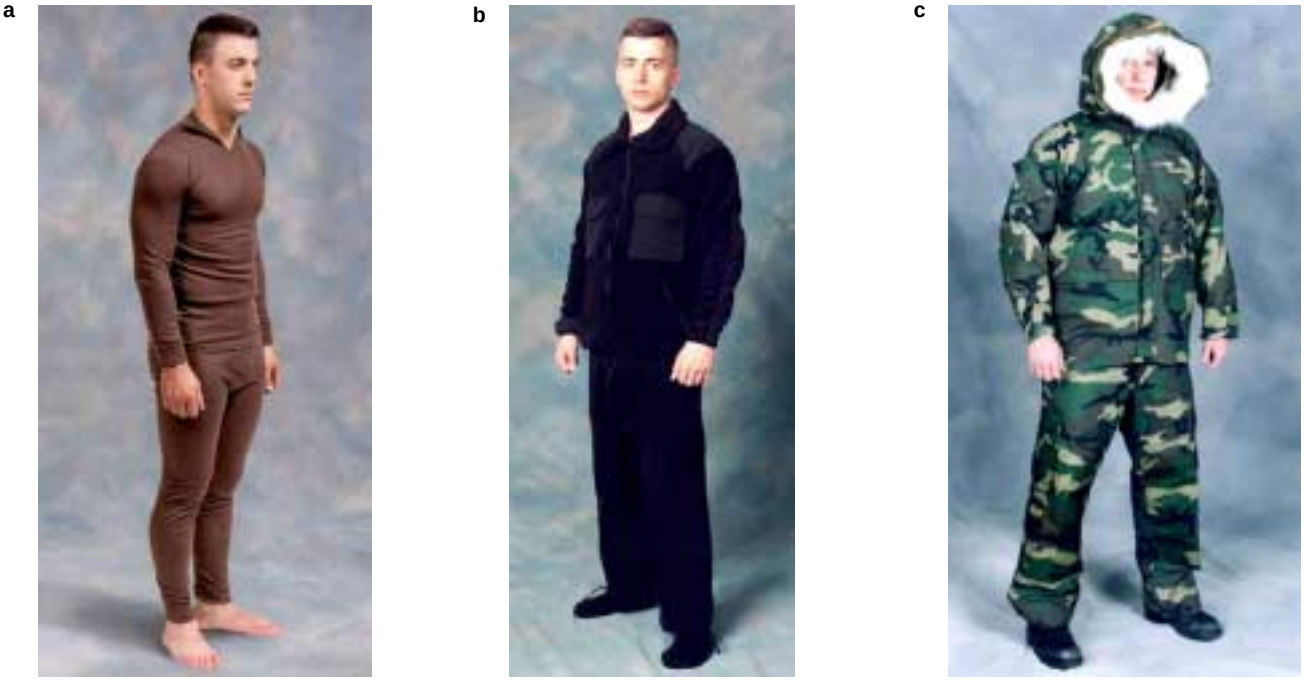

Fig. 13-4. These three views illustrate the layering principle on which the Extended Cold Weather Clothing System (ECWCS) is designed: (**a**) the polypropylene underwear, (**b**) the polyester pile insulation layer, and (**c**) the outer garment. Source: US Army Soldier and Biological Chemical Command, Soldier Systems Center, Natick, Mass.

sary to minimize the amount of perspiration produced by venting the clothing or removing clothing items when not needed to maintain warmth. Because the fabrics in the undergarments perform a wicking action and the outside garments rely on microscopic pores to repel moisture, it is very important to keep the clothing as clean as possible. These general guidelines have led to widespread use of the acronym COLD, which stands for *C*lean clothing, don't *O*verheat, dress in *L*ayers, and keep the clothing *D*ry.

New fabrics have been developed to allow one-way movement of moisture and have been incorporated into ensembles that stress the removal of moisture from the skin to negate some problems associated with the accumulation of perspiration. Under this concept, clothing made from natural fibers (cotton or wool) should not be worn with the uniform. Cotton retains moisture and becomes clammy, which encourages body cooling, whereas wool retains moisture and creates a barrier to the movement of moisture. The only time an exception is made is when wool garments (which retain insulation when wet) are worn as part of the cold weather uniform on US Navy ships.

The fabrics in the ECWCS ensemble require special attention. The polypropylene and polyester fiberpile undergarments must be gently washed in cold water. Liners, coats, and trousers can be washed in warm water. All items should be drip-dried or dried on very low heat settings. Overwhites (ie, camouflage garments for wearing in the snow) should be spot cleaned and washed in warm water using only a powdered detergent. Pressing any item in the ensemble will damage the fabric and should not be done. External rips and tears can be patched with fabric tape or stitched.

The most critical clothing component in a cold environment is the wearing of a proper boot. No single ideal boot exists for all cold weather military applications, so all boots offer a compromise solution. Cold feet will inhibit cold weather operations faster than any other environmental factor. The best approach to prevent cold injury is to practice these techniques for foot care:

- *Make sure the boots fit properly*. The sock combination involves wearing a thin polypropylene sock next to the skin and then a heavy wool sock to absorb moisture. If the boots are too tight in the toe area, get a larger size. If the operation will require the use of loaded backpacks, then the boots should be fitted

while the soldier is carrying the weight, because feet expand when a load is added.
- Remove the boots and socks two to three times daily. Wash, dry, massage, and exercise the feet to restore circulation. Put on clean dry socks. If possible, dry out the inside of the boots with a towel.
- *Do not sleep with footwear on*. Before sleeping, remove the footwear and dry it and the feet. When in a static position, avoid (as much as possible) standing in mud and water.
- Exercise the feet (wiggle the toes) and legs to stimulate blood circulation. Elevate feet to minimize swelling. Be alert for numbness. Seek medical help at the first sign of injury. Continued exposure will make the injury worse.

Several different boots can be worn with the ECWCS, and the decision of which to wear should be based on environmental conditions. The boot selection includes the cold weather boot (Type I), extreme cold weather boot (Type II) (Figure 13-5), standard combat boot, GORE-TEX (manufactured by WC Gore and Associates, Newark, Del) combat boot, and ski-march boot. When either the Type I or Type II boot is used, foot care becomes extremely important because these boots are waterproof and the skin of the feet will become macerated over time, due to the fluid buildup around the foot. The fluid is not able to evaporate, increasing the risk of incapacitating foot injury.

US Navy cold weather clothing is divided into two temperature ranges:

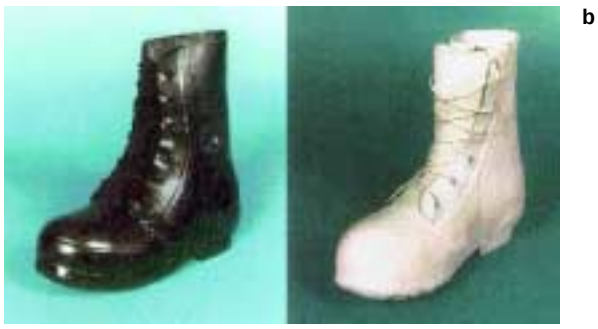

Fig. 13-5. (**a**) For cold weather, the vapor-barrier Type I boot is for use in temperatures down to –28°C (–20°F). (**b**) The vapor barrier Type II boot should be used in extreme cold weather, all temperatures below –28°C (–20°F). Reprinted from US Army Soldier and Biological Chemical Command, Soldier Systems Center, Natick, Mass.

- A-2 intermediate clothing, for temperatures down to –6.7°C (20°F), and
- A-1 extreme cold weather clothing, for temperatures down to –17.6°C (0°F).

One-piece antiexposure suits consisting of a durable external shell, a closed-cell foam insulating layer, and an inside liner are adapted for deck use on all ships as extreme cold weather clothing, with the added benefit of increased cold water survivability. These exposure suits are designed to be used only in certain environments, because they do not allow layering or venting to prevent moisture accumulation. Water-resistant insulated jackets and trousers can be worn over normal work uniforms but will require a life jacket. Headgear (wool watch caps, fleece-lined caps, balaclavas, and hoods), footwear (Type I vapor barrier boots or extreme cold weather mukluks), handwear (gloves or mittens), and underwear (thermal underwear and heavy wool socks) are included in the cold weather clothing ensemble. Because the environment on ships will be wet, the use of wool is more acceptable aboard ship than in dry, cold environments.

The availability of laundry facilities on US Navy ships makes it easier to maintain the cold weather clothing. To maintain its insulative effectiveness, it is essential that the clothing be washed and dried according to the manufacturer's specifications. In preparation for cold weather operations, backup clothing must be available. The greatest problem with the ECWCS system is the footwear because the boots are waterproof. Personnel must be encouraged to change socks often. Shipboard personnel should strive to stay dry (water spray or perspiration) and should change clothing as often as needed to maintain dryness.

Shelter

One individual must always be awake when a stove is used in a tent. The cold weather ensembles are not fireproof and can cause severe injury because the undergarments made from man-made fabrics will melt onto the skin. The four-man tent does not provide a vent pipe, and ventilation is accomplished by opening the outside door.

When regular tents are not available and shelter is required, either natural shelters or improvised shelters can be used for short durations. An effective shelter must meet the requirements for protection from the elements, heat retention, ventilation, and some type of facility for drying wet clothing. It is possible to use natural shelters such as caves, rock

overhangs, or hollow logs, but their use is risky when a fire is used. Controlled fire is necessary in cold environments, because uncontrolled fire is a threat to survival. Many types of shelters can be built if enough time and resources are available. A simple snow wall or snow trench can be used as a shelter from wind. A snow cave requires deeper snow and considerable effort to make, but it can offer excellent environmental protection for more than one or two people. If ponchos are available, then a one- or two-man shelter can quickly be assembled. With sufficient time and using trees and evergreen boughs, more elaborate tentlike structures (eg, a lean-to, A-frame, or tepee) can be constructed.

Individual Awareness During Cold Exposure

Cold injuries are preventable when each person of a ship's crew or a land-based military unit is prepared and trained for operations in cold weather. Awareness begins with the individual and should begin before deployment to a cold environment. Prevention of cold injuries begins with an assessment of preexisting risk for cold injury by the medical officer's examination of individual medical records for medications or existing medical conditions. Any medications that might affect blood flow, thermoregulation, or cognitive function would be contraindicated for cold exposure. (Existing conditions, such as Raynaud's syndrome, diabetes mellitus, chronic pulmonary disease, obstructive vasculopathies, peripheral neuropathies, or a prior cold injury will increase the risk of a cold injury; personnel with such conditions would not be deployed.) Immunizations for influenza and tetanus are important prophylaxes before deployment. The importance of maintaining a nutritional intake commensurate with physical effort, along with adequate hydration to prevent hypohydration, should be stressed. With command checking, the use of a communal spot for urination, coupled with proper discarding of uneaten rations, can be used as indicators of hydration as well as caloric intake. Individuals should be aware of the initial signs of cold injuries and encouraged to report these as soon as possible. The unit medics should document all reported incidences to the medical staff to avoid long-term problems.

All personnel should be advised to develop a buddy system to aid in the early detection of possible cold injury. Because early detection is important in limiting the extent of injury, it is important for buddies to observe and communicate effectively with each other. The medics should work individually with service members to make sure they know the early signs of cold injury, and emphasize that just because a body part has stopped hurting does not mean that the danger of cold injury has ended.

Leadership

The importance of leadership by example in a cold environment cannot be overstated. Several problems areas are unique to the cold environment. When an individual dons several layers of clothing and puts up the parka hood, the hearing and field of vision are restricted, and individuals can become oblivious to their surroundings. Team efforts are critical; a manifestation of cold stress is a tendency to withdraw within oneself and assume a cocoon-like existence. Mental processes become sluggish. Cold-stressed personnel may want to stay in warm tents and sleeping bags to escape the cold, and in the process will neglect their duties, even to the extent of compromising security. The remedy for these behaviors is physical activity. It is very important to keep individuals and entire units informed and involved in the activities. Special attention should be given to troops who grew up in warm environments and are not familiar with cold weather activities.

Military operations in mountain or cold climates require careful assessment of time and distance factors. These factors must always be considered when movements are planned, and safety must not be compromised so that certain timetables can be met. It is important to set a reasonable pace and maintain that pace to conserve as much energy as possible; energy conservation helps prevent cold injuries.

Field Signs and Prevention of Cold Injuries

To minimize cold injuries, an effective rule for the field is that individuals should always be able to feel their toes and fingers and, if the digits are numb, then the medic or buddy should check them. If the digits are insensitive, the tendency is to warm them as fast as possible. However, this approach has an inherent peril: a soldier may not perceive the heat that is being applied, and the cold injury will be exacerbated by a thermal burn. Cold injuries are classified as localized freezing (frostbite), localized nonfreezing (trench foot, immersion foot), and generalized (hypothermia).

Frostbite. Frostbite, a freezing injury, is characterized by an uncomfortable sense of cold, followed

by numbness. Individuals may complain of tingling, burning, aching cold, sharp pain, decreased sensation, or no sensation. Visually, the skin will turn from red to waxy white. The areas most likely to be affected are the nose, toes and fingers, ears, cheeks, forehead, and exposed wrists.

Prevention of frostbite involves dressing properly, keeping clothing clean and dry, avoiding fatigue, not touching bare metal, being aware of signs of frostbite, and using a buddy system to watch for signs of injury.

Trench Foot. Trench foot, a nonfreezing cold injury, results from long-term exposure of the feet to above-freezing cold and moisture, coupled with the failure of personnel to take proper care of their feet during cold-wet operations. It is one of the most common problems facing personnel in cold weather operations. With trench foot, the feet appear pale and feel cold, numb, and stiff, and walking is difficult. The feet swell and become very painful. They will appear blotchy purple with waxy skin and poor circulation.

Prevention of trench foot depends on keeping the feet clean and dry. To prevent trench foot, personnel should change socks often, use a sweat suppressant on feet (ie, DrySol, manufactured by Person & Covey, Inc, Glendale, Calif), and use exercise and massage to increase circulation when feet are warm and dry.

Hypothermia. Hypothermia occurs when the body is unable to maintain adequate warmth and the core body temperature drops. Signs of hypothermia include uncontrollable shivering with an impaired ability to accomplish complex tasks. Together, shivering and vasoconstriction of the fingers and toes indicate that the individual is in danger of developing—or has already developed—mild hypothermia. Shivering *by itself* is not a solid indicator of hypothermia. The victim may feel very tired, experience muscle weakness, exhibit loss of coordination, and display atypical behavior and poor decision making. PHYSIOLOGICAL WARNING: These signs *must* be taken seriously by the person in charge.

Prevention of hypothermia depends on dressing properly for the environment and maintaining body heat. On board a ship, wearing a one-piece antiexposure suit can reduce the risk of hypothermia associated with falling overboard.

Field Treatment

Freezing and Nonfreezing Cold Injuries. The first step in cold injury management is to detect the injury. Pain is evident when tissue starts to freeze but will subside as the injury worsens (see Chapter 14, Clinical Aspects of Freezing Cold Injury). In addition, the injured (ie, frozen) extremity may be difficult to examine directly due to its being covered by clothing. When an injury is suspected, the injured area must be protected from further injury by cold or trauma. The decision to rewarm the injury should be based on the safety of the individual and whether he or she can safely be evacuated from the cold environment. Once the tissue is thawed, it is more susceptible to a second freezing injury, which will worsen the consequences of the injury. The injury should be assessed, and evacuation should occur as soon as it can be done safely. The injured extremity should never be exposed to temperatures above 39.4°C (103°F), which could aggravate the injury. The injury should be protected against trauma during the evacuation. Careful records should be maintained concerning all aspects of the injury and treatment. If the injury is a nonfreezing cold injury (see Chapter 15, Nonfreezing Cold Injury), the same problems of detection, protection, and evacuation are encountered. Both freezing and nonfreezing cold injuries are slow to evolve and even slower to resolve, and evacuation—as speedily as is safely possible—is recommended.

Hypothermia. The challenge of rewarming the hypothermic individual in the field is to know his or her core body temperature, which indicates the degree of hypothermia. Assessing core temperature in the field, however, is not possible without low-reading thermometers to measure rectal or esophageal temperatures (for further information, see Chapter 11, Human Physiological Responses to Cold Stress and Hypothermia), and these are not available for field use. As a consequence, the rule of thumb is to stabilize the victim of hypothermia in the field and remove him or her from the cold environment.

There are many techniques for rewarming a victim of hypothermia, but they must be used *with caution*. First, in the field, a victim of mild hypothermia (the victim can talk but seems disoriented and is shivering) can be placed in an insulated, warmed sleeping bag so that he or she can shiver until warm. If the victim is wet, it is imperative to remove the wet clothing before putting him or her inside the sleeping bag. Putting the hypothermic individual between two volunteers, for sharing of body heat, can be effective, but only for mild cases of hypothermia. This method requires no heating units but is also the least effective. Other, more effective techniques for rewarming a victim of hypothermia in the field are not recommended because the equipment necessary to monitor cardiovascular changes

is rarely available. In general, the only method for rewarming available to field troops is the use of shared body heat. While this method will not be effective in rewarming a severely hypothermic individual, it (along with putting the person into a warm, insulated sleeping bag, out of the weather) will help stabilize the victim and prevent more heat loss. When treating a hypothermic casualty in the field, the main concerns are (1) to prevent further heat loss and (2) to remove the casualty from the cold environment, while (3) not decreasing the casualty's chances for survival by attempting to implement aggressive rewarming techniques.

The second method involves rewarming with warm water in a portable bathtub or life raft. The water temperature should be no greater than 42.2°C (108°F), and the warm water will have to be replenished. The casualty's core body temperature should always be monitored, along with the heart rate and blood pressure.

The third method involves the use of a warmed, humidified mixture of warm air and oxygen to limit heat loss and start rewarming. The temperature of the air mixture is critical, as it should not exceed 46°C (115°F). This method will inhibit shivering, which may give medical personnel a false indicator that the patient is improving.

The fourth method, which should be used in a medical treatment facility, involves infusing warmed intravenous solutions.

Beyond Cold Stress: Related Problems in Cold Climates

Cigarettes and alcohol consumption increase the risk of cold-related injury. Smokers maintain blood levels of CO that are 4- to 5-fold higher than those of nonsmokers, making smokers more susceptible to CO poisoning as well as decreased peripheral blood flow. Alcohol intake and CO poisoning can be related. Alcohol impairs judgment and coordination, which increases the risk of falls, and reduces alertness, which may result in a greater risk of fire and CO poisoning.

CO poisoning can result from sleeping in unvented, heated tents or from sleeping in a vehicle that is left running. Military personnel should always be aware of their surroundings and the direction of the wind. A vehicle parked downwind with its motor running can create a dangerous situation, in which exhaust from the vehicle will penetrate the cab.

Exposure of unprotected skin and eyes to sunlight may cause sunburn and snow blindness. Military personnel should use an alcohol-free sunscreen that contains *p*-aminobenzoic acid to block UV radiation (must have blocking factor ≥ 15). The use of sunglasses that block UV radiation will minimize the chance of snow blindness.

Lapses in personal hygiene can increase the chances of illness and disease. The armpits, groin area, face, ears, and hands should be cleaned daily by using sponge baths, air baths, or by rubbing with a dry cloth while in a tent or shelter. Hair, fingernails, feet, and mouth and teeth should be cleaned regularly. Facial hair allows ice to form near the skin, possibly predisposing it to frostbite.

Special Situations

Cold Water Immersion

Military personnel who are operating around open water must be protected against drowning and immersion syndrome. Immersion syndrome occurs when the water is cold enough to cause apnea and cardiac arrest in certain individuals (for additional information, see Chapter 17, Cold Water Immersion). Individuals who withstand or survive these immediate challenges may have a decrease in core temperature, causing excessive fatigue and confusion, leading to poor judgment that results in drowning. Because water conducts heat 32 times faster than air, any efforts to tread water or swim will increase the body's heat loss and hasten the onset of hypothermia; therefore, it is *essential* that shipboard personnel wear a personal flotation device.

Several techniques, all recommended by the military, will increase survival time in cold water. The most obvious is to avoid direct exposure to the water; in the case of a sinking ship, personnel should enter the water in a raft or lifeboat. They should wear or don a personal floatation device as soon as possible, and wear several layers of clothing. Personnel who are in the water should avoid movement and use the heat-escape-lessening posture (HELP; Figure 13-6). HELP minimizes the exposure to cold water of the swimmer's groin and the lateral surface of the chest because the arms are folded across the chest and pressed to the sides. The knees are drawn up and the legs crossed at the ankles. If more than one person is involved, the huddle position should be used to reduce heat loss. In this position, the chest, abdomen, and groin area should be pressed together. These survival techniques require that personnel practice and wear personal flotation devices. Treading water in the HELP position in heavy seas is a major challenge.

Fig. 13-6. (**a**) With the individual wearing a personal flotation device, the heat-escape-lessening posture (HELP) can increase survival time in cold water. The swimmer assumes a fetuslike position, which minimizes cold water exposure of the groin and lateral chest. (**b**) The huddle position should be used when more than one person is in cold water, and all are wearing personal flotation devices. In addition to conserving each individual's body heat, the huddle position helps prevent the swimmers from becoming separated before they are rescued. Reprinted with permission from Pozos RS, Born DO. *Hypothermia: Causes, Effects, Prevention.* Piscataway, NJ: New Century Publishers; 1982: 94, 96.

The huddle position requires that all participants be able to tread water.

The following is the basic order of cold water rescue:

- Make sure that you are in a safe position before you attempt a rescue operation. Do not make yourself another victim.
- Try to reach the individual and pull him out of the water.
- If you cannot reach the individual, throw him a floatation device and attempt to tow him to shore.
- As a last resort, because this places the rescuer at risk of hypothermia, swim to the individual. If the casualty is unconscious, then swimming to him will be the only option.
- Make sure that if you swim to the casualty, you are tethered to a buddy on shore or in a boat. Do not attempt this procedure by yourself.

Field management of a submersion incident involves the ordinary ABCs of resuscitation: ensure a patent *a*irway, ensure that the casualty is *b*reathing, and ensure that the casualty is not in *c*irculatory shock. The basic cardiopulmonary resuscitation procedures should be followed. Evacuate the casualty to an appropriate medical treatment facility as soon as possible.

Mountain Operations

Although the particular medical problems associated with military operations in mountain environments are discussed in Volume 2 of *Medical Aspects of Harsh Environments*, a brief overview of the prevention of cold-related injuries at altitude is included here in the interest of completeness. For soldiers fighting at high altitude, such as mountain terrain, the main physiological effects are hypobaric hypoxia, or a reduced atmospheric oxygen concentration leading to less oxygen in the blood, and cold stress. Humans can tolerate exposure to extreme altitude for only short periods without supplemental oxygen. With an acute ascent to high altitude, plasma volume immediately decreases owing to diuresis and the shifting of fluid into the cells. This

shifting of fluid accounts for the increased hematocrit observed at altitude.

Three major clinical problems can occur with acute exposure to high altitude. The first is acute mountain sickness (AMS). The signs of AMS are rapid or irregular breathing, rapid pulse, and vomiting. Symptoms include headache, nausea, depressed appetite, generalized weakness, and dizziness. The second condition is high-altitude pulmonary edema (HAPE), in which fluid accumulates in the lungs. The symptoms of HAPE include a persistent cough; discharge of a pink, frothy sputum; disorientation; and fainting. Signs include cool, clammy skin; rapid breathing; a rapid, weak pulse; and blue lips. The third condition is high-altitude cerebral edema (HACE), which causes swelling of the brain. Signs of HACE include bizarre behavior, hallucinations, confusion, excessive fatigue, and coma.

The best treatment for these conditions is to avoid them by careful staging of the rate of ascent to the high altitude. These medical conditions are rare below 8,000 feet but can occur with rapid ascent to 8,000 feet and beyond. Scheduling a 48-hour rest at 8,000 feet, followed by a 24-hour rest after reaching 10,000 feet, and proceeding no more than 1,000 feet per day up to 14,000 feet will minimize the occurrence of all three conditions: AMS, HAPE, and HACE. AMS is not fatal but may progress if not treated by descending to a lower altitude; however, both HAPE and HACE can be fatal and require immediate medical attention.

When military units operate in mountain terrain, increased demands are made on personnel, owing to the terrain and weather. Weather becomes a factor because it can superimpose the rigors of the cold climate on the altitude stress. A cold injury at altitude is not different from a similar one at sea level, but the variable weather at altitude can rapidly increase the risk of weather-induced injuries. In addition to the possibility of AMS, there is a greater exposure to UV radiation at altitude because of the thinner air, causing less filtering and increased reflection of the UV radiation off the snow cover. This leads to a greater risk for snow blindness.

Leaders of military units must be especially vigilant to methods of preventing injuries. Standard leadership practices include careful evaluation of the terrain to plan routes and time requirements, and to assess difficulty. Leaders should keep in mind the ability of the unit, keep the unit together as a group to minimize straggling, and maintain a steady pace, incorporating rest breaks to allow adequate maintenance of hydration and nutritional status. In a difficult environment, the unit is only as strong as its weakest link. The usual procedures of individual protection apply and include the proper dressing and establishment of protection from the environment.

SUMMARY

Cold injuries can occur at any temperature below freezing, and nonfreezing cold injuries can occur at temperatures above freezing. Hypothermia occurs when the core body temperature falls to 35°C (95°C). As the environmental temperature decreases and the wind velocity increases (eg, during mountain operations), the danger of cold injuries increases. Military operations in extreme cold pose great risk for individuals in terms of injury potential, and training and constant attention to detail are required to avoid these injuries. Winter weather does not forgive and it cannot be defeated. For the unit to survive and function, command leadership must understand the limitations and problems associated with operations in hostile environments, and train and prepare the personnel.

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USA AG Publications
2800 Eastern Boulevard
Baltimore, Maryland 21200

No authors, editors, or dates of publication are listed; the manuals are revised occasionally but the titles and identification numbers remain the same.

Department of the Army. *Basic Cold Weather Manual*. Washington, DC: DA. Field Manual 31-70.

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Chapter 14

CLINICAL ASPECTS OF FREEZING COLD INJURY

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INTRODUCTION

Military medical officers need to be aware of the various modes of freezing cold injury (FCI), or frostbite, because it has been a persistent problem over the ages, usually associated with mountain warfare; winter military campaigns; or the civilian catastrophe of deprivation of shelter, food stocks, and warm clothing in the wake of military conquest (Exhibit 14-1). In addition, natural calamities (such as famine, earthquake, pestilence, flood, and fire) have left many communities exposed to harsh winters, resulting in FCI. FCI due to altitude exposure has occurred worldwide; the injury is often associated with freeze-thaw-refreeze injury, or with hypothermia, mountain sickness, and cerebral or pulmonary edema.

To the various cold-weather activities on Earth we now have added space exploration. In spring 1993 this author had occasion to consult with and share the treatment of an astronaut who froze his fingers at –143°F in a simulated space chamber and space walk. Even lower temperatures may be anticipated in that environment. With the advent of men and machines into space, further exposure to extremes of temperatures, greater than those on Earth, are expected. As the explorations of man continue into the wilderness throughout the world,

EXHIBIT 14-1

MILITARILY RELEVANT MODES OF FREEZING INJURY

1. True freezing cold injury (FCI, also called frostbite), superficial or deep.
2. A mixed injury: immersion (cold–wet) injury (as in trench foot) followed by FCI. The result is often disastrous, with great tissue loss.
3. Freeze–thaw–refreeze injury, wherein freezing is followed by thawing at any temperature or by any method, followed by subsequent refreezing. This is also a disastrous event with total tissue destruction and early mummification of distal tissues, often within 5 to 7 days, even earlier.
4. High altitude environmental freezing; this injury is associated often with hypoxia, accompanied by hypovolemia, dehydration, and extremity freezing. The prognosis is poor if associated with other trauma. An interesting aspect of altitude injury occurs in mountain climbers wearing neoprene stockings with tightly laced boots. At high altitudes (eg, 3 15,000 ft on Mount McKinley) the atmospheric pressure is halved. This allows expansion of gases in cellular foot covering so that in a tight boot, often double-layered, expansion outward is blocked and the pressure is then directed downward against the foot, cutting off circulation and permitting a freezing injury. The ambient temperature at altitude decreases approximately two Centigrade degrees (3.5°F) for every 1,000 ft of increase in altitude. The temperature becomes stable at about –55°C (–67°F) at an altitude of 35,000 ft or higher, and exposure to these very low temperatures may instantaneously result in severe injury to exposed body parts.
5. Extremity compartment compression from any cause, followed by freezing. Very poor results follow if the compartment pressures are not relieved by medical or surgical means.
6. Extremity fractures or dislocations followed by freezing. The final result is poor if the fracture dislocation is left unreduced. The best results follow rapid rewarming.
7. Hypothermia, associated with superimposed FCI of the extremities. Paramount importance is given to restoration of heat in the victim, under total physiological control and monitoring. Best results for FCI appear to be associated with tub rewarming of hypothermia and simultaneous thawing in warm water of the frozen extremity. The danger here is a sudden release of metabolites and the release of excessive amounts of potassium from muscle degradation and injury, which may cause cardioplegia. The immediate balance of electrolytes and the restoration of normal pH levels is imperative. The excellent method of rewarming with peritoneal dialysis may require almost simultaneous warming of the frozen extremity by other means.
8. FCI with superimposed burn injury, or burn injury with superimposed FCI. In addition to military casualties, medical officers may also see civilians whose cold injuries are perhaps confounded by their age or previously existing conditions:
9. Freezing injury in children often results in epiphyseal necrosis. So fragile is the epiphyseal plate of small children that very little exposure time or lowered temperature is required to cause epiphyseal damage.
10. Freezing superimposed on small vessel disease, as found in diabetes or peripheral vascular disease.
11. Congenital deformity of hands or feet associated with neurovascular deficit and superimposed freezing.

searching for oil, minerals, and the bounty of the oceans, it is more appreciated that hypothermia, immersion injury (now called nonfreezing cold injury [NFCI]), and FCI are injury risks of those who work and live in the arctic and subarctic areas, including the circumpolar sea of the Arctic and Antarctic oceans and the cold northern seas of the Atlantic and Pacific oceans.

The Experience of Larrey

There probably is no better introduction to a chapter on FCI than excerpts from the *Surgical Memoirs of the Occupation of Russia, Germany and France*, by Baron Dominique Jean Larrey. The writings of Baron Larrey, Chief Surgeon of Napoleon's Grande Armée, indicate his distrust of warming by heat, and his opinion held sway in Europe and America for well over 100 years. One of the pillars of the "modern" thawing technique of the frozen extremity has been and is rapid rewarming in warm water (37.7°C–41°C; 100°F–106°F). It was not always so.

In his memoirs regarding the French army's advance and retreat during the Russian winter campaign (1812/13), Larrey writes:

> Persons were seen to fall dead at the fires of the bivouacs—those who approached the fires sufficiently near to warm frozen feet and hands, were attacked by gangrene, in all points, where the vital powers had been reduced. These fatal occurrences mutilating the majority of our soldiers, threw them into the power of the enemy—unfortunate was the fate of him, who, with his animal functions nearly annihilated and his external sensibility destroyed by the cold, should suddenly enter too warm a room, or approach too near a large bivouac fire. The projecting parts of the body, grown insensible or being frozen and remote from the center of circulation, were attacked with gangrene which manifested itself at the same moment, and was developed with such rapidity, that its progress was susceptible to the eye—or else the individual was suffocated by a sort of turgescence, that apparently invaded the pulmonary and cerebral systems. The individual perished in a state of asphyxia.[1(p83)]

Larrey continues:

> It will easily be conceived, after what I have just said, why, in mortification of some external part of the body, caused by cold, instead of submitting it to heat, which provokes gangrene, it is necessary to rub the affected part with substances containing very little caloric, but which may absorb a good deal at the moment of their melting, and transmit it to the frozen part by rubbing. For it is well known that the effect of caloric on an organized part, which is almost deprived of life, is marked by an acceleration of fermentation and putrefaction. Before pointing out the means to be employed, let us succinctly describe the symptoms which characterize congelation [freezing]. The part laboring under this affection is whiter than other parts of the surface of the body; all its sensibility is extinct and the individual has no longer any sensation in it. [Even today, an excellent description of the frozen part.—W.J.M.][1(p84)]
>
> . . . .
>
> Snow and ice are the substances, to which recourse should be had, for the first application. Dry frictions also, are very serviceable, and should always be made with substances which possess but little heat. I employed no other means for defending myself from gangrene affections, which would at least have taken place in my toes and fingers, for they were frequently deprived of all sensibility. In this state, I took care to rub the affected parts with snow, and continued, as much as possible, the use of these or dry frictions.
>
> Should these remedies fail, the part ought to be plunged in cold water, in which it should be bathed, until bubbles of air are seen to disengage themselves from the congealed part. This is the process, adopted by the Russians, for thawing a fish. If they soak it in warm water, they know from experience, that it will become putrid in a few minutes; whereas, after immersion in cold water, it is as fresh as if it had just been caught.[1(pp84–85)]

Larrey further comments that

> [n]ot even the overwhelming Russian forces, nor the distances traveled in the retreat, caused so much problem as the weather. The most cruel sufferings experienced in our retreat (from Moscow) were undoubtedly cold and hunger.[1(p76)]

In addition, he gives notice to the fact that

> not far from the situation in which we endured

Fig. 14-1. *The Crossing of the Berezina River, 26 November 1812.* Watercolor by an anonymous artist; attributed elsewhere to General Fournier-Sarloveze, a participant in the action. Harassed by the enemy, overwhelmed by cold, the remnants of the summer campaign of Napoleon's Grande Armée retreated from Moscow in the fall of 1812; many attempted to swim the Berezina River, falling victim to hypothermia as well as extremity freezing. Thousands of French soldiers perished from starvation, dehydration, hypothermia, and freezing; and from unrelenting Cossack attacks on their flanks. The Grand Armée lost more than 80% of its remaining troops, one of winter's greatest triumphs over a military force. Reproduced with permission from Musée de L'Armée, Paris, France.

> so many hardships, Charles the XII of Sweden, a hundred years before the French debacle of 1812–13, lost an entire division of his army, in consequence of those two united causes, hunger and cold.[1(pp79–80)]

In retrospective evaluation, Larrey's comments concern men who were constantly harassed by the enemy, overwhelmed by freezing weather, hungry, emaciated, and without shelter or adequate clothing—and who certainly were hypovolemic, dehydrated, and in various states of hypothermia, often with severe peripheral extremity freezing. Regardless of current thought, Larrey's graphic, descriptive passages on the plight of soldiers at bivouac fires, written almost 2 centuries ago, state resoundingly that no more disastrous event occurs to the frozen extremity than thawing with excessive heat, with perhaps one exception. The catastrophe following thawing with excessive heat is matched only by a further event common in retreating or disorganized, beaten armies: that of freezing, then thawing by any means, followed by refreezing.[2–7] For a military surgeon, or anyone in a leadership position, no better instructional course on the tragic freezing disasters of winter warfare exist than the memoirs of Larrey, in his description of the invasion of Russia with a French army of more than 400,000 men and its subsequent destruction during the famed retreat from Russia, the French losing nearly 80% of the 100,000 men who survived to retreat from Moscow (Figure 14-1).

Implications for the Future

It comes as no surprise then, that in the same Russian area, more than 130 years after the destruction of Napoleon's Grande Armée and more than 200 years after the winter loss of the army of Sweden's Charles XII in the nearby forests of Lithuania (see Chapter 10, Cold, Casualties, and Conquests: The Effects of Cold on Warfare), that in

World War II, a modern, massive German army was roundly defeated in the winter of 1941/42. Failing, as had other armies, to overcome Russian troops and the civilian patriots, the German army's mechanized forces and infantry sustained a major loss of equipment and men to the cold, the Russian winter, and the Russian forces.

An introduction to a chapter on militarily relevant aspects of FCI would seem to permit a viewpoint that troops may fail to avoid cold injury of epidemic proportions due to the destructive forces of nature, encompassing severe lowered temperatures, high winds, deep snow, and extreme cold, all combined with a relentless enemy force. Constant leadership even at the lowest squad level is required to avoid disaster. A major field-rescue problem faces military leaders at every level and all field medical personnel, if the weather; the pursuing enemy; and the lack of food, clothing, and shelter combine to give trouble to an army in retreat.

Avoidance of overwhelming cold disaster obviously favors the winning troops, not those in retreat. Prevention of cold injury is enhanced by forces' being able to stop; establish shelter; provide heat; and dispense food, warm clothing, and proper foot and hand gear. Naturally, a warm army requires leadership and knowledge of cold weather's effects on machines, equipment, and personnel at all levels. Prevention requires outstanding demonstrations of morale, fortitude, and courage for beleaguered troops of all ranks, to prepare for and overcome the lethal developments of cold weather and enemy forces.

Winter weather and its associated freezing temperatures are likely to always be a threat to military forces. Failing their ability to prevent cold injury, it behooves medical and nonmedical personnel alike to understand, under the worst as well as the best circumstances, how to diagnose, treat, and transport the casualty with cold injuries. This is no mean task, as the casualty often has FCI, or immersion injury, or hypothermia with accompanying battle injuries, penetrating or open wounds, or the myriad nonbattle diseases of the local area.

The study and evaluation of hardship and catastrophe, the result of winter wars, may prevent future military disasters and medical complications. Certainly, as in Napoleon's retreat from Russia in the fall of 1812, the destruction of the Grande Armée should be explained so that such a debacle would not be repeated in the future, and the numerous losses to cold by other armies over the centuries be similarly evaluated. For example, Larrey, describing clearly the clinical signs and symptoms of freezing (and what was obviously hypothermia with associated freezing) noted that "men warming near bivouac fires developed early gangrene and those entering warm rooms suddenly died."[1(pp82–83)] We can only hazard a guess that as a frozen extremity usually does not in itself cause "sudden death," it is likely that the casualty was also hypothermic, probably with an electrolyte imbalance and with destruction of muscle as a result of FCI. Any warming then, in an uncontrolled situation without physiological control of electrolytes and acid–base balance, may cause a influx of released potassium into the vasculature from injured muscle cells or cooled tissues, and with the resultant hyperkalemia, the cold-injured soldier would die a victim of cardioplegia.[7,8] In wartime conditions, it will be impossible to carry out the current recommendation: to rewarm cold soldiers in warm water or in tubs or whirlpools. Possibly, if we can restore circulation by spontaneous thawing so as to (1) control the endothelial injury, (2) avoid damage caused by ice-crystal intercellular growth and the subsequent cellular dehydration, and (3) avoid the development of overwhelming thrombosis, we may produce an adequate protocol, particularly for field use. This would obviously require a new form of rapid thawing of the frozen area so that heat can be provided to tissues without tissue destruction.

THE EVOLUTION OF UNDERSTANDING FREEZING COLD INJURY

Advances in General Knowledge From Wartime Experiences

Paton,[9] in an excellent summary of the pathophysiology of frostbite, noted that "frostbite is as old as history itself."[9(p329)] And indeed, as an extensive bibliography demonstrates, particularly during periods of war, cold injury has played a paramount role in the outcome of military operations throughout history. A review of the world's literature on cold injury would indicate the casualties to be literally in the millions, resulting in destruction of tissue, loss of function, neurocirculatory loss, amputation (minor and major), and death.

Wartime experience with cold has advanced our knowledge of cold injury in at least two directions. First, from the varied cold injuries, especially FCI, incurred by massive numbers of troops, much clinical experience resulted, allowing for new, innovative, and comparative treatment regimens. Second, military medical personnel in the field, and clinical and laboratory investigators in research laborato-

ries and hospitals, were given impetus, encouragement, and funding (especially by the Department of Defense and the Office of Naval Research) to provide insight into the etiology, pathophysiology, and treatment of cold problems.

Before World War II and the Korean War, little information regarding the basic physiological response to cold was available to the physiologist or clinician. One reason was the unfortunate fact that in the United States, little clinical or scientific data from Europe, Russia, or Japan were available in translated form. Further, clinicians and physiologists did not as a rule read each others' journals to the extent seen today. Consequently, particularly in the United States, the massive clinical and investigative material from Europe and Asia was little known. For example, until the translated World War II works of Killian (German),[10] Ariev (Russian),[11] and Yoshimura (Japanese)[12] were published in this country, we did not realize that the medical departments of these armies and navies had utilized rapid rewarming for initial thawing for frozen extremities.

The period following World War II and the Korean War found an exciting and rewarding liaison between the clinician in the field and the hospital on the one hand, and the physiologist, biochemist, biophysicist, and cryobiologist on the other. This interchange allowed the clinician to evaluate the results of drug, surgical, and manipulative procedures first performed on laboratory animals, and permitted the physiologist to perform on laboratory animals previously untested surgical or medical procedures that appeared to have promise.[3,13-16]

Highly recommended to the student of cold injury are the texts or monographs summarized below, which form a nucleus of military and civilian research activities. Each explores, reports, and summarizes a concept of pathophysiology and treatment, and a pattern of injury sequelae.

One of history's earliest accounts of an army decimated by cold was that of Xenophon,[17] who in 400 BC led 10,000 Greek soldiers from Sardis to Babylon and back, through the mountains of Armenia, battling the hazards that a retreating and disorganized army faces when pursued by the combined unrelenting foes of severe cold weather and harassing enemy forces. Warmth was obtained by campfire heat; friction massage of the body with greases, oils, and unguents; and preservation of heat by covering up in snow. Amputation and death from exposure was common.

An even more detailed, factual, and vivid description of the effects of cold on a retreating army was related by Larrey,[1] whose memoirs (previously quoted) precisely described FCI and its etiology, including data on general body cooling. Larrey recommended slow rewarming, or delayed warming with ice and snow techniques, and friction massage, all out of favor now although accepted for well over 150 years after his reports. His monograph is replete with the problems of prevention and care involving massive numbers of troops—in an army in which more than 80% of its force perished from cold and cold-related problems. Much can still be gained by a study of this classic. Larrey disapproved of rapid rewarming, and his words discouraged the use of rapid thawing for more than 150 years. He did note the disastrous effect of excessive heat (as has also been documented in 20th-century reports[18]), apparently recognizing that frozen extremities, warmed in close proximity to bivouac fires, sustained a second thermal injury—a burn—with disastrous effect. For the student of FCI, Larrey's monograph is highly recommended.

Monographs of the experience of the military surgeons of modern armies soon appeared following World War II. Whayne and DeBakey,[19] In their official history of the US Army experience in that war,

- pointed out that the lessons of previous wars were poorly understood and often forgotten by military surgeons in World War II;
- reported and discussed the 71,000 cold casualties in the European theater;
- recorded the new syndrome of high-altitude freezing in air crewman (freezing and hypoxia); and
- stated that cold injuries were due to the intensity of combat, wet cold, inadequate clothing, and lack of troop education.

Injuries were to be considered a calculated risk. It was the stated hope of the authors "that if their volume was read well, there would be no need for problems in future wars."[19(pviii)] But as was later found in Korea (1951/52), in the Yom Kippur War in Israel (1973) on the Golan Heights of Syria, and in the Falkland War (1982), weather, enemy action, military demands, and unforeseen events determine the effects of wet and cold.

Killian[10] related the cold problems of the axis forces in World War II with a discussion, still of concern today, of slow as compared to rapid rewarming, the latter being preferred by many German military physicians despite slow-thawing methods being the European dictum. He reminded his readers of van't Hoff's law, which implies

> that under conditions of hypothermia, local or general, metabolic processes are slowed down

> so that oxygen demand of the tissues is reduced, thus prolonged survival of ischemic tissue."[10(p81)]

Much later, Mills described this condition as "being in a metabolic icebox."[7(p58),20(p134),21(p410),22(p10)] He characterized the victim in this condition as in a midlethal state, so that further exposure would result in death, as cooling of vital organs continued without intervention and warming. Warming in this state, however, if not controlled physiologically, would often result in death because of uncorrected acid–base imbalance and electrolyte imbalance, usually acidosis, with hypovolemia and dehydration. Warming of the hypothermic victim, when FCI is also present, may release potassium from increased cell permeability or cell destruction. The often-sudden, high-level hyperkalemia may result in cardioplegia and death. From Russia, Ariev,[11] in a little-known but true classic on cold, reviewed the current European pathophysiology concept, listing the nomenclature for describing cold injury and classification of frostbite, as well as recommending that rapid rewarming be the method of choice in the thawing of FCI. This report was followed by a report from Japan, in which Yoshimura[12] proposed thawing methods similar to those of Killian and Ariev.

A further report of low-temperature investigation, encouraged because of the needs of military surgeons, was Burton and Edholm's classic monograph, *Man in a Cold Environment*.[23] This monograph, sponsored by the Defense Research Board of Canada, originally proposed to review experiences in cold in World War II but was later changed to include all aspects of cold, and is included here as a "must" reference in the field of cold. Viereck[24] edited the proceedings of a US Air Force-sponsored symposium on frostbite in 1964, which brought together clinicians, laboratory investigators, pathologists, and others experienced in cold-related problems. An outstanding textbook edited by Meryman[25] soon followed, which gathered together the works of 18 prominent physiologists, biophysicists, and cryobiologists delving into the theoretical basis of FCI. It is a definitive review and a framework text on the comprehensive background of biological freezing, and it includes studies of the physical and chemical bases of injury in single-cell microorganisms.

In 1975, LeBlanc[26] published another landmark monograph, *Man in the Cold*, unique in that his observations were made primarily on humans rather than laboratory animals. In his section on frostbite, LeBlanc graphically describes the cooling, freezing, and postfreeze states, and reviews data regarding the pathophysiological stages of vascular disturbance, membrane permeability, and postthaw edema formation.

Chronology of Important Investigative Reports and Results

As much of this section consists of a review of our understanding of FCI, it is perhaps pertinent to preface what follows with Meryman's concept of freezing:

> The single most important and fundamental concept in biological freezing is that regardless of the mysterious complexity of the biological matrix, freezing represents nothing more than the removal of pure water from solution and its isolation into biologically inert foreign bodies, the ice crystals.[27(p515)]

With slow freezing, ice-crystal formation is generally confined to the extracellular spaces. Frozen tissue cells may, upon thawing and refreezing, demonstrate uniform intracellular crystallization, with formation of large, destructive crystals of ice. The formation of these large crystals may account for the disastrous freeze-thaw-refreeze injury seen clinically, in which after initial extraction of cellular water, with increased permeability and trauma to cell membranes or endothelial lining of small vessels, a second freeze will affect intracellular supercooled water, resulting in the destruction of the cells.[18] Meryman's concept needs to be borne in mind as the chronology of FCI research is set forth.

The 1930s and 1940s: Supercooling and Tissue Freezing

Love,[28] in his description of freezing phenomena, points out that Koonz and Ramsbottom,[29] working with poultry, proposed in 1939 that tissue initially froze extracellularly. This was so because the freezing point of lymph (equated with extracellular fluid) is higher than that of the cellular fluid. Love concludes that ice first forms outside the cells and is then augmented by intracellular water that diffuses through the cell wall and condenses on the ice surface because of the high osmotic pressure of the extracellular solution, which has been concentrated by freezing. The temperature of the intracellular fluid never falls below its freezing point, because intracellular water is continuously being lost with a corresponding continuous reduction of freezing point.

Karow and Webb[30] further note that pure water cooled below 0°C does not crystallize until a tem-

perature is reached that will permit substances within the water to act as a nucleus for ice formation. The nuclear material may consist of relatively large inclusion bodies, such as colloids; dissolved substances; or it may simply be water molecules clumped together by hydrogen bonds, called microcrystals. Karow and Webb acknowledged Mazur's[18] work, which reported that in slow freezing there is a tendency for water in cells to supercool, as there is a low probability that such a minute volume would contain a nucleation center. At relatively high temperatures (–10°C), extracellular water freezes. As water freezes in the external medium, its vapor pressure drops below that of the still-supercooled intracellular water and thus draws free water from the cells.

Supercooling is defined as the cooling of a substance below the temperature at which a change of state would ordinarily take place, without such a change occurring (eg, the cooling of a liquid below its freezing point without freezing taking place). This results in a *metastable* state, defined as an excited stationary energy state whose lifetime is unusually long. A further example of the state of supercooling is provided by Burton and Edholm reporting on the observations of Sir Thomas Lewis,[31] who states that the freezing point of skin is about –1°C to –2°C, but that supercooling often occurs, during which freezing is not demonstrated until the surface temperature falls to –5°C to –10°C. Lewis further notes that the fat content of the skin affects supercooling. These concepts permit us, then, to define FCI as true tissue freezing that occurs when there is sufficient heat lost in the cooling area to allow ice crystals to form in the extracellular spaces in slow freezing (the usual human freezing event) and the extracellular water.

Blackwood[32] in 1943 and Denny-Brown[33] in 1945 demonstrated microscopic degenerative histologic changes in nerve and muscle in immersion injury as a direct action of cooling. In 1962, Sayen[34] similarly reported the histological changes found after immersion injury and noted that in extending cooling, tissue destruction was more severe and recovery more prolonged, with vacuolization and fragmentation of nerve axons.

Utilizing rabbit ear chambers, Lange[35,36] in 1945, Quintanella[37] in 1947, and Crismon and Fuhrman[38] in 1947 demonstrated that after immediate thawing, ear tissue appears normal, the circulation returns, followed by hyperemia, then massive edema, circulatory slowing, and red cell clumping in the capillaries. Shumacher and colleagues[39–44] investigated the pattern of vascular change and injury following cold insult, and investigated varied drug therapy in an attempt to improve circulation and thawing.

In 1949, Kreyberg[45] entered the controversy as to whether actual freezing of tissue is the lethal event. He considered that low temperatures, whether freezing or not, damage cells and tissues, but that freezing is not as lethal as heat coagulation. He postulated that after the freeze, further tissue damage ensues during the period of thawing, as demonstrated by hyperemia and stasis.

Also in 1949, Scow[46] reviewed the direct effect of cold on tissues of newborn rats, demonstrating remarkable distortion and retardation of growth in limbs and tails. He found cartilage cells to be susceptible to even brief refrigeration and concluded that changes occur as the result of necrosis of cartilage cells that are normally active in skeletal growth. He considered his findings to support the hypothesis that the lethal effects of cold act directly on these cells by altering protein in the cytoplasm and nucleus. In support of these observations, Bigelow[47] in 1963 and Hakstian[48] in 1972 reported that cold can destroy the cartilage of the epiphyseal plate, which may result in digital shortening or angulation deformity of the digits, or joint dysfunction. Lytic destruction of periarticular cartilage and bone have been reported in adults and children.[8]

The 1950s: Extracellular Ice Crystals and Vascular Stasis

In 1951, Lewis[49] similarly demonstrated that degenerative changes in muscle occur almost immediately after exposure to freezing. Later, in 1953, Lewis and Moe[50] studied the effects of rutin, a flavonol glucoside, and Hydergine (ergoloid mesylates; manufactured by Sandoz Pharmaceuticals, East Hanover, NJ), a dehydrogenated ergot vasodilator, to determine their effects on experimental cold injury. Shumacher[42] had also studied the effects of rutin in 1951, demonstrating more beneficial effects in reducing gangrene than had Lewis. Of interest is that both rutin and Hydergine are now considered efficacious in overcoming the effect of free radical injury, which is a likely cause of cell damage after thawing and reperfusion of the vascular system.[51]

In keeping with the military interest in cold injury, Orr[52] reported in 1953 on findings of his cold injury research teams in Japan and Korea during the Korean War. One member of that team, H. T. Meryman (Figure 14-2), a US Navy medical officer, was to go on to become one of the world's leading investigators in cold-related science and cryobiol-

Fig. 14-2. H. T. Meryman, MD, an early proponent of rapid rewarming in warm water for frozen extremities.

ogy. His contributions include the description of the role of extracellular ice formation and cell damage, the effect of extracellular solute concentration, and the methods of ice-crystal nucleation. During the years 1955 through 1957, he presented a lucid explanation of the effect of the rate of freezing (slow or rapid) on cellular biological systems.[25,27,53] His work includes the development of injury classification, the postulation of a time–temperature relation in clinical cold injury, and the presentation of a pattern of ice-crystal formation with the resulting biochemical effects. Meryman summarized the knowledge to that date on the mechanism of slow freezing injury in tissue, namely, extracellular ice crystals, which displace and partially dehydrate soft-tissue cells. While some tissues may be injured or destroyed by this process alone, most tissues passively collapse without significant mechanical injury. However, the removal of water results in high concentrations of electrolytes and other cell constituents, which produce a cumulative injury to the cell. As the temperature falls from the freezing point to between –10°C and –15°C, more water is frozen out, increasing the solute concentration and the potential for injury.

Bellman and Adams-Ray[54] in 1956 and Sullivan and Towle[55] in 1957 investigated vascular response to cold, finding that cold trauma involving rapid freezing, then rapid thawing, injured the tissue less than slow thawing. Other studies[46,56–58] indicated that an increase in the rate of blood flow occurred during the postthaw stage, accompanied by the emergence of many platelet emboli from the area of injury. Stasis was found to begin in the venules, spreading throughout the vascular bed. Hemoconcentration was considered the cause of stasis following local cold injury.

The 1960s: Intracellular Ice and Freeze-Thaw-Refreeze Injury

In 1960, Mills, Whaley, and Fish[3] proposed a total-care system approach, including the avoidance of trauma to the frozen extremity, rapid rewarming in warm water (preferably in a whirlpool bath) at 42°C to 48°C (later changed to 38°C to 42°C). Postthaw whirlpool at 32°C to 37°C was utilized to massage tissue, promote circulation, and dilute the superficial accumulation of bacteria and thus discourage infection. Isotope and enzyme studies were utilized to determine circulatory status, and for early diagnosis of extent of injury. Recognition of the freeze-thaw-refreeze injury was noted, the latter quite possibly a result of intracellular ice formation as a result of the second freeze, and therefore, usually lethal to the affected tissue. The authors also suggested that the time-honored description of first-, second-, third-, and fourth-degree frostbite be changed to a more descriptive clinical diagnosis of *superficial* and *deep* (discussed later).

In 1964, one of cryobiology's pioneers, Luyet,[59,60] published, with his colleagues, observations on the invasion of living tissue by ice, listing three stages of invasion: (1) superficial freezing, (2) intercellular freezing (extracellular spaces), and (3) intracellular freezing. He demonstrated shrinkage of erythrocytes by osmotic differential in extracellular freezing. Also in 1964, Mundth[56] demonstrated platelet clumping soon after thawing, arising from injured endothelium of vessel walls followed by corpuscular aggregation that eventually became occlusive. Mundth recognized that local tissue injury from freezing was associated with local vascular damage after thawing, involving increased endothelial permeability, intravascular cellular aggregation, capillary stasis, occlusion of small vessels by cellular aggregates, and thrombosis. He demonstrated that low molecular weight dextran (MW 41,000), given intravenously prior to freezing, improved tissue survival after freezing by improving capillary

flow and inhibiting corpuscular aggregation.[57] This work was corroborated in 1965 by Anderson and Hardenbergh,[61] but only when test animals were rapidly thawed after freezing.

In 1965, Karow and Webb presented their "Theory for Injury and Survival."[30] They assumed that bound water, in the form of lattices, was essential to cell integrity, especially protein structure and function. Tissue death in freezing seemed to occur primarily as a result of the extraction of bound water from vital cellular structures. This extracted water, incorporated into growing ice crystals, left proteins dehydrated and denatured. Their explanation of why extracellular freezing initially began was that water cooled below 0°C does not crystallize until a temperature is reached that will permit the utilization of substances within the water to act as a center or nucleus for ice formation. That nuclear material may be relatively large inclusion bodies, such as colloids, it may be dissolved substances, or it may be simply water molecules clumped together by hydrogen bonds, the microcrystals.

In 1969, Hanson and Goldman[62] reviewed the etiology of cold injury, with particular reference to injuries of World War I, World War II, and the Korean War, deciding that after review of all data, prediction of the incidence of cold injury was almost impossible. In 1969, Hardenbergh and Ramsbottom[15] confirmed the findings of Mills and colleagues[3–5] that "double freeze" injury (ie, freeze–thaw–refreeze) was indeed more clinically significant, causing much more harm than a single freeze.

In 1969, Knize and colleagues[63] proposed a system for clinical prognosis for tissue loss after frostbite, based on duration and condition of exposure and the lowest temperature reached. In similar fashion, in 1970 Sumner and colleagues[64] developed a prognostic sign based on experiments with dogs that indicated that blood flow in the involved extremity 24 hours after freezing had prognostic significance. Also in 1970, Sumner and a different group of colleagues[65] used xenon 133 to predict the extent of tissue loss in frostbite as early as 10 minutes postthaw. Mills[3-5] and Salini and colleagues[66] in 1986 reported similar prognostic use of technetium 99m pyrophosphate.

The 1970s: Hyperosmolality and Membrane Damage

In 1971, Meryman[16] proposed that hypertonic alteration of cell membranes is preceded by a stress that increases as osmolality is increased. His contention is that reduction in cell volume leads to membrane injury.

Meryman considered salt concentration as a cause of injury when salt denaturation of membrane components occurs. In 1974, he reported[67] that the primary site of cell freezing injury is the cell plasma membrane, which includes membrane permeability alterations, along with the effect of elevated extracellular osmolality, which results in loss of cell water and cell volume reduction.

In 1970, Mazur[18] reviewed the responses of living cells to ice formation and considered that although the freezing point of cytoplasm is usually above –10°C, cells generally remain unfrozen and therefore supercooled to –10°C or –15°C even when ice is present in the external medium. This indicated that the cell membrane can prevent the growth of external ice in the supercooled interior and further suggested that cells neither are, nor contain, effective nucleators of supercooled water. Mazur believed that to understand the solution effect as a mechanism of cell damage, one needs to consider that four discrete events occur during freezing: (1) water is removed as ice, (2) solutes of high and low molecular weight concentrate, (3) cell volume decreases, and (4) solutes precipitate. Contrary to the theories of Lovelock and Meryman,[68] Mazur considered the cause of injury from extracellular ice to be that the ice exerts sufficient force to rupture not only the plasma membranes but also the membranes of organelles such as mitochondria. His rationale for this suggestion is that recrystallizing ice crystals can disrupt protein gels, and that cells killed by intracellular freezing have suffered membrane damage and become leaky.

In 1971, Carpenter and colleagues[69] demonstrated the beneficial effects of rapid rewarming at 42°C, reporting that endothelial cells remain attached to the arterial intima, with the internal elastic lamina remaining intact and the surrounding media appearing less distorted. During slow thawing, the endothelial cells are almost completely shed into the vascular lumen, the internal elastic membrane is disrupted, and the cells of the media are distorted and necrotic.

In 1972, Molnar and colleagues[70] attempted an analysis of the events leading to freezing, using finger temperatures. They concluded that the incidence of either freezing or cold-induced vasodilation could not be correlated with the relative cooling rate because of indeterminate supercooling. These authors concluded that the factors that induced both crystallization and vasodilation remain to be discovered.

In 1973, Bowers and colleagues,[71] investigating in vivo freezing, viewed ultrastructural changes occurring in capillary endothelium by electron microscopy.

He discovered no precipitous changes in muscle cell mitochondria or capillary endothelium as a result of hypoxia after cooling tissues to 2°C or supercooling to –13°C. However, reducing the temperature by one Centigrade degree per minute until freezing occurred, and continuing to cool for 10 minutes, followed by rapid rewarming, resulted in consistent mitochondrial damage in muscle cells. There was also marked degeneration of associated capillaries.

The 1980s: Vasoactive Metabolites and Microvascular Dysfunction

In 1980, Vanore and colleagues,[72] followed by Purdue and Hunt[73] in 1986 and Britt and colleagues[74] in 1991, published well-detailed summaries of the events leading to the tissue effects of cold injury. The events following cold insult were categorized using the direct effect of the cooling and cold period, the freezing period, and the immediate and delayed postthaw periods. Their reviews are comprehensive and rich in the pertinent bibliography of cold injury; their method of event analysis is utilized in the Pictorial Atlas of Freezing Cold Injuries at the end of this textbook.

Beginning in 1981, Robson and Heggers[75] published observations on metabolites of arachidonic acid (the eicosanoids dinoprost [$PGF_{2\alpha}$] and thromboxane [TXB_2]) in frostbite blister fluid. These authors suggested these two vasoactive metabolites as a cause of dermal ischemia and a possible cause of the progressive vascular changes that are seen in cold injury. Based on these data, the authors developed a "rational approach" to treatment of frostbite based on the pathophysiology of freezing injury. Having demonstrated the breakdown products of arachidonic acid, they used antiprostaglandin agents and thromboxane inhibitors to preserve the dermal microcirculation.

In 1989, utilizing light and electron microscopy, Marzella and colleagues[58] studied morphologic changes in the vascular endothelium of the skin. They concluded that the endothelial cell is the initial target of the injury induced by freezing, and further, that the injury is mediated by a non–free radical mechanism. They stated that "by now it is generally agreed that direct thermal injury alone is not sufficient to cause cell death,"[58(p67)] and also suggested that the initial freezing impairs microvascular function, leading to edema, stasis, thrombosis, and finally, to ischemic necrosis. This may be followed by the production of arachidonic acid metabolites after thawing. These physiologically active substances result in inflammatory responses that modulate vascular contraction and permeability, platelet aggregation and recruitment, and activation of leukocytes. Marzella and colleagues introduced a "new player" into the game, indicating the participation of free radicals in the induction of tissue damage. They pointed out that the consideration of free radical injury has been suggested by evidence showing that there are at least three approaches for preventing free radical injury if given at the time of thawing: (1) use of the enzyme superoxide dismutase, which destroys the superoxide anion radical; (2) use of a radical scavenger, which reacts with free radicals and converts them into less-destructive moieties; and (3) use of substances such as chelators of free iron, which prevent the generation of free radicals in the first place.

In their studies with experimental rabbits, however, Marzella and colleagues[58] found that freezing caused an immediate separation of endothelial cells from the internal elastic lamina. They believed that the separation was present even in samples removed immediately after freezing and before skin thawing, so that reperfusion could not be considered responsible for that lesion. Separation of endothelial cell junctions was seen in venules and capillaries soon after freezing. They also suggested that other inflammatory mediators released after injury, such as leukotrienes, may have contributed to separation of cell junctions.

The 1990s: Oxygen-Derived Free Radicals and Reperfusion Injury

During the 1980s and 1990s, research interest focused on the formation of oxygen-derived free radicals as a cause of tissue injury in both freezing and nonfreezing conditions. This reaction has been noted in postoperative organ warming, as after cardiac surgery, suggesting that free radical formation may follow rewarming and reperfusion of cooling or cold or frozen parts.[76,77] Investigation of irreversible tissue damage in cold injury was considered to be related to oxidation of protein sulfhydryl groups, and that the oxidation process probably involved hydroxyl radicals (OH•). Miller and Cornwell[78] suggested that removal of the hydroxyl radical by a scavenger would add cryoprotection to cell membranes. In this regard it is noted further that the classic cryoprotective agents, dimethyl sulfoxide (DMSO) and glycerol, are hydroxyl radical scavengers.

Studies have investigated reperfusion injury and involvement of the oxygen-derived free radicals (and also activated neutrophils), which are incrimi-

nated in endothelial injury during reperfusion.[79] Because various methods of warming are accepted treatment of hypothermia and of both nonfreezing and freezing peripheral cold injury, this form of reperfusion must be examined for free radical formation. Laboratory and clinical studies[7,58,71,80–82] regarding cold problems have brought our attention to a somewhat overlooked area of organ anatomy subject to cold insult–and one that may assume an area of great importance. The new investigations have demonstrated the impact of cold on the vascular intima, specifically the endothelial cell.

The importance of the endothelial cell structure is realized when we recall that the entire circulatory system is lined with a single layer of endothelium. The normal endothelial cell wall forms a tight, smooth surface on the luminal side of the vessel and serves as a semipermeable membrane for the interchange of material between blood and tissues. The intact wall prevents the larger elements of the circulating medium from leaving the lumen of the vessels. Any alteration of the endothelium affects flow patterns; changes adsorption capability; and causes release of substances that influence platelet and fibrin deposition, and therefore enhance clot formation and capillary blockage–the latter factor causing the vascular ischemia and occlusion that may cause severe, often irreversible, tissue damage after freezing.

Important to the endothelial cell response to cooling are the surface cell receptors that respond, for example, to interleukin 1 and other factors that are said to constitute the prime starting signals for the inflammatory response. Endothelial cells contribute to the manufacture of plasminogen activator and inhibitor, prostaglandins, reactive oxidant, and cytotoxic proteases. Cooke and Theilmeier[82] in 1996 reviewed the importance of this fragile lining in the formation of vasodilating mediators such as prostacyclin and nitric oxide (NO; the substance was originally called endothelium-derived relaxing factor [EDRF] but is now recognized to be NO). The most potent endogenous vasodilator known, NO is released when circulating thrombin is present or when serotonin is released by aggregating platelets. Such triggered NO induces vasodilation and inhibits further growth of platelet thrombi. There are also receptors in the endothelial wall for agonists, so that when their effect is to cause NO to be released, potent vasoconstriction may occur. As a result, normal endothelium tends to maintain vascular potency by inhibiting platelet adherence and aggregation, attenuating the response to vasoconstrictors. When the endothelium is injured (as in cold insult) or vascular trauma occurs, this protective influence of endothelium is lost, and as a result, vasoconstriction and platelet aggregation occur, allowing hemostasis. Further, NO inhibits platelet adherence and aggregation. NO and endothelial-produced protocyclin confer resistance to platelet–vessel wall interaction, and NO also inhibits the adherence of leukocytes to endothelium. Cooke and Theilmeier further reviewed the evidence that when reperfusion is associated with further injury to the myocardium, in part due to adherence and infiltration of neutrophils and concomitant release of oxygen-derived free radicals, this phenomenon may be inhibited using perfusion with sodium nitrate or other exogenous NO donors.

The endothelial cell plays an important role in vascular wall defense by hemostasis and the removal of thrombin and vasoactive substances from circulating blood. Thus the cell's importance as an organ that is especially susceptible to cooling and freezing cannot be neglected. Freezing causes immediate alteration of the vascular intima, especially the endothelial cell, as well as causing separation of the endothelial cell layer from the basement membrane.[83] This separation may result in overwhelming platelet aggregation (further contributing to vascular injury), luminal occlusion, ischemia, and eventually gangrene. If the target cell of severe cooling and freezing insult is the endothelial cell, then its early protection, preservation, and early reendothelialization is a matter of the highest priority for future laboratory and clinical research.

In 1990, Vedder and colleagues,[84] studying reperfusion injury using the rabbit ear, determined that tissue injury from ischemia and reperfusion formed the basis of several important disorders, including circulatory shock. Using the rabbit model, and monoclonal antibody (MAb) 60.3 directed to cluster of differentiation 18 (CD18, the human leukocyte in adherence glycoprotein), Vedder noted that intravascular neutrophil aggregation and neutrophil adherence to endothelium were blocked in his rabbit model of forehead tissue ischemia and reperfusion. Antibody treatment–before and after ischemia but prior to reperfusion–resulted in the same degree of significant protection against endothelial, microvascular, and tissue injury. From this finding, Vedder and colleagues concluded that under these circumstances, injury is primarily a result of reperfusion. This eventually may be important to us, as the frostbite injury, warmed after freezing insult, is primarily a problem of reperfusion.

In 1993, Mileski and colleagues[85] tested the hypothesis that blocking neutrophil adherence or aggregation or both reduced the tissue injury that re-

sults when tissue is frozen and rewarmed. Using the rabbit model, the extremities were frozen and then rewarmed. The test rabbits were then treated with MAb 60.3, thereby blocking adherence and aggregation. Tissue edema and tissue loss were less in the MAb-treated rabbits, supporting the view that a substantial component of severe cold injury is neutrophil mediated and occurs after rewarming.

In 1994, Buckey, Vedder, and colleagues,[86] working on another thermal injury, burns, recognized that monoclonal antibodies directed to the β_2-integrin adherence receptor family on leukocytes have shown significant tissue preservation in myocardial, intestine, rabbit ear, and hypovolemic shock models of ischemia of reperfusion injury. They noted that neutrophils can exert their damaging effect via several mechanisms. After being exposed to inflammatory mediators, neutrophils become activated and adhere to endothelium. This adherence allows the establishment of a microenvironment that is protected from regular plasma inhibitors. The adherent neutrophils then release cytotoxic phospholipase products, granule constituents, and toxic oxygen metabolites, resulting in endothelial injury and leading to increased neutrophil infiltration, microvascular occlusion, and tissue destruction. They state further, of interest in cold trauma, that oxygen-derived free radicals are known to be involved in neutrophil attraction and activation. Neutrophils are not only attracted by products of oxygen-derived free radicals but are themselves an important source of the oxygen-derived radical species that cause direct tissue destruction and may further amplify tissue damage. As in the foregoing studies, the use of MAb 60.3 in rabbits demonstrated less tissue edema and thinner eschar, and the researchers believed that in burns (as in cold thermal injury), the moderate burn injury may be significantly attenuated by blocking neutrophil adherence functions with CD18 and MAb.

Summary of the Pathogenesis of Freezing Cold Injury

The pathophysiological changes in FCI occur in two phases: (1) those occurring in and induced by the cooling, supercooling, and freezing stage, and (2) those occurring during the thawing (rewarming) and postthaw stage (Exhibit 14-2).

EXHIBIT 14-2

PATHOPHYSIOLOGICAL CHANGES DURING FREEZING, THAW, AND POSTTHAW

First, the following changes occur in and are induced by the freezing state:

- structural damage by ice crystal growth;
- protein denaturation;
- pH changes (intracellular and extracellular);
- dehydration within the cells as a result of extracellular ice formation and extraction of cellular water;
- endothelial cell disruption;
- loss of protein-bound water;
- rupture of cell membranes;
- abnormal cell-wall permeability;
- destruction of essential enzymes;
- ultrastructural damage to the capillaries; and
- consistent mitochondrial damage in muscle cells.

Second, the following changes occur during the thaw and postthaw stages:

- circulatory stasis;
- corpuscular aggregation;
- piling of red cells back to the capillary bed;
- development of hyaline plugs in the vascular tree;
- marked tissue edema;
- anoxia–ischemia of tissues;
- increase of compartment space pressure;
- capillary and peripheral vessel collapse, with endothelial cell disruption; and
- eventually, thrombosis of vessels, ischemia, regional necrosis, and tissue death if the process is not reversed.

The Cooling, Supercooling, and Freezing Stage

With exposure to cold, there is an early tissue response to cooling. This is described as a cold-induced vasoconstriction and is followed by a cold-induced vasodilation, also called the "hunting response."[87] Another hypothesis states that at the same time, or as cooling continues, arteriovenous anastomoses open, with shunting of blood away from the periphery of the vascular bed.[88] Soon, after sufficient heat loss occurs to allow freezing, ice crystals form in the extracellular fluid spaces, with extracellular freezing.[53] This event is precipitated by inclusion bodies and microcrystals. It is possible that some structural damage may result from continued ice-crystal growth.[30] Extracellular osmotic pressure increases,[89] resulting in cell volume reduction and solute concentration in the extracellular spaces and interstitium.

As freezing continues, there is an elevated concentration of electrolytes, protein denaturation, intercellular and extracellular pH changes, intercellular and extracellular dehydration, freezing of extracellular water, loss of protein-bound water in the cells, and destruction of essential enzymes.[18] As further cooling and freezing continue, cell membrane damage occurs, with impairment of microvascular function and increased cell wall permeability, and with critical endothelial cell injury and endothelial separation from the internal elastic lamina of the arterial wall.[18,58] At this time, severe injury to chondrocytes may occur because cartilage, particularly epiphyseal cartilage, is susceptible to freezing damage. Further insult causes ultrastructural capillary damage, mitochondrial loss in muscle cells, and injury to other intracellular structures.[49]

The Thawing (Rewarming) and Postthaw Stage

Depending on the method of thawing, postthaw hyperemia, ischemia, cyanosis, or even total circulatory failure usually develop.[37,80,90] Proximal blebs, distal blebs, or no blebs appear. The usual event is that of vasodilation, edema, and stasis.[43,67] Corpuscular aggregation begins with thawing, often associated with progressive ischemia or with hyaline plugs in the vascular tree.[56,58,80] Occasionally, because of associated or combined injury, increased pressures may develop in soft-tissue compartments.[6] Changes related to reperfusion injury occur, with formation of oxygen-derived free radicals, neutrophil activation, and other inflammatory events.[81] An early response in the thawing stage, and perhaps in cooling too, is the arachidonic acid cascade, liberating prostaglandins and thromboxane, and predisposing to vascular clotting.[75,91] Proteolytic enzymes are produced, with increased membrane permeability.

Eventually, (*a*) vascular reconstitution and clot dissolution begin or (*b*) capillary and peripheral vessel collapse occurs, followed by microvascular and macrovascular thrombosis, venule and arterial obstruction by thrombosis, tissue ischemia, necrosis, and gangrene, resulting in loss of the affected part or area. Following thawing, should refreezing occur, intracellular ice formation is most probable, resulting in the destruction of cells and vital organs. Thawing in this usually deep injury results in unrelieved thrombosis, stasis, failure of cell repair, and loss of body parts usually near or at the level of the second freeze.[7,15,27]

DESCRIPTION OF THE COLD-INJURED PART

In general, the cold-injured or frozen body part may be described as in two states, frozen or thawed. In the frozen state the part is hard, cold, and usually white and apparently bloodless, but is occasionally pale yellow. The part frozen often represents the condition of the extremity at the time of the injury (eg, one that was partially cyanotic would remain so, with a purplish hue in the frozen state). The digits are usually rigid, obviously anesthetic, and generally immobile at the interphalangeal joints and often at the metacarpal- or metatarsal-phalangeal junction (although as a mass the fingers or toes might move, owing to the function of the long flexor or extensor tendons). The surface appears solidly frozen, as do the total digits, even in areas where freezing may actually be superficial. The thawing is usually painful, particularly when accomplished rapidly. Delay in thawing is associated with less pain and may account for the popularity of ice, snow, or ice water as a thawing medium in many areas where self-care is practiced. The area frozen, as well as being hard, insensitive, and cold or white, is occasionally covered with frost particles and, occasionally, has a yellowish cast or areas that are obviously avascular, depending on skin pigment. The outer shell of the skin (epidermis and dermis) is rigid and pulses are absent. Pain generally is nonexistent in that area.

In the thawed state, the clinical pattern often depends on (*a*) the method of thawing, (*b*) the duration of freezing, and (*c*) the depth of injury. Following thawing the part becomes flushed, often with

an ominous purple hue, particularly if extremes of temperature are used. With rapid rewarming, the part is usually flushed pink or red and occasionally a violaceous hue. The thawed extremity is usually edematous with large, serum-filled blisters (ie, blebs) developing an hour to several days following thawing. The formation of blebs often gives an indication of the severity of the injury and perhaps is a result of the thawing method. If the blebs are small, dark, or hemorrhagic, and are present above the interphalangeal joints, the prognosis is generally poor. On the other hand, if the blebs are clear or yellowish, even pink-tinged, and extend clear to the tips of the digits, that usually is an indication of an adequate response to thawing and good prognosis. It may also indicate a lesser depth of freezing than much more proximal blebs. Unless accidentally broken, the blebs will remain intact until the 4th to the 10th day, when resorption of fluid begins and spontaneous rupture of the blebs may occur. As these blebs dry, a hard eschar develops throughout and circumferentially on the injured surface. This eschar may be quite black, giving a false impression of deep gangrene. However, within 14 to 21 days, the eschar begins to separate spontaneously, revealing delicate but healthy tissue below. This eschar, once formed, should be carefully incised and split down to newly forming epithelial tissue so that joint motion is permitted, and also exposing the underlying, newly epithelializing tissues for massage and whirlpool therapy.

Patients have generally described initial feelings of cold discomfort in the extremity, then often loss of pain or discomfort, followed by a sensation of tingling or numbness, then followed by a complete loss of all sensation, including pain. It is presumed that at this point the tissues are frozen. Anesthesia lasts until thawing occurs. Some individuals describe walking on a numb foot, or feeling almost as if they had a "wooden leg." On the other hand, it is not uncommon for those who are inured to the cold, particularly American Indian and Eskimo individuals, to have had none of the warning signs usually demonstrated by black or white people and not be entirely aware of the frozen state, particularly of the toes, until they are changing their boots or mukluks. Again, because of the anesthetic nature of all cold injuries, patients often say that they were unaware that they were developing FCI.

For additional information about and illustrations of the cold-injured part, interested readers can consult the Pictorial Atlas of Freezing Cold Injury at the end of this textbook.

Traditional Classification of Injury (by Numerical Degree)

Because some physicians and many manuals still describe FCI by the degree of injury, it is included and described here for the sake of completeness; another classification method follows.

First Degree

First-degree injury is considered to be very superficial freezing, usually of short duration, with few residual findings and only occasional blisters, if any. In first-degree injuries, erythema and edema, along with transient tingling or burning, are early manifestations. The skin becomes mottled blue-gray and red, and hot and dry. Swelling begins within 2 to 3 hours and persists for 10 days or more, depending on the seriousness of the injury. Desquamation of the superficial epithelium begins in 5 to 10 days and continues for as long as a month, but no deep tissue is lost. Paresthesias, aching, and necrosis of the pressure points of the foot are common sequelae. Increased sensitivity to cold and hyperhidrosis may appear, especially with repeated first-degree injuries. It should be noted that it is difficult to differentiate first-degree frostbite from the abrasion produced by the insulated vapor barrier boot; medical personnel must be cognizant of the difference, as both injuries occur in the same clinical setting.

Second Degree

Second-degree injury is considered true freezing, demonstrated by pallor of the skin, very little pain, and loss of sensation with freezing of the skin and subcutaneous tissues. Second-degree FCI begins as does first-degree, but progresses to blister formation, anesthesia, and deep color change. Edema may form but it disappears within days. It should be noted that if the part is hard, cold, and white, it is very difficult to determine the degree of injury; because the part is frozen, the segment is immobile and usually without joint motion; only after thawing may further change be identified. Vesicles appear within 12 to 24 hours. They generally appear on the dorsum of the extremities, and when these vesicles dry, they form an eschar. Blisters are a good clinical sign as long as they are filled with clear fluid. If the fluid is hemorrhagic, the prognosis is often poor. As these vesicles dry, they slough cleanly with pink granulation tissue demonstrated beneath, or they may form a cover of black eschar involving

primarily the dermis and epidermis. Throbbing and aching pain occur 3 to 10 days after this injury. Hyperhidrosis is apparent at the second or third week. Early rupture of the blisters with subsequent infection often occurs in second-degree FCI. This infection, if present, significantly increases the severity of the injury. It is the purpose of the whirlpool treatment (described later in this chapter) to completely wash the fractured blebs and dilute the bacteria if any are present.

Third Degree

Third-degree injury involves full skin thickness and extends into the subcutaneous tissues. Vesicles are smaller and may be hemorrhagic. Generalized edema of the extremity may occur, but it usually abates within 5 to 6 days. Subfascial pressure increases and compartment syndromes are common in third- and fourth-degree FCI. If pressure rises significantly with loss of distal blood flow, then fasciotomy along with vasodilators are indicated for therapy. The skin soon forms a black, hard, dry eschar, usually thicker and more extensive than that of second-degree FCI. When the eschar and area of involvement finally demarcates, sloughing with some ulceration occurs if there is no complicating problem of infection. Patients often complain of burning, aching, throbbing, or shooting pains beginning on the fifth day and usually lasting through 4 to 5 weeks. Hyperhidrosis and cyanosis appear later, and extreme cold sensitivity is a common postinjury sequela (discussed later in this chapter).

Fourth Degree

In fourth-degree injury there is destruction of entire thickness of the part, including bone, resulting in extensive loss of tissue. After rewarming, tissue is cyanotic and insensitive, and blister formation, if present, is hemorrhagic. Severe pain on rewarming, along with deep cyanotic appearance, regularly occurs. In rapidly frozen extremities or the freeze-thaw-refreeze injury, dry gangrene progresses quickly, with mummification occurring as rapidly as 4 to 5 days and up to 7 to 10 days. With slower freeze injury, there is some early swelling and deep pain and demarcation takes much longer to occur. The line of demarcation becomes obvious at 20 to 36 days and extends into the bone in 60 or more days. Because freezing is usually deep, and occurs often over an extended period of time, tissue damage in fourth-degree injury is irreversible, with major necrosis and gangrene, and often associated with severe infection.

Modern Classification of Injury (by Depth)

Because of the difficulty in clinically differentiating the various degrees of FCI, the diagnostic nomenclature that specifies freezing injury by degree needed to be simplified. As a result, Mills, Whaley, and Fish[3–5] proposed the terms "superficial" and "deep" injury instead of the traditional degrees:

- *superficial* injury is defined as FCI limited to the skin, and corresponds to the traditional first- and second-degree injuries.
- *deep* injury is defined as FCI involving tissues beneath the skin, including muscle, tendon, nerve, blood vessel, and bone; it is comparable to the traditional third- and fourth-degree injuries.

It is important to note that under the traditional classification, some of the first- or second-degree injuries, depending on the depth of treatment, could, as a result of abnormal warming, infection, or trauma, suddenly become third- or fourth-degree injuries.

Following a clinical examination of patients with FCI using all the usual means and proper tools, medical officers may find that other diagnostic modalities are useful in diagnosing thermal injury (Exhibit 14-3).

EXHIBIT 14-3

DIAGNOSTIC TOOLS FOR COLD INJURY

- Sensory measurement utilizing sterile needles, or equipment for measuring two-point discrimination and tests for proprioception
- Thermography
- Electromyographic and nerve-conduction studies
- Examination for pulses, by manual and by Doppler examination
- Measurement of tissue compartment pressure
- Examination for carpal and tarsal tunnel pressures
- Examination of tissues by radiographic means, including routine flat-plate anteroposterior roentgenograms, computed axial tomography, magnetic resonance imaging, and radioisotope examination (technetium 99m) for the status, presence, or absence of cellular perfusion
- Arteriography if required

MANAGEMENT OF FREEZING COLD INJURY

For armed forces medical personnel, management of cold-injured service personnel depends on the military situation at the time that cold-injured individuals are seen and rescued or treatment begins.[92] Medical care will also be determined by the type of facilities available for treatment or for transfer elsewhere, from the foxhole, the tent, the battalion aid station, or upward in the echelon system of the military medical services. Exhibit 14-4 contains examples of the kind of questions that medical officers must ask themselves before they begin the first of two categories of medical management of cold-injured casualties: field treatment and care after rescue and evacuation.

Despite all we have listed as causative elements in the sequence of freezing, little has been said of the other factors that are so important in determining the final result in the human. These variables—often difficult to anticipate, measure, or predict—are in the realm of weather, inadvertent accident and trauma, and individual human physiology and anatomy. The state of health and physical condition of the victim are also vital, and include the associated factors of alcohol and drug use and mental state at the time of exposure. The individual's neurovascular integrity in peripheral areas is also a major factor if disease states such as diabetes, arteriosclerosis, vasculitis, labile vasomotor disturbances, and Raynaud's syndrome or Buerger's disease are present.

Associated trauma that precedes freezing, such as extremity strain, sprain, or fracture, pose major problems, as do penetrating wounds, blunt trauma, or blood loss from any cause. FCI is further influenced by the degree of hypovolemia or dehydration present, causing further distal vascular deficiency prior to the onset of freezing. Rescue and survival often result in refreeze injury and perhaps result in the irreparable trauma that occurs at the junction of frozen-nonfrozen tissue, as "brittle" tissue segments are stressed when the victim with FCI must walk to survive a wilderness catastrophe.

Once the victim of any cold injury (eg, immersion NFCI, FCI, hypothermia, separately or in any combination) is evaluated, an effort must be made to avoid further cold exposure. As soon as the patient has been sent to an adequate care area, total physiological control should be attempted—which means monitoring the cardiovascular system, blood gases, electrolytes, and airway care, as in any emergency care.

In the absence of hypothermia, victims of immersion-type NFCI or of true FCI are seldom in a life-threatening condition, unless the condition is associated with hemorrhage from battle trauma, the onset of infection, or the presence of associated trauma to vital areas. With those exceptions, extremity FCI or NFCI rarely results in death unless accompanied by hypothermia, with or without other trauma, or unless—even with hypothermia—total physiological control is not obtained. In the absence of total physiological control of systemic hypothermia or extremity cold injury, death may result from potassium release after warming, causing cardioplegia and cardiac arrest.

At present, as this chapter is written, we are reviewing nearly 1,500 cases of cold injury, all seen by or consulted on by the author. Only one death in more than 1,000 cases of FCI occurred during hospitalization after rescue. That patient, a man older than 80 years of age, died from pulmonary embolism more than 1 month after he was hospitalized after freezing all four extremities. The analysis of case records is not yet complete, but a preliminary review indicates that of 250 cases of hypothermia with associated cold injury, death occurred much more frequently and was related primarily to the depth of hypothermia. When hypothermia and

EXHIBIT 14-4

EXAMPLE QUESTIONS FOR MEDICAL OFFICERS

1. What are the battle conditions in the field? Are your forces in attack mode or retreat? Are they pinned down by enemy fire or able to move freely?
2. From previous troop education, can the injured combatant care for himself, or will he need help from a squad member or leader?
3. For further care of the combatant with frozen lower extremities particularly, is transport care available if necessary by stretcher, ground vehicle, or helicopter to rear areas such as the company medical area or the battalion aid station or even farther?
4. For the casualty with severe injuries, is a helicopter or fixed-wing vehicle available for rapid transport to mobile hospital units, hospital ships, or permanent hospital facilities established elsewhere?

freezing occurred in the same patient, no deaths resulted if the patient was treated by *total physiological control* and warmed either by rapid rewarming in a tub or by peritoneal dialysis for treatment of hypothermia and simultaneous tub treatment of the freezing injury.

Field Care and Prehospital Management

In the field, the military situation may dictate the method of care and transfer or return to duty after the initial examination, especially if it is militarily necessary that the troops remain on line or participate in retreat. If possible, instruction should be instituted concerning the care of hands and feet, change of stockings, and reiteration of all methods of detection and avoidance of cold injury. Weather–continued cold–wet or freezing–may hamper care. No matter the environmental condition, it is expected that if thawing an extremity is proper, then refreezing should not occur. The decision must be made as to whether thawing or warming be done on the spot or after transfer to a rear area. If evacuation is intended, instruction for care during transport should be given. This is especially so to avoid refreezing, and also to avoid further trauma to the part frozen.

Other factors must be considered, including the diagnosis of cold injury, whether freezing or nonfreezing; diagnosis of hypothermia; and the presence of combat injury including gunshot or shrapnel wounds, or open wounds with hemorrhage, which must then take priority for treatment over the cold injury. It becomes necessary, therefore, to practice triage at all echelons. An adequate triage examination must be performed so that other or more-severe medical problems besides cold injury be identified, and so that essential care be given at that time and during rescue.

Some controversy may exist regarding initial and continuing basic battlefield care, because there is no adequate definition between the care rendered in the field, where so little aid is available, compared with the ultrasophistication of modern hospital care, where so much in the way of personnel and equipment is at hand. Whether at the discovery site or in the field, there may be many field-care variables, including

- the rescue experience of the discovery party;
- the number of victims found, and the stress of combat conditions;
- more pertinent, the depth and duration of hypothermia of the victims or the cold injury;
- the associated injuries or medical problems of the victims, including FCI, NFCI, and battle wounds; and
- even more pertinent, the local weather, which may hinder not only the rendering of care but also the care itself.

If hypothermia is present along with other injuries, the victim may have been in that state for quite awhile. The first responder usually has time to view the area, consider the problem, and assess the condition of the victims before rushing forward. Not the least consideration is an evaluation of the discovery site to determine whether rescuers can safely work there, and if not, whether they can take precautions with whatever equipment is at hand to make the area safe. Immediate transport of the casualties may be indicated. Even without a low-reading thermometer, the first responder can make an assessment of the victim's hypothermic state. The onset of hypothermia in the field may be sudden (eg, immersion in cold or icy water) or insidious, or a result of exposure to wind, rain, snow, falling temperatures–all perhaps under the pressure of combat conditions. *Penetrating wounds and hemorrhage may be leading causes of hypo-thermia.*

Not too many years ago, the treatment of the frozen extremity was fraught with controversy. The differences of opinion ranged from varying methods of thawing, a paucity or a plethora of aftercare methods, and debate regarding utilization of drugs of all types and uses, and debate, too, over the need for invasive techniques or surgical procedures. At the least, the approach to care of the casualty with FCI should be purposeful and systematic, utilizing all treatment that is now known to be helpful. Treatment should be offered in an orderly and proper sequence from initial thawing to demonstrable conclusion, with or without immediate sequelae. This sequence may be listed briefly as follows:

1. Avoid further trauma to frozen or, in the event of thawing, injured parts.
2. The trauma to be avoided includes exposure to excessive heat or mechanical trauma, or any factor causing loss of circulation to the involved limb (for instance, in the use of a tourniquet if a compress will suffice).
3. Prevent refreezing of thawed extremities, in the rescue or initial care or transport.
4. Carefully insulate the involved extremities to avoid further injury from helicopter wash, which acts as an increased chill factor, as refreezing under those conditions is likely to occur.

5. Recognize and treat the associated trauma, realizing the need for immediate care for the condition of hypothermia, if present, along with freezing injury.
6. In the process of triage, be vigilant for trauma more severe and life-threatening than extremity freezing. Penetrating trauma, including hemorrhage, must be treated to avoid worsening any circulatory loss to the frozen, now thawed, extremities.

The squad leader or medic or field surgeon should look for and be prepared to treat problems of hypovolemia, dehydration, and electrolyte and acid–base imbalance. The diagnosis of other problems may require selective triage and rear-echelon hospitalization where laboratory facilities are available.

If transport is not available and the soldier with frozen extremities must walk without stretcher or vehicle support–especially if large numbers suffer freezing cold injury–it is important that they ambulate on frozen, nonthawed extremities rather than thawed, which usually results in painful, edematous feet with decreased circulation, which increases the likelihood of refreezing injury. The decision in the field to have troops walk on frozen feet rather than to rewarm them is difficult for troop commanders and medical personnel alike.

Definitive Treatment in a Hospital

It is convenient to describe the definitive care phase in two stages: before thawing and after thawing. The frozen part must be protected to avoid trauma and the risk of irreversible injury at the frozen-nonfrozen interface, which may result if motion occurs at that level, fragmenting partially frozen tissue. This may be beyond the control of the treating physician, if it has been necessary for the injured individual to walk in order to survive. The frozen part should then be thawed using the approaches described below. The thawing is completed when the distal tip of the thawed part flushes. The thawed part should not be massaged.

Postthaw treatment including surgical procedures should be carried out in a hospital, where pain can also be managed if necessary. Many medical inpatient treatment modalities proposed in the past have not proved particularly effective (eg, sympathetic blockade, sympathectomy, anticoagulants, vasodilators, alcohol, and enzymes). This may well be because of the development of thromboses or corpuscular aggregation in vessels that have not permitted an increase in circulation because of a blocked transport system. The same consideration applies to the use of hyperbaric oxygen. In the past, on a single occasion, the use of a single-man hyperbaric oxygen chamber delivering 2 atm pressure appeared beneficial in postthaw frostbite. However, hyperbarism is not likely to be helpful if the oxygen transport system is blocked by vessel thrombosis or destruction of the vascular endothelium. Finally, smoking is forbidden. Alcohol may be given in moderation if requested by the patient.

Prethaw Treatment and Rewarming

In caring for patients with FCI, it is important that a system of care be utilized and that all treatment modes be purposeful. If the extremity is still in the frozen state, it must be thawed. *How* warming is achieved becomes of paramount importance. Some warming methods may be entirely out of the control of medical personnel, as warming, by one means or another, often may have occurred prior to rescue or prior to the patient's being seen by medical personnel. Many thawing methods are utilized by the victims or by helpful rescuers. *Not all are appropriate.* Generally, the victim presents to the rescuers or emergency department and is treated with one of the methods of thawing discussed below, in decreasing order of effectiveness:

1. Rapid rewarming in warm water, 32°C to 41°C, in a tub or whirlpool bath or by means of a crane-lift platform in a Hubbard tank. Rapid rewarming by external means appears to produce the best results but does not always give protection from tissue loss, especially where the injury is deep or of long duration. This method has also been used to warm and thaw the combined injury of hypothermia with extreme freezing.
2. Gradual, spontaneous thawing at room temperature (which varies from 7°C to 32°C owing to cabin heat); or thawing occurring in travel by foot, by vehicle, or during rescue; or thawing due to the warmth of a sleeping bag, often in the wilderness, at altitude, or both. Spontaneous thawing gives variable results, which are often determined by the depth of injury; the duration of freezing; and the patient's activity during survival, rescue, and thawing.

The following two methods are presented for completeness but are not recommended:

3. Delayed thawing using ice and snow, cold water, and friction massage. Warming by cool methods, usually at temperatures near freezing in warm areas, often give poor results.
4. Thawing by excessive heat, such as from a campfire, oven, or engine exhaust (temperatures < 50°C). This method generally results in heat injury (burning) to a part already injured by cold. The final results are disastrous, resulting in great tissue loss and usually major amputation, usually with spontaneous demarcation after the third to the eighth day.

At present, rapid rewarming is favored, this method seeming to demonstrate the greatest tissue preservation and the most adequate early function, especially in deep injury. Results by gradual thawing vary in deep injury, but seem satisfactory in patients with superficial injuries. Ice- and snow-thawing give variable results, most often poor, with marked loss of tissue. The use of excessive heat as a thawing method has resulted in disaster in most cases, especially with dry heat at temperatures of 66°C to 82°C (150°F–180°F), for example, as with the use of a diesel exhaust, wood fire, or stove heat.

An early diagnostic clue as to the exact freezing event is the condition of the digital tips when the patient is first seen. If spatulate mummification occurs within the first 3 to 5 days after thawing, the diagnosis is usually that of a freeze-thaw-refreeze injury, or thawing by excessive heat. Freezing and thawing modes generally seen without the two conditions of excessive heat or refreeze injury usually demonstrate necrotic or avascular changes from the 10th to the 21st day.

It has often been stated that rapid rewarming by internal means (through either arm intravenous or arterial line), administered at temperatures of 37.1°C to 41.1°C (100°F–106°F), is more physiological and may be a method of choice in dissolving ice crystals and restoring cellular hydration. Although this method appears most logical and seems to be a new consideration on the horizon of care, in fact, since the mid 1980s in Alaska and elsewhere, it has been a method of choice in the treatment of combined hypothermia and freezing injury, by both adding heat and restoring fluid volume. The results, however, are still no better than by rapid rewarming. In addition, the development of an arterial line, especially in the areas of ankle and wrist, may cause local arterial spasm and further decrease digital perfusion. The ideal method is obviously not yet at hand, at least for the thawing of the frozen part, but tissue loss is less now than it was in past decades, regardless of thawing method.

Postthaw Treatment

When the injury is severe and deep and hospitalization is required, the extremities are kept on sterile or clean sheets, with cradles over the frostbitten part to avoid trauma and pressure. This is not necessary for the upper extremities, which may be placed on sterile or clean sheets and placed over the chest or trunk. If edema is present or, as required, arms and hands are often elevated in stockinette sleeves to permit drainage and encourage digital motion and decrease dependent edema. The injured part should be protected from maceration with cotton pledgets placed between the digits. In the presence of severe digital edema, however, pledgets or cotton swabs may compress digital vessels, further compromising the circulation. The treatment is open, not occlusive, without the use of wet dressings, unguents, ointments, or petrolatum gauze. Whirlpool baths are utilized twice daily for 20 minutes per bath, at temperatures of 32°C to 35°C (90°F–95°F). In addition, a program of bedside digital exercises of all the joints is initiated and should be done through the entire waking day. Buerger's exercises for the lower extremities are recommended four times daily at least. Biofeedback training can, and has, been utilized to enhance postthaw blood flow to the cold-injured extremity. This technique has demonstrated much benefit, especially in the early stages of injury. Even more interesting is its probable aid to the rifleman in the cold, where biofeedback training might help to avoid vasoconstriction and onset of cold injury.[93]

Surgical soaps, such as hexachlorophene or 4% chlorhexidine gluconate and povidone iodine, are employed in the whirlpool. Occasionally, following the methods of Moyer and colleagues[94] for treatment of burns, 0.5% silver nitrate solution may be lavaged over the area of frostbite. The result is similar to that produced by the surgical soap; with Moyer's solution epithelialization is similar with one outstanding difference: pain is reduced and infection, even superficial, is much less obvious using the silver nitrate solution. The whirlpool clears the debris from the injury and removes superficial bacteria. The tissues are debrided without trauma by the whirlpool action at a time when they are physiologically prepared for the separation of the viable tissue from the overlying eschar.

Occasionally, when severe drying and premature

rupture of blisters have occurred, 1% silver sulfadiazine solution has been utilized on open wounds secondary to freezing injury, usually followed by superficial infection. Blebs are usually left intact because their contents are usually sterile, as is the underlying tissue; they are debrided or trimmed only if they are infected and contain purulent material. Others believe that the bleb fluid should be removed. In the event that aspiration of the fluid is performed, it should be done under sterile conditions.

Over the years, multiple drugs have been recommended for prethaw and postthaw treatment of cold injuries, especially of the extremities. Some of these drugs are listed in Exhibit 14-5, grouped under their principal functions, including plasma volume expanders, vasodilators, hypotensive agents, calcium channel blockers, sympatholytic agents, anticoagulants, thrombolytic enzymes, cryoprotectors, antiinflammatory agents, hyperbaric oxygen, antibiotics, and surgical soaps.

Surgical Procedures

Over the years many surgical procedures have been proposed for postthaw care of the frostbitten extremity. The benefits of surgery should always be weighed against possible injury to the regional vascular structures in the injured area. If possible, the surgical approach should improve the prognosis by relieving compartment pressure, increasing joint mobility, limiting infection, or increasing vascularity. Surgical procedures appropriate to treating FCI are listed in Exhibit 14-6.

Fasciotomy to Relieve Compartment Pressure. Severe FCI, with or without rapid rewarming, often includes signs of increased compartment pressure. Various methods of measuring compartment pressure have been utilized in the past, but the Stryker stick apparatus (295 Intra-Compartmental Pressure Monitor System; manufactured by Stryker Surgical, Kalamazoo, Mich), is one of the favored methods at the present time. Experience has dictated that although capillary pressures greater than 37 to 40 mm of mercury are suspect, we must consider fasciotomy in the presence of clinical evidence of vascular compromise. In 1973 and later, Mills[6] described fasciotomy as a method of relieving the lethal tissue effects of increased compartment space pressure. This mode of decompression was further investigated in 1978 by Franz and colleagues[95] on laboratory dogs, demonstrating its effectiveness.

Several imaging modalities may help supplement clinical judgment and compartment pressure measurement. Most common are arteriography, or the injection of radioisotopes such as technetium 99m that demonstrate the state of cellular perfusion. Isotope studies have been performed as an indicator of cellular perfusion for more than 30 years in Alaska. Doppler ultrasound has also been used as a vascular study tool. Interestingly, at Providence Hospital, Anchorage, Alaska, patients in the Thermal Unit with Doppler evidence of good pulses in the distal extremities (digital vessels) have had conflicting isotope evidence, showing failure of extremity perfusion in the same area. In all cases but one, the isotope study was the accurate one. Evidently, large digital vessels may remain patent for a short while even when the deep capillary system is blocked.

The ultimate decision to do the fasciotomy rests with the attending physician or surgeon, and that judgment often may demand compartmental pressure release regardless of pressure readings. This is important to note, because unless the compartment pressure reading is on a continuous monitor, a near-normal pressure may, in 8 to 24 hours, change to destructive pressure levels and cause severe tissue necrosis, especially in muscle, in a short period of time. If an extremity has remained in a frozen state for some considerable time, even rapid thawing and general supportive care may not be effective in restoring the circulation, and a condition similar to anterior tibial compartment syndrome may be clinically demonstrated. This problem may require fasciotomy.

Failure of sophisticated tools has also been demonstrated in the use of devices for measuring compartment pressure. If one's clinical judgment and experience suggest that an immediate fasciotomy is required, whereas the pressure transducer indicates that the pressure is high but not lethal or indicates a marginal reading, it is often better to trust one's own judgment. A later measurement may indicate sudden pressure increase. A delay in performing the fasciotomy may be disastrous. This diagnostic problem may be avoided by the use of continuous pressure monitoring. However, there are many pitfalls. The monitoring device is still only a machine, and if one's own studied opinion is that the fasciotomy should be performed, then it may be best to rely on one's own surgical judgment and release the pressure. An example of a situation in which even the best clinical judgment in conjunction pressure measurements is inadequate to make the correct diagnosis is the individual with what appears to be a straightforward, superficial, or deep freezing injury who, after rapid rewarming in warm water at the recommended or even lower tempera-

EXHIBIT 14-5

THERAPEUTIC DRUGS USED TO TREAT FREEZING COLD INJURY

- ***Plasma Volume Expanders***
  Low molecular weight dextran (dextran 40), when given intravenously, has a major effect of plasma volume expansion. Dextran 40 also enhances blood flow through correction of hypovolemia (often present in freezing injury owing to marked peripheral vasoconstriction of the arterial capillary tree) and improves microcirculation. It is considered to diminish or reverse erythrocyte aggregation. A good beginning dose may be 25 mL/h given by intravenous infusion or pump.

- ***Vasodilating Agents***
  Tolazoline hydrochloride (Priscoline hydrochloride*) and isoxsuprine hydrochloride (Vasodilan†) presumably function by relaxing vascular smooth muscle. Priscoline is administered intravenously and Vasodilan is administered orally. Generally the dose of Vasodilan is 10 to 20 mg, 3 or 4 times daily. Priscoline is also given subcutaneously at a dose of 10 to 50 mg/d, 4 times daily, beginning with low doses.

- ***Hemorheological Agents***
  Pentoxifylline (Trental*) is said to increase vascular blood flow in patients with peripheral vascular disease by correcting pathologically altered platelet reactivity. It is administered orally or intravenously. The drug exhibits an inhibitory effect on platelet aggregation and on disseminated intravascular coagulation. Males usually are given oral doses of 400 mg three times daily.

- ***Hypotensive Agents***
  - Guanethidine monosulfate is said to produce a selective block of efferent peripheral sympathetic pathways, and is given orally, initially at a dose of 10 mg/d.
  - Reserpine, an ester alkaloid from certain *Rauwolfia* (plant) species that causes sympathetic inhibition resulting in vasodilation and increase cutaneous flow with flushing. It is given orally, 0.05–0.1 mg initially and then 0.1 mg every other day.

- ***Calcium Channel–Blocking Agents***
  Nifedipine (Procardia*) presumably functions by inhibiting calcium ion influx. Nifedipine inhibits the contractile processes of cardiac and vascular smooth muscle, thereby dilating the main coronary and systemic arteries. It is given orally, 30–60 mg once daily.

- ***Sympatholytic Agents***
  Phenoxybenzamine hydrochloride (Dibenzyline*) blocks a-adrenergic receptors. The drug acts on vascular smooth muscle to block epinephrine-induced vasoconstriction and induces peripheral vasodilation. Dosage initially is 10 mg twice daily, increased to 20 mg twice daily after 24–48 h. The hypotensive effects require increased intravenous or oral fluid intake. The drug is effective as a "medical sympathectomy." Dibenzyline and low molecular weight dextran are among the most effective postthaw drugs in the drug armamentarium.

- ***Anticoagulating Agents***
  Heparin was used often in past decades, particularly for its antithrombotic effects. There is no substantial evidence of its effectiveness in freezing injury as compared with other drugs.

- ***Thrombolytic Enzymes***
  Streptokinase, urokinase, and tissue plasminogen activator. Although great promise has been held out for all three enzymes, at present, patients with intracranial or intraspinal injury, or other trauma that would permit increase of local or systemic bleeding, cannot receive them. Using these drugs in patients whose cold injury has caused endothelial or capillary wall injury may cause further local bleeding and increased compartment pressure. Ongoing research[1–3] involving these drugs in selected patients and centers may eventually determine an adequate, safe protocol for their use.

(**Exhibit 14-5** *continues*)

Exhibit 14-5 *continued*

- ***Industrial Solvent***
  Dimethyl sulfoxide (DMSO) has long been used by cryobiologists and animal researchers. It is a cryoprotective agent, a penetrating agent that may someday hold promise in bringing needed drugs to areas via the skin, which cannot be readily reached otherwise because of small vessel or capillary thrombosis. It is said to have vasodilating properties. Its use has been frowned on by the Food and Drug Administration, although DMSO is available in some states by prescription, and it has been used to a great extent in veterinary medicine.

- ***Antiinflammatory Agents***
  Acetylsalicylic acid and ibuprofen, both nonsteroidal antiinflammatory drugs (NSAIDs), are recommended because of their ability to interfere with the arachidonic acid cascade in trauma including freezing cold injury by inhibiting the formation of prostaglandin and thromboxane, thereby aiding and avoiding intraluminal clot formation in small vessels.

- ***Hyperbaric Oxygen***
  In a single case, the use of hyperbaric oxygen in a single-man unit (Union Carbide type), at 2 atm twice daily, was believed to be helpful in a patient with bilateral severe injury of hands and feet. The problem arises if the blood transport system, arteries, and venous channels are destroyed or plugged with intraluminal clots so that oxygen cannot be brought to the cellular level by an adequate blood flow. Then the oxygen, regardless of the atmospheres applied, will be of little help. The absorption of oxygen in this fashion, despite the blocking by thrombus or by aggregated corpuscles, is made even more difficult if the endothelial lining of the vascular tree is severely traumatized or destroyed.

- ***Antibiotics***
  - Moyer's solution,[‡] 0.5% $AgNO_3$, has been utilized as an agent in burns to overcome superficial and even deep infection of the involved extremity.[4]
  - Silvadene, a drug used in burn therapy, has been helpful in the treatment of digital freezing injury, particularly after bleb loss or rupture. Although salves and ointments have not been considered helpful in treating freezing cold injury because they prevent lavage of the injured tissue with whirlpool therapy, hexachlorophene detergent cleanser (pHisoHex[*]), povidone-iodine (Betadine[*]), chlorhexidine gluconate (Hibiclens[*]), or benign surgical soaps have all been helpful.

[*]Manufacturing and availability information: Tolazoline hydrochloride (Priscoline hydrochloride; mfg: Ciba Pharmaceutical Co, Summit, NJ); Pentoxifylline (Trental; mfg: Hoechst-Roussel Pharmaceuticals, Somerville, NJ); Nifedipine (Procardia; mfg: Pfizer Inc, New York, NY); Phenoxybenzamine hydrochloride (Dibenzyline; mfg: SmithKline Beecham, Philadelphia, Pa); pHisoHex (hexachloraphene detergent cleanser; mfg: Sinofi Winthrop Pharmaceuticals, New York, NY); Betadine (povidone-iodine; mfg: Purdue Frederick, Norwalk, Conn); Hibiclens (chlorhexidine gluconate; mfg: Stuart Pharmaceuticals, Wilmington, Del).
[†]Not often used; listed in *AHFS Drug Information*. Bethesda, Md: American Society of Health-System Pharmacists, American Hospital Formulary Service; 1999.
[‡]The solution is made fresh by the hospital pharmacy on request. Moyer's solution was developed by Moyer for burn care at St Louis University School of Medicine, Department of Surgery, St Louis, Mo. It is good for superficial infection in frostbite too, and it also lessens pain.
FCI: freezing cold injury
Sources: (1) Salini Z, Wolverson M, Herbold D, Vas W, Salini A. Treatment of frostbite with IV streptokinase: An experimental study in rabbits. *AJR Am J Roentgenol.* 1987;149(Oct):773–776. (2) Tuomey J. An open label pilot study to evaluate the efficacy and safety of intra-arterial tissue plasminogen activators (TPA) in the treatment of severe frostbite. Minneapolis, Minn: Trauma Center, Henepin County Medical Center; December 1989. Letter. (3) Zdeblick T, Field G, Shaffer J. Treatment of experimental frostbite with urokinase. *J Hand Surg Am*. 1988;13A(6):948–953. (4) Mills WJ. Frostbite: A discussion of the problem and a review of an Alaskan experience. *Alaska Med*. 1973;15(2):31,32.

tures, gradually develops tissue demarcation and gangrene, requiring amputation of the part. Severe endothelial damage or reperfusion injury is probably the reason for the bad outcome.

Escharotomy. Escharotomy should be performed on the dorsum or lateral aspect of the digits when the eschar is dry and has firmed sufficiently to have a cast effect on the digits, limiting their joint motion. Digits will be debrided further in the whirlpool, without prematurely exposing underlying

EXHIBIT 14-6

SURGICAL PROCEDURES FOR POSTTHAW CARE OF EXTREMITIES WITH FREEZING COLD INJURY

- Escharotomy and escharectomy
- Bleb, bullae, and wound debridement
- Fasciotomy
- Arteriotomy
- Vascular wound repair
- Dermal graft procedures:
  - Reverdin (Davis) pinch grafts
  - Split-thickness skin grafts
  - Split-thickness skin graft (mesh)
  - Free, full-thickness skin graft
  - Cutaneous pedicle flap graft
  - Muscle, musculocutaneous vascular flap transfer with associated nerve supply
  - Very early digital debridement with vascular cutaneous flaps
- Controlled subcutaneous balloon tissue expansion, often fraught with danger because of pressure over the small vessels in the region
- Modified guillotine amputation
- Closed amputation with or without suction irrigation
- Closed or open reduction of fractures and dislocations
- Joint contracture releases, joint excision, and replacement and joint fusion
- Soft-tissue and web-space releases
- Surgical regional sympathectomy
- Periarterial sympathectomy, microdigital sympathectomy
- Excision of sinus tract for the presence of squamous-cell carcinoma (a rare occasion)
- Tissue compartment releases, carpal and tarsal tunnel syndromes

epithelializing tissues.

Sympathectomy. In patients with apparently equal bilateral injury, results of sympathectomy within the first 24 to 48 hours have demonstrated that although there is no further preservation of tissue, there is

- decrease in pain;
- marked decrease in edema;
- much less infection, both superficially and deep; and
- early and more proximal tissue demarcation.

Pharmacological sympathetic blockade may be considered as an alternative. Particularly effective has been the use of phenoxybenzamine hydrochloride (Dibenzylene; manufactured by SmithKline Beecham, Philadelphia, Pa), given 10 mg daily and increased to 20 to 60 mg/d, depending on the effect and need. This drug is used for vasospasm and appears to be an effective α-adrenergic blocking agent. It is important that the patient be well hydrated after surgical or chemical sympathectomy. *Sympathetic blockade is especially effective if accompanied by fasciotomy in casualties with severe FCI, who have associated increased tissue compartment pressure.* Lumbar block for the lower extremities and stellate ganglion block for the upper extremities are appropriate, with an epidural approach to the former being especially useful. Epidural blockade is often given in the continuous mode and may be repeated as necessary. The treatment is seldom maintained for longer than 4 days but has been highly effective in relieving the severe pain, edema, and pallor often associated with NFCI.

Debridement and Amputation. Debridement or amputation, if required, should be delayed until sufficient time (often 15–45 d) has elapsed to demonstrate

a line of demarcation, mummification, and tissue death; and with no danger of further retraction of tissues. It should be emphasized that premature amputation during the edematous stage often results in further retraction of tissue that could be utilized to cover the residual stump. Overwhelming infection, often found in freeze-thaw-refreeze injuries or in extreme extremity trauma complicated by freezing, may result in overwhelming sepsis, requiring immediate amputation to avoid toxic shock.

Skin Grafts. The grafting of split-thickness skin to large granulating areas or areas where skin cover is considered proper may be carried out from the 3rd to the 14th day. The results of skin graft are best when it follows thawing by rapid rewarming. The pedicle grafting of full-thickness skin is a late procedure. The use of a mesh skin graft at the time of fasciotomy, or soon after, reduces the morbidity, lowers the incidence of scarring and infection, and allows proper draining through the mesh openings.

Infection. Antibiotics are not necessary except where infection is deep. Common bacterial organisms found in the injured tissues include staphylococci, streptococci, and *Pseudomonas* species, and often an abundance of Gram-negative species. Clostridia species are occasionally found. Routine cultures and sensitivity studies are taken, and at the first indication of nonsuperficial infection that is not cleared by whirlpool washing, aggressive antibiotic therapy by oral, intramuscular, or intravenous methods is utilized. Particularly for injuries that occurred in the field and are associated with frostbite, and for frostbite alone, it is helpful to utilize the toxoid booster; in the event of no booster being available, antitoxin may be given.

Pain Management

Narcotics are used sparingly in uncomplicated cases after initial thawing; tranquilizers or aspirin will generally suffice, although pain obviously varies with each individual. Pain also varies with the type of injury, the degree of edema, and the presence or absence of infection. Pain is lessened by immediate physiotherapy, activity, and whirlpool bath. In severe cases of immersion injury, with edema prior to fasciotomy, or with high-level extremity freezing (postthaw), pain relief is provided with continuous epidural block for 24 to 48 hours. Requests for pain medication often increase when it is apparent that necrosis of tissue is eminent or present–pain and distress having both a physical and a psychological cause. The discomfort, too, is lessened by a healthy doctor–nurse–patient relationship; by adequate physiotherapy and occupational therapy; and by whirlpool baths and analgesia as required.

In severe cases of FCI, and particularly so with the acute stage of postwarming immersion injury, pain relief may be provided with continuous epidural block, the use of a long-acting anesthetic such as Marcaine (bupivacaine hydrochloride; manufactured by Sanofi Winthrop Pharmaceuticals, New York, NY), for 24 to 48 hours, and repeated if necessary. This treatment is especially effective if accompanied by fascial release in severe cases of freezing or immersion injury, when increased compartment pressures are present. Helpful to these patients is an anesthesiologist or pain center physician. Choices of epidural medication usually are 0.25% Marcaine in a bolus or with an infusion pump. Marcaine is utilized with the axillary or stellate blockade to achieve a long-acting effect; occasionally, indwelling axillary catheters are utilized with an infusion pump or 1.5% Xylocaine (lidocaine hydrochloride; manufactured by Astra USA, Westboro, Mass), occasionally supplemented with a steroid such as Depo-Medrol (methylprednisolone acetate; manufactured by The Upjohn Company, Kalamazoo, Mich).

Prognosis

Prognosis for recovery depends on the extent of freezing, other associated trauma, the manner and timing of thawing, and if freeze-thaw-refreeze or reperfusion injury has occurred. After FCI, the prognosis for a good result with the least number of complications occurs if

- the frozen state is of short duration and the depth of injury is minimal;
- thawing is by rapid rewarming;
- blebs develop early and are very distal or pink and large;
- blebs extend to the distal tips;
- in the process of rescue, trauma has been minimal and refreezing has been avoided; and
- there is an early and rapid return of capillary perfusion, especially as demonstrated by technetium 99m radioisotope studies.

Prognosis for an adequate result is uncertain if

- the frozen state is of long duration;
- the depth of freezing is deep;
- thawing is spontaneous or delayed (other than rapid rewarming); and
- freezing is superimposed on fracture or dislocation, or associated with moderate to se-

vere hypothermia.

The prognosis is almost certain to be poor and with complications if

- thawing is delayed (by means of ice, ice water, snow, friction massage);
- thawing is by excessive heat (> 49°C, 120°F);
- postthaw blebs are proximal, dark, hemorrhagic, and do not extend to the distal phalanges;
- necrosis is early, with early advent (4–5 d) of mummification or liquefaction necrosis of the involved part;
- freeze–thaw–refreeze injury has occurred;
- there is obvious loss of cellular perfusion, as identified clinically and confirmed by technetium 99m studies, on early and repeated views; and
- reperfusion injury is suspected.

Throughout all our experience with cold injuries obvious unsolved problems have remained, namely, the poor results when FCI is superimposed on immersion injury or hypothermia; when the epiphyseal plates of children are subjected to freezing; and one of the most disastrous events of all, when freeze–thaw–refreeze injury occurs. This latter injury is of major consequence at altitude, where, in addition to the refreeze injury, compression injury may also occur above the level of 15,000 ft or in aircraft, and particularly so at altitude in arctic mountains. At that altitude and latitude, the barometric pressure–different from on mountains near the equator–is nearly 0.5 atm and permits expansion of cellular stocking or boot-liner material, such as neoprene stocking material. Nonpliable leather or plastic outer boots, unable to expand, allow the increased pressure from the underlying cellular expansion to be directed downward against the soft tissue of the foot. This compressive force deprives the foot of required microcirculation; ischemia soon develops, followed by local circulatory failure and freezing.

SEQUELAE OF COLD INJURY

Tissue freezing is almost always followed by some residual loss of anatomy or function, slight though that loss may be. The sequelae of cold injury may be categorized as transient or long-standing; before we list the sequelae of FCI and especially of NFCI, it is pertinent to list their present importance. As previously noted, in World War II, especially during the Battle of the Bulge and the winter campaign of 1944/45 in Europe, severe cases of FCI and NFCI occurred. These injuries are well documented in a volume of the official history of the US Army Medical Department in World War II, published by The Surgeon General.[19]

In the Korean War that followed, especially during the 1950 winter months of November and December and during the 15-day retreat from the Chosin Reservoir, many more cold casualties were obtained as the beleaguered troops—US Marines, US Army, US Air Force observers, and allied support, including the Royal British Marines and the South Korean forces—fought back to the sea, surrounded by more than 150,000 Red Chinese troops, who had just entered the war. The allied group numbered 15,000 and despite the overwhelming odds managed to reach the sea, carrying their wounded with them, marching, many of them, with cold-injured extremities (Exhibits 14-7 and 14-8).

For years, this group was told that for purposes of compensation, cold injury was considered a nonservice–incurred event. This decision has since been overruled by the US government and the Department of Veterans Affairs. Beginning in the fall of 1996, veterans of World War II and the Korean War began to be evaluated, 45 to 55 years after the injury for service-incurred disability. Most are aged 60 to 70 years, and a few are 80 or more years of age. On physical examination, many of these survivors demonstrate signs of sequelae (Exhibit 14-9). As more veterans are evaluated, the demonstration of cold-injured sequelae becomes more apparent—so much that it may be said that any significant FCI or NFCI will demonstrate some residual finding, often increasing as the veteran ages and time goes by.[96]

In examining the cold-injured patient for sequelae, first thought should be directed to an adequate history of exposure (eg, the patient's having been a member of the 84th Infantry Division, Battle of the Bulge, 1944/45; or a member of the 1st Marine Division in their march to the sea from the Chosin Reservoir in Korea, November or December 1950). A further history should include the type and the duration of exposure, the apparent type of injury as FCI or NFCI, and medical verification by the medical records, if they still exist. In the march south from the Chosin Reservoir, many of the records of the beleaguered troops were lost or destroyed, adequate medical history was taken on very few, and unfortunately, according to their current history, very few had a discharge physical examination.

All these details would be helpful: the duration

EXHIBIT 14-7

THE RETREAT FROM CHOSIN RESERVOIR: A MEDICAL OFFICER'S EXPERIENCE

The Chosin Reservoir is a large, lakelike reservoir in northeast North Korea, one of a chain of lakes used to create hydroelectric power for that area. Then a lieutenant in the Medical Corps, US Navy, I served as a battalion surgeon in the 2nd Battalion, 7th Marines, in the Chosin Reservoir Campaign in November/December, 1950. Temperatures at that time were some of the lowest ever recorded in North Korea, reaching –30°F and –40°F on occasion. My battalion aid station was located in Hagaru-ri, a small village on the south end of the Chosin Reservoir. On the northeast side of the reservoir were stationed US Army units that had been placed in an isolated geographic location, with only a single winding road offering supply and evacuation possibilities. On the west side of the reservoir was located a small town called Yudam-ni, approximately 10 miles over rugged terrain north of Hagaru. As we marched into Hagaru-ri, the temperature was well below zero. Marines were forced to stand in line for long periods while the area in front of our troops was cleared. It soon became apparent that men were freezing while standing still, with white discoloration of the nose, cheeks, and ears. I assigned my corpsman the job of going up and down the standing line of troops to establish a buddy system, so that each Marine would monitor his buddy for frostbite. Despite this, many of the troops developed redness and blistering of their faces and ears that day.

The Marines were assigned positions in the mountains surrounding Hagaru. The Chinese had come into the war. Unfortunately, with the first contact with the Chinese, some of our troops were bayoneted in their sleeping bags. This story was quick to spread throughout the entire battalion. As a result, many of the men were unwilling to even get into a sleeping bag, using it as a blanket instead. We were assigned shoepac boots. These contained a 3/4-inch felt innersole. A spare innersole was issued to the soldier or Marine; this was to be kept against his body inside his undershirt. The theory behind this special boot was that body heat would evaporate any perspiration in the innersole, and at night the shoes could be taken off, the toes massaged and possibly powdered, and a fresh, dry, warm innersole would be inserted into the boot. The other perspiration-laden innersole was to be removed and placed against the body for evaporation. Unfortunately, the men in the field were unable to take off their boots, especially if they were not sleeping in their sleeping bags. In –30°F weather, the shoepacs froze. This caused a layer of ice to form across the sole of the shoepac, causing freezing of the feet, painful walking, and, when boots were removed, swelling to a point that boots could not be placed back on the feet. The soles of these men's feet looked like raw meat. Many had bloody blisters on one or more toes. In addition, some sustained cold injury to the fingers and hands from outdoor contact with metal weapons.

At the time the 1st Marine Division moved into northeast Korea, General Douglas MacArthur's headquarters had issued statements that, for practical purposes, the war was over and that we would be home by Christmas. The issuance of winter gear was delayed until late in November, since there was no reason for issuing gear if we would be out of there in such a short time. Unfortunately, the weather was already below freezing before we received winter parkas, heavy gloves, and shoepac boots.

No education regarding frostbite prevention was even attempted. In fact, the medical personnel were lacking in knowledge of short-term effects of frostbite and how best to treat this. Fortunately, in our aid station we had several petty officers who had served in World War II and had some cold weather experience. They gave us advice as to slow warming of frozen tissue versus rapid warming, dry warming versus immersion in water, and so forth.

A small airstrip was bulldozed out of the ice in Hagaru. This strip was able to take small, propeller-driven airplanes, which could evacuate only 28 stretcher cases or about 32 seated casualties at a time. Evacuation of many of the frostbite victims would have been desirable. This certainly would have allowed them to get to Japan, where the weather was warmer and where slow thawing of tissue and proper treatment could have been administered. Unfortunately, we were overwhelmed with severe combat casualties, many of them from the 31st and 32nd Infantry Regiments, which had been stationed east of the Chosin Reservoir. Many of their officers were killed, and the Chinese took their time to destroy this trapped fighting unit. Many soldiers crawled across the ice of the Chosin Reservoir to try to reach Marine positions. A tank infantry team was organized to place drivers on trucks that had been stalled on the single-lane road. These trucks were brought into Hagaru-ri, and many of the casualties were assigned to my battalion aid station. We treated hundreds of casualties

(**Exhibit 14-7** *continues*)

Exhibit 14-7 *continued*

over the next 3 to 4 days. Many of the casualties had multiple gunshot wounds, fractures, skull injuries, sucking chest wounds, and the like. All of these soldiers were frozen. We had to cut their boots off their feet. Initially when we tried to remove the boots, some of the toes would come off in the boot. We found we could do less damage by cutting the leather boots and slowly removing the foot, almost dissecting the foot out of the boot, in an attempt to preserve tissue. With these multiple, severe, life-threatening injuries, the evacuation of Marines and soldiers with simple frostbite had a lower priority. In fact, we had orders to keep all troops in the line who could ride "shotgun" and protect the driver by riding in the right front seat, even though they could not walk! At this same time, Marines were returning from Yudam-ni, having successfully escaped from Chinese encirclement. All of these troops had experienced prolonged outdoor exposure to freezing weather and snow. All had some degree of frostbite of toes and, in many cases, hands and ears.

After the march back from the Chosin Reservoir to board ships at Hungnam to move to Pusan, South Korea, many of the troops who had blistered feet found that the fluid was absorbed and that the tissue returned to "normal." These men stayed in the line and no record of frostbite was ever documented in their medical records. Now, years later, many of them suffer the long-term effects of cold injury. One interesting observation after we reached Masan, South Korea (where the weather was warm), we suddenly found our sick-call line two city blocks long! These men had upper respiratory infections with nasal congestion, cough, and sneezing. Apparently, while they were in North Korea with its subfreezing weather, the common cold virus somehow did not cause trouble. We did see respiratory illness, such as bronchitis and even some cases of pneumonia, but even these were not common. The sudden emergence of upper respiratory infections certainly was somehow associated with exposure to a warmer environment.

Lessons Learned From My Chosin Reservoir Experience

1. The shoepac boot theoretically could prevent cold injury, if properly used. In a combat experience where replacement of perspiration-laden innersoles is impossible, however, they contribute to severe cold injury.
2. While it is impossible to anticipate all medical problems, the possibility of exposure to harsh winter conditions should have prompted the leaders of the medical departments of the US Navy and Army to offer educational seminars on how to handle these injuries. We spent 2 weeks patrolling north and south in the Yellow Sea, while the largest mine field in history was cleared from Wonsan Harbor in North Korea. This would have been ample time to brief the medical departments on what might be in store. Perhaps we could have prevented some of the severe cold injury with this information.
3. A constant review of equipment for harsh weather conditions should be mandatory in the armed services. Our troops deserve the best possible equipment, which should not be compromised for financial reasons.

Exhibit prepared for this textbook by Stanley I. Wolf, MD; formerly, Lieutenant, Medical Corps, US Navy Reserve; Second Battalion, 7th Marines, Hagaru-ri, Chosin Reservoir, North Korea, November/December, 1950; currently, 7213 Greentree Road, Bethesda, MD 20817-1507.

of exposure, depth of injury, diagnosis of degree of injury or description of the injury as superficial or deep. Did a refreeze injury occur? What was the appearance of the extremity? Was there associated trauma including gunshot wound?

An ongoing medical history and physical examination are in order to determine or rule out specific or general systemic diseases that might affect the sequelae. For example, preexisting or present diabetes, arthritis, peripheral vascular or neurovascular disease such as Raynaud's syndrome or labile vasomotor changes, small-vessel disease from any cause, or neurovascular loss all might cloud the picture when evaluating for sequelae. Equally, the sequelae may be altered by, or actually be arthritic, or undergoing musculoskeletal change. The appearance of edema, swelling from any cause, skin-color change, dependent rubor and elevation pallor or loss of pedal pulses-any and all of these are pertinent findings during the examination.[97] Specifically, the general appearance of the limb should be noted, including skin-color and musculoskeletal changes (muscle atrophy or limitation of joint motion, digital deformity, scarring, edema, and swelling).

It is wise to have the patient go through a range of motion of the fingers and to have gait evaluation by heel-toe walking, as well as examining the appearance of the toes, looking carefully for onycho-

EXHIBIT 14-8

THE RETREAT FROM CHOSIN RESERVOIR: A CORPORAL'S EXPERIENCE

I joined the US Marine Corps Reserves on 6 March 1948, and was then attached to B Company, 11th Infantry Battalion. On 5 August 1950, I was called into active duty along with the rest of our reserve unit from Aberdeen, Washington, Grays Harbor County. At that time we consisted of about 350 Marine Reserves. I was sent to Camp Pendleton, California, where I received overseas medical shots; in mid September I was sent with a work party to San Diego, California, where we spent about 12 days loading winter gear onto a ship that was to go to Korea. On 29 September I boarded the *General Nelson M. Walker* with about 2,000 other troops and about 15 October docked at the port of Yokahama, Japan. We trained in Japan about 2 weeks at the Marine base in Otsu, Japan, then boarded the USNS *Aiken Victor*y on 3 November and arrived in Won Son, Korea, on 6 November 1950. We were the first replacements for the 1st Marine Division. We were told that winter gear awaited us at our units and that everything would be distributed there. But when we arrived and were assigned to our outfits, there was no winter gear.

Later, six other Marines and I were sent to B Battery, 11th Marines, as replacements. It was nearly dawn when we arrived. We immediately began to dig our foxholes but the ground was frozen solid—almost like digging into solid rock. We were told at the time that the temperature was –42°F. Along with my best friend from Aberdeen, Leland Godfrey, and one other GI, I got up every 2 hours during the night to do calisthenics to keep warm. The next morning the four other replacements, who had slept straight through the night, were sent back to the hospital in Seoul, South Korea, where they were treated for frostbitten feet, hands, ears, face, and buttocks. The three of us who had exercised to keep warm were OK. After 2 days and nights of freezing weather we were finally issued winter clothing. Later that morning we were informed that the decision had been made: we would fight our way out of the trap. We thought we had protection on our flanks: the 8th Army on one side and a division or so of the Republic of Korea (ROK) Army covering the other. Unbeknown to us, however, the 8th Army had been overrun and had fled down the mountain. They had been ambushed and had left all their equipment (many of the trucks still running) along the treacherous road. Also unbeknown to us, the ROK forces covering our opposite flank had also been overrun and annihilated. We were unprotected, alone, and surrounded by almost 120,000 Chinese troops. Within the Chosin Reservoir, our own forces consisted of two battalions of the Army's 7th Infantry Division and the 1st Marine Division: 20,000 men. We were outnumbered 6 to 1.

All troops were ordered to rendezvous at Yudam-ni, and from there the battle would begin. Because we Marines are trained first in infantry, many of us volunteered to fight with the 7th Marines. I was placed in a group of 20 other GIs from the 7th. While on patrol, we were attacked by (from what we could tell) about 75 Chinese troops. We were all huddled together and were able to kill most of them. We took 14 back with us as prisoners. They were wearing shoes that looked like tennis shoes. A foot of one of the prisoners had come off his leg just above the ankle; it had been frozen and gangrene had set in. I don't know how he remained alive.

I had been with the 7th Marines only 3 or 4 days when I was sent back to my artillery unit from the 11th. Things went fairly smoothly while I was with the artillery. But we each took our turn on the front lines as a forward observer, whose job was to pinpoint targets for the artillery and give the coordinates over the radio. And regardless of what our position may have been, we were poorly equipped. Our carbines froze, our M1s and 105-mm howitzers were left over from World War II, and our thermoboots left much to be desired. As long as we were moving, our feet stayed warm and even sweated; but as soon as we were still, the sweat would freeze and so would our toes. But we accomplished what we needed to: our howitzers wreaked havoc on the Chinese troops and enemy equipment. (Each Chinese soldier carried simple but effective equipment: a backpack, which held a straw mat and a white sheet. In a field of grass, they would cover themselves with the straw mats, then with the sheets. In snow, the white sheets blended perfectly and they were able to hide entire divisions.)

The enemy demolished every bridge to keep us from withdrawing from the trap, so we were forced to find other means of crossing waterways and valleys. Huge roadblocks were placed in our path to buy time for the opposing forces. Between Yudam-ni and Toktong Pass we had 30% casualties. The Chinese constantly shelled us with white phosphorus, and even lobbed phosphorus grenades into truckloads of wounded Marines. The fighting at Hagaru-ri was fierce. We had a makeshift airstrip there, which was used to evacuate the wounded

(**Exhibit 14-8** *continues*)

Exhibit 14-8 *continued*

and victims of extreme frostbite. The enemy wanted that airstrip and concentrated all their strength there. Being outnumbered (now nearly 9 to 1), we could not hold. We had previously carried all our dead with us but now would be forced to leave them; we used bulldozers to push the bodies of several hundred men into a mass grave. We then loaded our wounded and almost all our equipment (destroying the little we could not take) and withdrew from Hagaru-ri.

We were fighting not only the enemy but also the weather, and we lost as many men to frostbite as we did to the Chinese. We were not used to such cold conditions; during the winter it was never above –20°F. The enemy had lived all their lives in this weather, however, and benefited from that. Our artillery could not recoil it if got too cold, and our carbines were worthless. The M1 rifle was our best weapon. It took 10 to 15 days for us to fight completely out of the trap. We lost nearly half our men and more than half of those to the cold weather. Most of the troops had not been able to sleep since the rendezvous at Yudam-ni and had little to eat because our C rations were completely frozen. On the last leg of our retreat, when we were headed for the collection center at Hamhung (the port of evacuation was at Hungnam), the Chinese had set up roadblocks all along they way and fired on our left flank from machine gun nests along the way. Men were slaughtered; we had no defense. I tried to return fire but my fingers were so frozen that I couldn't even pull the trigger on my M1. As the Chinese continued to fire on us, a group from the 7th Marines came up from behind and destroyed their guns. By the end of the battle we had lost just over 12,000 troops: 3,000 men were dead; 6,000 were wounded; and 3,000 more had frostbite. When we finally arrived at Hungnam, the Marines there had prepared hot food and warm tents for us. The next day we boarded troop transports, carriers, freighters, and fishing boats, which had been held for our retreat.

The battle from the Chosin Reservoir was called "the Christmas Miracle" by the Koreans. Because we had been able to hold out so long, we were able to take more than 100,000 North and South Korean refugees out with us. I have been told that this was one of the largest rescue operations in history; never before had so many enemy civilians been rescued in the midst of battle. Although some historians are said to have called this the "most savage battle of the 20th century," I believe that we would have been at least 50% more effective if our military had known how to train us properly for such battle conditions. The training and equipment provided now, half a century later, far surpass nearly everything we had during the Korean War.

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mycosis or onychogryposis. Range of motion of ankle and toes should be examined, with evidence of intrinsic muscle loss of hands or feet noted, and consideration given for the presence of tarsal or carpal tunnel syndrome, which are often sequelae of freezing injury.[98] Instruments are available to measure range of motion, both flexion and extension, lateral and medial deviation, and claw toe or hammer toe, for which small goniometers are available and helpful. For assessing sensation, a pin wheel may be utilized to measure hyperesthesia, normal sensation, hypesthesia, or anesthesia. For determining nerve function, a tuning fork is helpful; a tape measure is useful for measuring circumference to determine the presence of edema; and a palpable examination is necessary to determine the presence of pitting edema. An anesthesiometer measuring 2-point discrimination is helpful, and a telethermometer recording in Centigrade or Fahrenheit is useful, particularly for comparing extremities. Ultrasound Doppler for measurement of pulses not palpated manually is also helpful.

EVALUATION OF MILITARY PERSONNEL FOR RETURN TO DUTY

The range of disability varies following FCI and, depending on the degree of injury, may vary from very little to a large amount. A greater degree of disability precluding return to normal duty may follow NFCI (trench foot) than FCI (frostbite). In both cases, sequelae may begin to demonstrate themselves in 6 to 18 months and thereafter. The ultimate in loss, amputation, may not preclude the patient's return to duty, full or limited, depending on the level of amputation and the ability of the soldier to function in the field. These factors can readily be determined by examination or actual ac-

EXHIBIT 14-9

SEQUELAE OF COLD INJURY

Transient sequelae
- Hyperhidrosis
- Hypesthesia or anesthesia of digits
- Limitation of motion of the interphalangeal and metatarsal or metacarpal joints
- Swelling of the interphalangeal and metacarpophalangeal joints
- Edema of digits, hands, and feet
- Thin, fragile epidermis in involved areas
- Nail loss
- Intrinsic muscle atrophy
- Fat pad loss from distal tips of digits
- Pain from injury to peripheral nerves and small vessels as a result of ischemia

Late, long-lasting, or increasing degree of sequelae
- Deep, fixed scars over the affected area
- Atrophy or fibrosis of the affected musculature
- Flexion, extension deformity of distal joints especially involving the toes, often with hammer toe or claw toe deformity
- Volar fat pad loss of fingers and toes
- Hyperesthesia of distal tips of digits, with increased sensitivity to heat and cold
- Residual hypesthesia of digital tips
- Decreased proprioceptive sense of digital tips
- Permanent nailbed deformity, as onychogryphosis, often with associated onychomycosis
- Roentgenographic evidence of subarticular and periarticular lytic destructive changes of bone and cartilage, especially in phalangeal joint areas
- Avascular necrosis of bone, especially in the phalanges, metatarsi, and tarsi (eg, the calcaneus)
- In children or young adults, epiphyseal necrosis or total destruction of physis or epiphysis (growth plate destruction) with joint or phalangeal angulation deformity or shortening
- Chronic ulceration, infection, or osteomyelitis in area of cold injury
- Loss of fibrocartilage of ears and external ear part loss
- Decreased capillary perfusion by isotope examination (indicative of endothelial cell loss)
- Rare findings of carcinoma (usually squamous cell) in long-standing draining sinus tracts, or with chronic wound infection or osteomyelitis
- Interphalangeal joint immobility, marked limitation of joint motion or fusion of interphalangeal joints
- Carpal and tarsal tunnel syndromes (seen in acute stage, too)
- Variable findings or hyperhidrosis, hypohidrosis, anhidrosis
- Intermittent or consistent pain in hands or feet, often extending in the lower extremities to levels above the apparent injury level
- The ultimate in long-standing sequelae, or the result of severe early cold insult, namely, amputation of the involved part at any level

tivity under field conditions. Pain, swelling, neurological loss, poor vascular supply, and the presence of wounds that fail to heal properly usually preclude return to full duty.

Generally, problems of cold sensitivity may limit duty in arctic or subarctic or mountainous regions. Following FCI, sequelae usually are not debilitating enough to demand medical discharge and the

sequelae may consist of only hyperhidrosis, lack of proprioceptive sense, and limited capillary perfusion. Should thermography be available, this could determine the degree of superficial circulation. Studies using radioisotopes such as technetium 99m could determine deep capillary perfusion, and flat-plate roentgenograms could determine any lytic destructive changes near the small joints of the carpal or tarsal areas, as well as the interphalangeal joints. These findings—by roentgenography, thermography, or isotope studies—are often accompanied by physical examination changes, including swelling or limited range of motion.

On the other hand, in NFCI, residual problems of pain, formication, anesthesia of the extremities, or hypesthesia or hyperesthesia are often found to increase as time evolves. Pain along the course of peripheral nerves and digital vessels and marked intrinsic muscle loss or flexion or extension contractures of the interphalangeal joints often occur (see Exhibit 14-7). After FCI or NFCI, exposure to cold *must* be avoided for 6 months following minimal injury and for 12 months following major injury. A second exposure to FCI or NFCI within 6 months or a year may result in major tissue loss-even like those seen in freeze-thaw-refreeze injuries. As is often the case, US Army Medical Manuals; Technical Bulletins, Medicine; and Survey Board Recommendations after examination may help medical officers to decide the wisdom of retention for full or limited duty or separation from the service.

SUMMARY

Freezing cold injury (FCI, the occurrence of ice in and around cells), also called frostbite, has been a frequent source of attrition in mountain warfare and military campaigns fought in the winter. Eight modes of freezing injury can be found in soldiers fighting in a cold environment:

1. true FCI, superficial or deep;
2. a mixed injury: immersion (cold-wet) followed by FCI;
3. freezing, then thawing at any temperature, followed by refreezing;
4. hypoxia; high-attitude environmental injury, usually associated with hypovolemia, dehydration, and extremity freezing;
5. extremity compartment compression from any cause, followed by freezing;
6. extremity fracture or dislocation, followed by freezing;
7. hypothermia, associated with FCI to the extremities; and
8. FCI with superimposed burn injury or burn injury followed by freezing.

FCI leading to cell death has been graded in terms of the magnitude of tissue necrosis both as degree of injury and as superfical and deep. Superficial freezing injury (first and second degree) is manifested as transient hyperemia, edema, and possibly bullae, but there is no permanent tissue loss. Deep freezing injury causes skin necrosis (third degree) or death of underlying tissue (fourth degree), the latter condition frequently leading to amputation.

The principal pathophysiological event of FCI is the formation of ice crystals in the extravascular fluid spaces. Not only may the crystals cause mechanical damage to nearby cells but the loss of liquid water causes extracellular osmotic pressure to increase. The osmotic pressure gradient across the cell membranes draws liquid water from the intracellular space, resulting in electrolyte and acid–base changes that degrade enzymatic functions. As freezing continues, cell membrane damage occurs, especially in the endothelial cells of the microcirculation. On thawing, the initial event is vasodilation, followed by edema and vascular stasis, the latter resulting from intravascular cellular aggregation and consequent microvascular and macrovascular thrombosis. Edema may further contribute to tissue ischemia through mechanical compression of vessels and may be of a severity consistent with the diagnosis of compartment syndrome. During reperfusion of the thawed part, four factors may contribute to the tissue damage seen in cold injury: (1) generation of oxygen-derived free radicals, (2) neutrophil activation, (3) formation of vasoactive prostaglandins and thromboxane from the metabolism of arachidonic acid, and (4) activation of proteolytic enzymes. Tissue damage is especially severe when there is refreezing of a previously thawed part, possibly because of the formation of intracellular ice crystals.

The most important intervention in managing FCI is thawing. Four methods of thawing are commonly seen:

1. rapid rewarming in warm water (37°C–41°C);
2. gradual (spontaneous) thawing at room, cabin, or tent temperature, or in a sleeping bag, so that the thawing range varies;

3. delayed thawing utilizing ice, ice water, or snowpacks, and often accompanied by friction massage; and
4. thawing by excessive heat, which has included car heater, diesel generator exhaust, oven heat, hot water, campfire, or any heat greater than 48°C, often as hot as 65°C to 90°C.

Experience has shown that the first two methods are the least harmful, whereas the fourth is quite deleterious. Systemic hypothermia accompanying FCI can be treated by immersion of the patient in a warm water bath (32°C–37.7°C) and infusion of warm intravenous fluid.

Subsequent management of the freezing cold cold-injured body part includes open treatment (ie, no compressive bandages), whirlpool baths, nonsurgical management of blebs and bullae, fasciotomy when compartment syndrome is diagnosed, and possible use of measures designed to provide vasodilation such as sympathetic blockage or pharmacological vasodilation. Escharotomy should be performed only when an eschar is dry and severely limits motion at a joint. Extensive debridement or amputation should be delayed until sufficient time (often 30–90 d) elapses to allow a stable degree of mummification and tissue death to be reached. Blebs are debrided or trimmed only when infected and containing purulent material. Physical therapy has an important role in determining the functional outcome of a serious freezing injury.

Nonmedical factors that may not be sufficiently appreciated by the clinician are important in determining the final result in the human. These variables are in the realms of weather, inadvertent trauma, and individual human physiology and anatomy, which are often difficult to anticipate, measure, or predict. The state of health and physical condition of the casualty are also vital factors, including the associated factors of alcohol and drug use, mental state at the time of exposure, and the presence of additional injuries. The latter factor is of special importance in soldiers. Trauma preceding freezing, such as extremity strain, sprain, or fracture poses major problems, as does the presence of a penetrating wound, blunt trauma, or blood loss from any cause. FCI is further influenced by the degree of hypovolemia or dehydration present, causing further distal vascular deficiency prior to the onset of freezing. Little considered by most clinicians are problems of rescue and survival, often resulting in freeze–thaw–refreeze injury, and perhaps the irreparable trauma occurring at the junction of frozen and nonfrozen tissue, as "brittle" tissue segments are stressed when the FCI victim must walk, whether out of the wilderness before rescue or during a military retreat (eg, Napoleon's retreat from Russia or US and allied troops at Chosin Reservoir).

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Chapter 15

NONFREEZING COLD INJURY

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INTRODUCTION

Nonfreezing cold injury (NFCI), a syndrome (formerly known as trenchfoot, pernio, and other names) resulting from damage to peripheral tissues in the extremities exposed to cold temperatures,[1–3] remains a major threat to individual military personnel and to military operations carried out in cold weather. NFCI is an injury that does not involve freezing of tissues, which normally distinguishes it clinically and pathologically from freezing injuries,[1] although it is not uncommon for freezing and nonfreezing forms to coexist in the same individual or limb. Whenever military forces are required to operate in cold, wet conditions for more than a few hours, it is always possible that those operations and the health and well-being of personnel will be severely jeopardized or curtailed by NFCI.

The clinical syndrome of NFCI has been known for centuries; however, it is primarily within the military context that it has engendered sustained clinical and historical interest. Observations of NFCI in the military, from earlier times to the present, have been detailed in well-documented historical reviews.[3–5] There has been little continuity in the research efforts to better understand the pathophysiology of NFCI because episodes of interest in the prevention and treatment of NFCI have usually followed periods of major military operations in inclement weather conditions but then unfortunately have been abandoned until the cycle is repeated in further military operations (Exhibit 15-1).

Cold injury is believed to be far more prevalent in combat environments than noncombat or civilian situations under similar conditions of exposure to cold weather.[4,6,7] Because NFCI has been infrequently reported in civilian personnel, it has until recently remained rather obscure as a medical problem. It often goes unrecognized during assessment of individuals suffering from the consequences of cold exposure, as it does not attract emphasis in traditional training about cold-weather medicine. NFCI has received some attention in the civilian sector, however, as a problem related to cold weather medicine in areas such as the homeless,[8] alcoholics,[9] the elderly,[10] and outdoor winter activities.

At present the only if often unattainable strategy for control of NFCI is prevention. As this is frequently not an option, the injury remains a threat to individual military personnel carrying out missions in cold weather. Because NFCI is likely to continue as a substantial medical problem in any extended military cold weather operation, it is currently as important as it ever has been historically to develop effective preventive, diagnostic, and treatment methodologies.

CLINICAL FEATURES OF NONFREEZING COLD INJURY

To further define the syndrome of NFCI, the clinical features will be described (Figures 15-1 through 15-7) before addressing more general considerations of the etiology of the pathology, the treatment, and the prevention of the condition.

The evolution of NFCI in humans, as revealed by clinical studies, involves quite distinctive stages.[1,11–15] Generally, the clinical literature distinguishes four stages, including during and after exposure, and during and after hyperemia, in the progression of the condition; the stages are commonly attributed to Ungley and colleagues.[11,12] The signs and symptoms of different stages are not always clearly demarcated and often blend from one to the next. The time course of each stage is highly variable and some are often short-lived. The presentation and differentiation of the various NFCI stages depend, in a complex fashion, on both the exposure (its duration and the severity of the cold) and on the condition of the individual.

First Stage: During Cold Exposure

The initial stage of NFCI occurs during the period of exposure to the cold environment. Cold exposures that promote NFCI have been reported to vary from just below freezing to 20°C, although the instigation of NFCI may occur at even higher temperatures. Depending on the specific environmental circumstances, exposure durations of less than 1 hour have been reported to induce injury, while exposures as long as a week have been recorded[3] under less severely cold conditions.

The single most important diagnostic criterion is the loss of a sensory modality, most typically complete local anesthesia, which is distinct from premonitory feelings of extreme cold in the affected periphery, almost invariably in the foot although hands can also be affected. Consequently, the most common symptom expressed is that of numbness, although some patients give characteristic descriptions of feeling as if their feet were made of cotton wool, or that they were "wearing someone else's feet," indicating loss of proprioception. As a consequence of these sensory disturbances, casualties may also report disturbance of gait, clumsiness, and stumbling. The extremities may initially be a bright red color, but later almost universally change to a paler color, even completely

EXHIBIT 15-1

THE FALKLANDS WAR: STIMULUS FOR NONFREEZING COLD INJURY RESEARCH IN THE UNITED KINGDOM

It was quickly apparent during the Falklands War in 1982 that a substantial number of casualties with nonfreezing cold injury (NFCI) would be returning to the United Kingdom. Because a high proportion of those were to be Royal Marines, who had a commitment to the protection of the North Atlantic Treaty Organization (NATO) northern flank, and thus would normally be deploying to Arctic Norway within a few months, it was apparent that there could have been a problem. The existing team in the Department of Survival and Thermal Medicine, at the Institute of Naval Medicine (INM), already had great interest and some clinical experience in cold injury. Led by then-Surgeon Commander FStC Golden, Royal Navy, already a leading authority on hypothermia, they quickly assembled the equipment needed to follow up patients with NFCI so that they could advise the command on the future care and disposal of patients.

Guided by the results of questionnaire surveys of returning Royal Marines and Army personnel and both existing literature and outside advice, they knew that they would need to make objective assessments of peripheral nerve and neurovascular function. At first, they employed three techniques:

1. conventional neurophysiological measurements including conduction velocity,
2. noninvasive measurements of peripheral blood flow such as strain gauge, and
3. infrared photoplethysmography and infrared thermography.

A variety of cold stress tests were evaluated, including cold water sprays and forced air convection, to try to elicit evidence of prolonged vasoconstriction following cold exposure. Although the other techniques continued in use until 1985, it was clear that infrared thermography yielded as good information as any, and this later evolved into the standard cold stress test with concurrent thermography still used for clinical assessments today.

Although never reported in full in the open literature, Golden's work demonstrated a number of key features of the mild NFCI that plagued British servicemen in the Falklands. Most important among these was the severity and persistence of cold sensitization following even the most minor cases of NFCI: many of their patients had not reported sick during the conflict but had managed to cope with their symptoms while still fighting. However, on their return to the United Kingdom, most were markedly cold sensitized and few deployed to Norway for the winter warfare training of 1982/83. Golden's work established the INM Cold Injuries Clinic, which today sees about 150 cases each year, drawn from the service and civilian populations in the United Kingdom. It has also reviewed veterans of the Chosin Reservoir campaign (November–December 1950) during the Korean War, who continued to suffer the sequelae of their predominantly freezing cold injuries.

Research at INM has continued in the hands of Oakley, a pupil of Golden's. The former introduced in 1987 the use of thermal sensory threshold measurements to augment thermography, following this serendipitous introduction to the technique by Fowler, one of its developers. In 1991, Golden and Oakley were advised to introduce laser Doppler systems to measure skin blood flow in the peripheries, which Oakley then used for his PhD research at INM between 1992 and 1996. Although laser Doppler techniques can provide intimate detail of the responses of injured microvascular beds to a range of physiological maneuvers, they have not supplanted thermography in clinical assessment. In 1995, Oakley became one of the first investigators to use single capillary laser Doppler velocimetry in his quest for better understanding of the processes promoting cold sensitization. Perhaps most remarkable of all is that the Royal Marines and INM have continued to support a small research program for 18 years; had programs of similar durations been maintained in the past, it is likely that NFCI would be much better understood today.

blanched white. This pallor reflects intense ischemic vasoconstriction in the extremities, which is one of the key features in the pathogenesis of NFCI. Pain and swelling are properly absent at this time, although attempts at rewarming during routine foot care may introduce these third-stage features here at the first stage, and thus complicate the clinical picture.

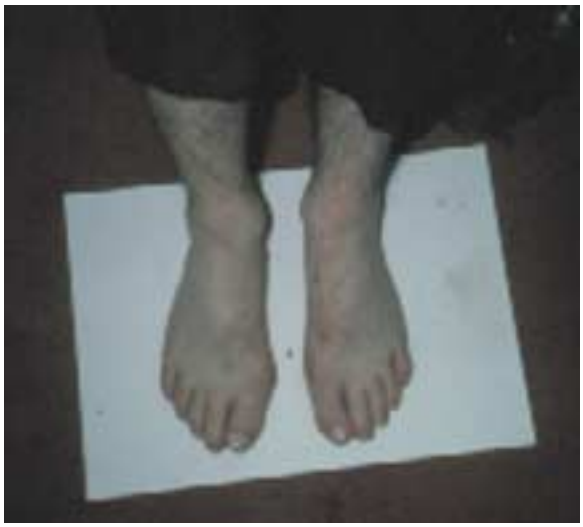

Fig. 15-1. Ungley stage two nonfreezing cold injury in a trainee British Royal Marine. At the time this photograph was made, the feet were still mostly numb and very cold to the touch, but were starting to swell and had changed from white to mottled blue-white. © British Crown Copyright/MOD. Reproduced with the permission of Her Britannic Majesty's Stationery Office.

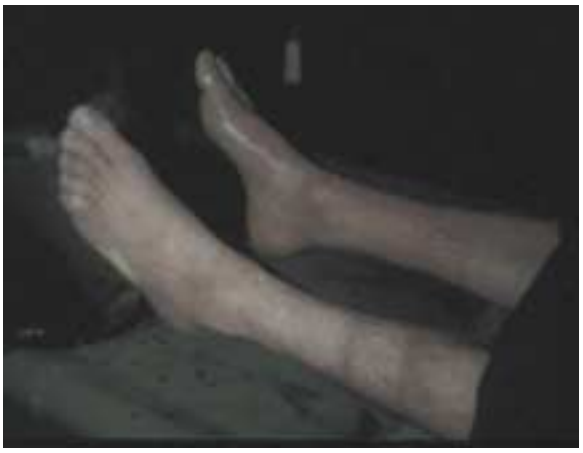

Fig. 15-2. Severe nonfreezing cold injury from the Falkland War. This casualty was recovered from among corpses in the Argentine defensive trenches around Darwin and Goose Green, 3 days after suffering a gunshot wound. The feet (and hands) were very cold, swollen, discolored, and apparently sweaty. Demarcated white patches suggested that some areas had suffered freezing injury. The casualty was also deeply hypothermic (estimated rectal temperature at time of photograph: 28°C). After repatriation, the patient underwent bilateral below-knee amputation. © British Crown Copyright/MOD. Reproduced with the permission of Her Britannic Majesty's Stationery Office.

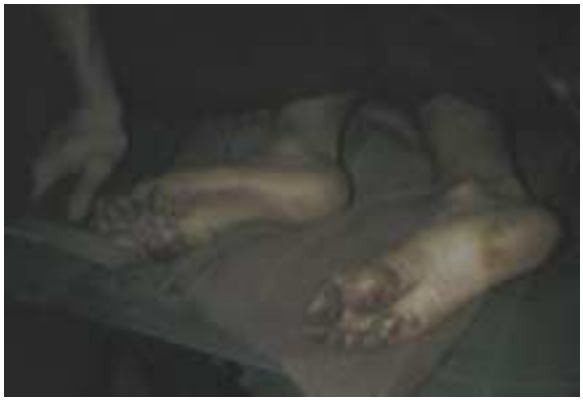

Fig. 15-3. Severe nonfreezing cold injury from the Falkland War. This Argentine soldier had tissue damage in the pulps of the toes and fleshy prominences of the soles of the feet. These appearances are common in those who have been unable to care for their feet for many weeks. © British Crown Copyright/MOD. Reproduced with the permission of Her Britannic Majesty's Stationery Office.

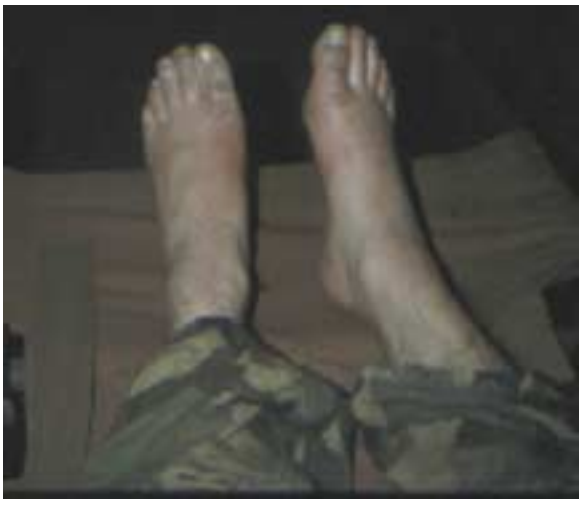

Fig. 15-4. Less severe nonfreezing cold injury in a British infantry soldier from the Falkland War. The feet were swollen, red, and the site of persistent pain. © British Crown Copyright/MOD. Reproduced with the permission of Her Britannic Majesty's Stationery Office.

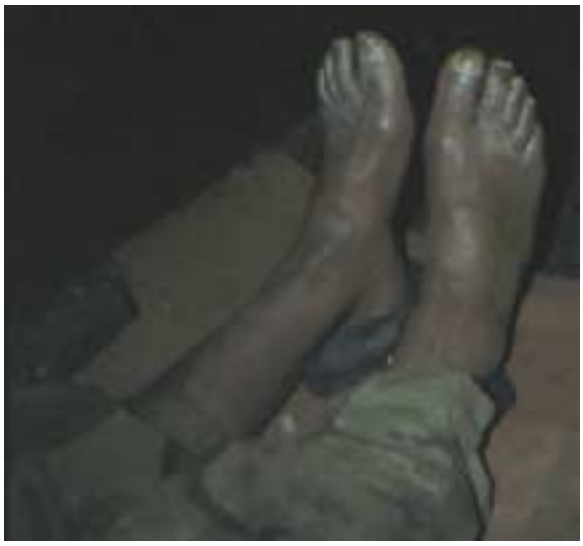

Fig. 15-5. Ungley stage three nonfreezing cold injury in a British infantry soldier with pigmented skin from the Falkland War. The casualty is of African-Caribbean origin, and the characteristic redness of stage three was absent, although the feet were swollen and painful.

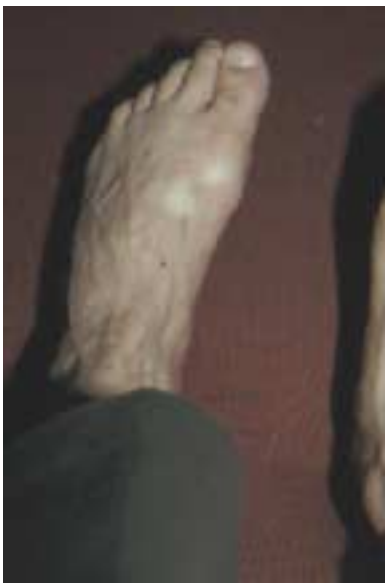

Fig. 15-7. Slow capillary refill demonstrated in mild nonfreezing cold injury. The examiner's two fingers rested gently on the skin of the dorsum of the foot for 10–20 seconds, sufficient to blanch the capillaries. When the pressure was removed, the blanched patches disappeared very slowly, reflecting capillary stasis.

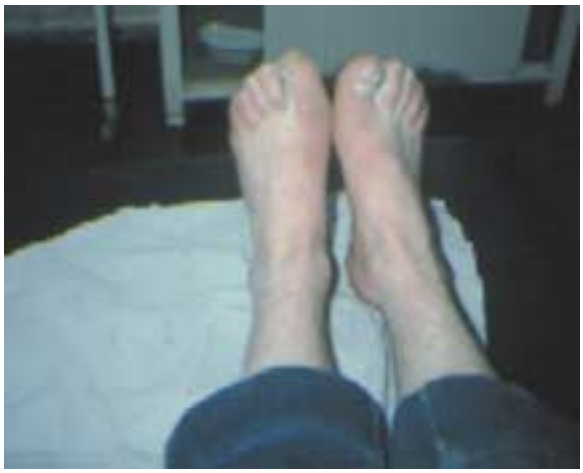

Fig. 15-6. Typical mild nonfreezing cold injury in a trainee British Royal Marine. This recruit had returned from a field exercise feeling well. While showering, his feet rapidly became swollen, red, and painful. (The white powder around his toes is foot powder, not rampant fungal infection.)

Second Stage: Following Cold Exposure

The second stage appears immediately following removal from the cold exposure environment and during or immediately following warming. The duration of this stage is typically fleeting, lasting only a few hours at most, although exceptional cases may persist for several days.

The postexposure stage is characterized by early small increases in peripheral blood flow as reperfusion of the ischemic tissues starts (see Figure 15-4). The extremities characteristically change color from white to mottled pale blue (see Figure 15-1), although pigmented skin may obscure these color changes (see Figure 15-5), while remaining cold and numb so that the individual may be unable to walk or maintain balance. The loss of sensory and motor function is often related to a pattern of anesthesia to pain, touch, and temperature, in any combination. Initial edema or swelling, or both, of the extremities is sometimes observed during this stage, and the peripheral arteries are often impalpable. Clinical assessment of neuromuscular function usually indicates an absence of sensory action potentials and a lengthening of distal motor latencies.

Third Stage: Hyperemia

The third stage of NFCI, hyperemia, in which the affected extremity receives increased blood flow,

may last for a few days to a few months, usually from 6 to 10 weeks in fully developed cases.

The third stage is quite distinct from the previous stages, and its onset is abrupt. During this stage the affected extremity becomes hot and flushed, with obvious redness (see Figures 15-2 through 15-6); these are among the most prominent signs of the progression of NFCI. The previously weak peripheral pulse changes to become full and bounding. The microcirculation appears sluggish, however, as illustrated by simple tests for capillary refill. A fingertip pressed onto the area to blanch the underlying skin will leave a white spot that persists for many seconds instead of disappearing rapidly (see Figure 15-7).

Anesthesia that was seen at stage two typically resolves during stage three. Intense pain is usually reported from the affected extremities, including hyperalgesia to the slightest touch. The persistence of the pain during the third and fourth stages, despite little overt tissue damage, is another outstanding and characteristic symptom of this injury. Many casualties report that the pain is worse at night, and it may deprive them of sleep. When NFCI occurs in the feet, pain is normally localized in the sole of the foot, typically across the base of the metatarsals, and may mimic metatarsalgia. Attempts to relieve the pain using conventional analgesics and antiinflammatory agents are usually completely unsuccessful; parenteral morphine merely removes the displeasure of painful sensation, but regional analgesia using local anesthetic may bring complete (if short-term) relief. Anesthesia in the most distal areas of the exposed extremities may still persist, however, in the face of this pain.

Edema frequently becomes obvious during this stage and in the most severely injured casualties, blisters containing serous or bloody fluid may form. In the rare cases severe enough to have suffered gross tissue damage, areas of skin may start to declare their nonviability before becoming overtly gangrenous in the next stage. Anhydrosis is often present at this time. This anhydrosis is not entirely predictable nor is its location, but often it is observed to occur in areas associated with sensory loss, particularly of warm sensation. Reflex vasomotor activity to both hot and cold stimuli is often absent. Clinical examination may demonstrate some loss of neuromuscular function and general muscle weakness. Diminished electrical excitability of the muscle has also been recognized during this stage.[11,12]

Fourth Stage: Following Hyperemia

The final stage of NFCI may last for weeks to months, in some individuals for years, and in some apparently for the remainder of their lives. However, stage four is remarkable for its lack of obvious physical signs. (Figure 15-8).

During this stage the previously observed inflammatory responses are usually reduced and limb temperature falls. The affected distal extremities remain cold, however, and demonstrate an increased sensitivity to cold stimuli, with prolonged vasoconstriction, in more than 60% of cases of NFCI.[3] Although milder cases may demonstrate normal vascular and vasoconstrictive tone at this stage, when any environmental or spontaneous event starts to cool the limbs, they may remain cold for many hours after removal of the initial stimulus. In some cases the injured extremities show a permanent temperature sensitivity. Usually these increased sensitivities are only in response to cold stresses, but rarely they may also be to heat. The long-lasting increase in temperature sensitivity, or "cold sensitization," is another prominent component in the progression of NFCI.

Persistent pain, often triggered by cold exposure and associated with vasoconstriction, has been reported in more than 70% of cases of NFCI and may be the dominant symptom.[3] Small areas of numbness may remain in perpetuity, although more substantial lasting sensory loss is unusual. Hyperhydrosis is found in a significant but smaller proportion of cases, and may provide a fertile environment for recurrent fungal infection. Such excessive sweating may be prompted by exposure to thermal stimulation, but interestingly, it may also often result from emotion. Shedding of the nail in affected digits is less common still, may become recurrent, and can progress to disturbances of nail growth such as onychogryphosis. The pathophysiological processes of the most severely affected casualties, who have dying tissues, will evolve similarly to those with gangrene from freezing cold injury (FCI; see Chapter 14, Clinical Aspects of Freezing Cold Injury), and all the long-term sequelae that have been reported following freezing injury can occur in casualties with NFCI alone.

The clinical investigation of fourth-stage NFCI is obviously of considerable interest and is discussed later in this chapter.

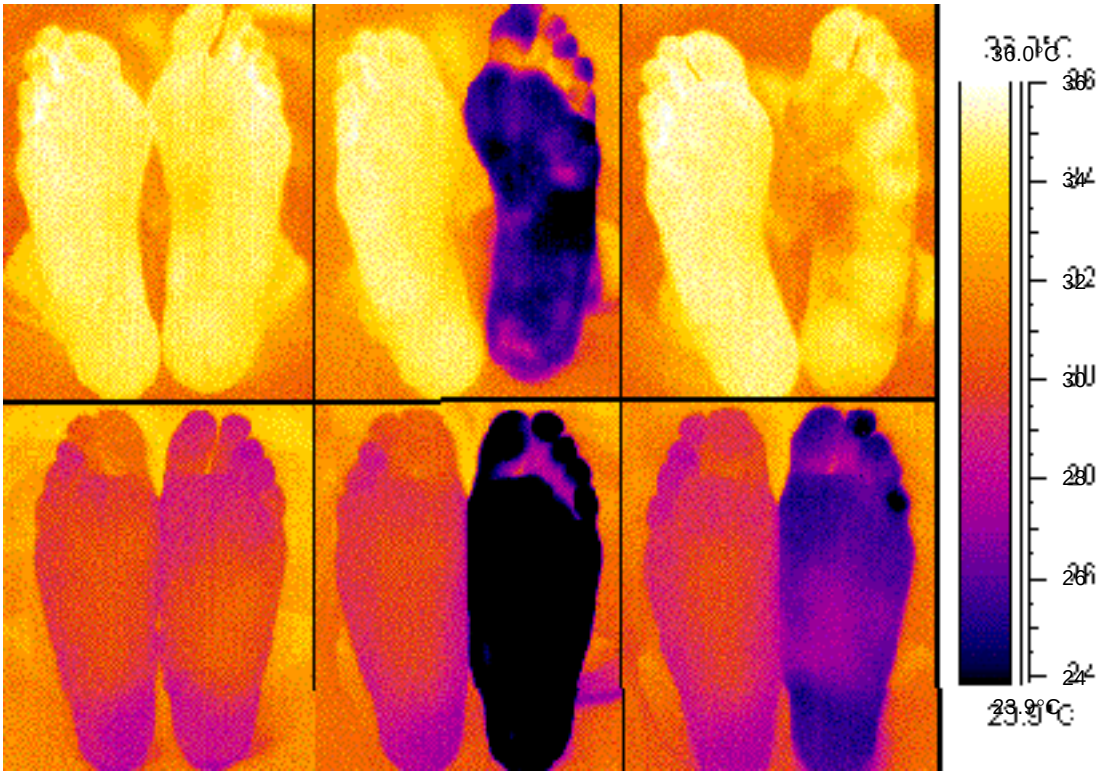

Fig. 15-8. Infrared thermography in the assessment of the consequences of nonfreezing cold injury. The upper sequence of three images was taken from an uninjured, asymptomatic control; the lower sequence from a patient who had sustained nonfreezing cold injury and was subsequently complaining of sensitivity to the cold. In both control and patient, the first (left) image was taken after resting in an ambient air temperature of 30°C. The second (center) image was taken immediately after the foot had been immersed in water at 15°C for 2 minutes. The final (right) image was taken 5 minutes after removal from the water, again in 30°C air.
The upper series shows feet that were warm at rest, which rewarmed briskly after mild cold stress, recovering almost completely within 5 minutes after removal from the water. The lower series shows a severe degree of cold sensitization: the feet were much colder than the surrounding air at rest, and once cooled, took a long time to rewarm, remaining much cooler than the control foot at 5 minutes after immersion. The scale at far right indicates the color–temperature relationship.

ETIOLOGY AND PATHOGENESIS OF NONFREEZING COLD INJURY

Although there is evidence that other tissues become damaged in nonfreezing cold injury, the site of primary injury appears to be the neuro-endothelio-muscular components in the walls of local blood vessels. Assumed for many decades to be a relatively simple system, more recent work on the modulation of neural control and non-neural regulation has shown that there are very sophisticated and interrelated regulatory mechanisms, any or all of which could be influenced by cold exposure and become disrupted as a consequence.

Vascular and Circulatory Pathology

The present view is that NFCI is brought about by intense and prolonged cold-induced peripheral vasoconstriction. Although contributions from direct cold effects on neural function and from reperfusion following ischemia often complicate the injury development, the majority of clinical and experimental studies to date suggest that intense vascular activity is an initial and most significant feature in the etiology of NFCI.

In addition to the present view regarding the potential role of cold-induced *vasoconstriction* in the etiology of the injury, clinical and experimental studies are consistent in showing that cold-induced *vasodilation* (CIVD) often disappears quite early during initial cold exposure. The observed elimination of the CIVD response indicates the profound effect prolonged peripheral vasoconstriction may have in the development of NFCI. The loss of control over peripheral vasodilation may be one of the early mechanisms involved in the onset of NFCI. Recent research has confirmed experimentally the reduction or elimination of CIVD during prolonged cold exposure.[16,17]

Francis and Golden[1] have presented an excellent summary of the etiological factors related to the development of NFCI and an overall conceptualization of the interrelationship among those factors. An updated schematic description of their conceptualization is shown in Figure 15-9. At the core of the diagram is the concept of vasoconstriction as a major modulator in the development of NFCI. Other critical etiological factors contribute to the sympathetic tone that in turn directly relates to peripheral vasoconstriction. In reviewing various classic and contemporary research articles on the pathogenesis of NFCI, Francis and Golden suggest that the most important mechanism is the "vicious circle" of cooling and vasoconstriction associated with a high level of sympathetic tone, which no longer can be affected by CIVD. They also conclude that it is now important to direct research efforts toward the prevention of NFCI and that as the central focus of any research effort on the prevention of NFCI, it is important to consider how the vicious circle can be broken.

The Roles of Circulating Vasoactive Substances

Norepinephrine, whether circulating in the peripheral blood or released from sympathetic nerve endings, induces constriction of vascular smooth muscle. It is well established that the vasoconstriction is primarily mediated by the activation of sev-

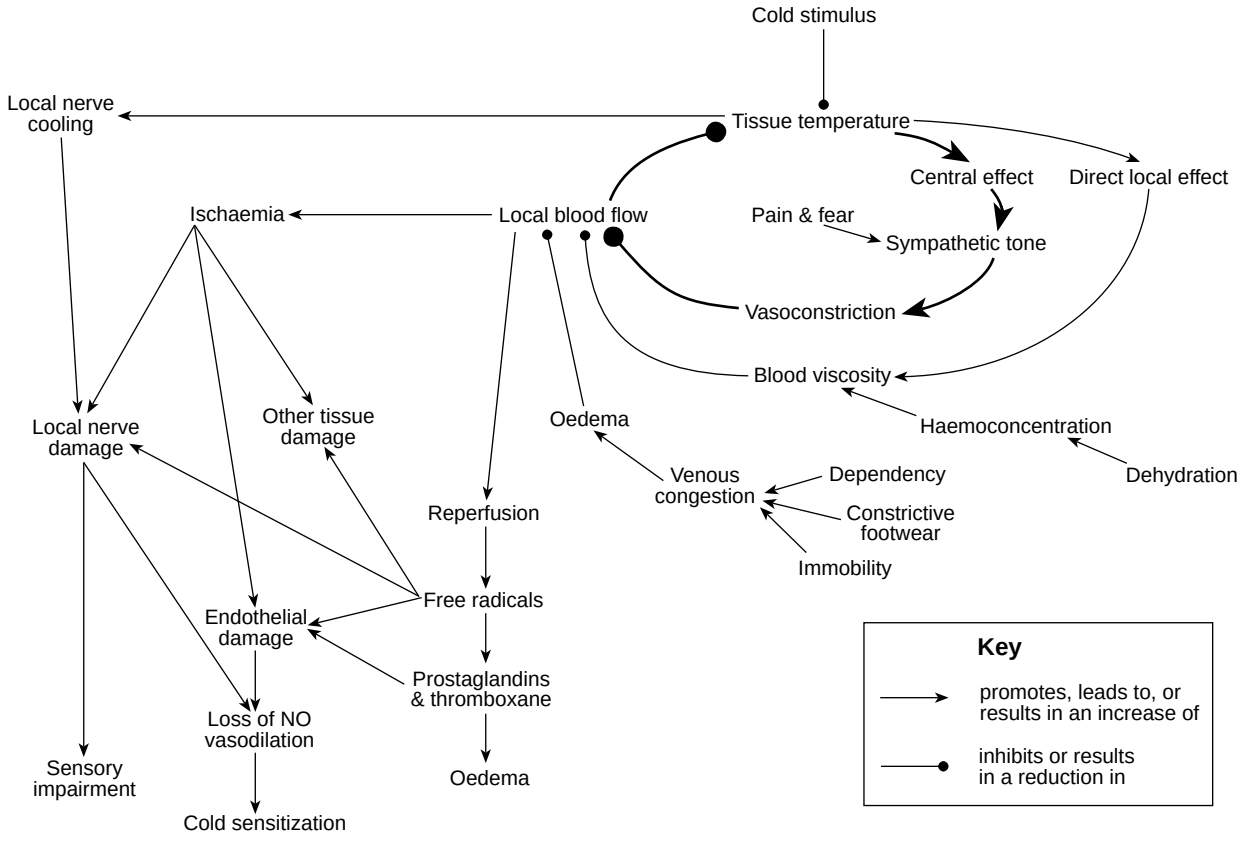

Fig. 15-9. Schematic diagram illustrating the postulated etiological factors and mechanisms of the pathogenesis of nonfreezing cold injury. Adapted and updated with permission from Francis TJR, Oakley EHN. Cold injury. In: Tooke JE, Lowe GDO, eds. *A Textbook of Vascular Medicine*. London, England: Arnold; 1996: 360.

eral subclasses of α-adrenergic receptors.[18] At present there is still lack of agreement regarding the role of specific receptor subclasses in cold-induced vasoconstriction in different tissues, but experimental evidence indicates that α_2 receptors play a prominent part.[19]

Exposure to cold temperatures has been demonstrated to stimulate the release of norepinephrine in the peripheral circulation by as much as 300%,[20,21] leading to intense and prolonged peripheral vasoconstriction. Cold-induced peripheral vasoconstriction often lasts quite long due to additional modulation of norepinephrine levels. Cold reduces the rate of uptake and metabolism of norepinephrine that results in continued vasoconstrictor stimulation.

Cold temperatures have an additional direct effect on the walls of blood vessels that contributes to prolonged vasoconstriction. The initiation of the vasoconstriction response involves an increase in the concentration of cytosolic-free calcium. Depending on tissue specifics, the calcium source can be intracellular, extracellular, or both.[22] Additionally, during exposure to cold, blood flow to the skin is reduced owing to the increase in sympathetic tone and the direct sensitizing effect of cold on the cutaneous blood vessels.[23] Cutaneous vasoconstriction reduces heat loss by redirecting blood from the vasoconstricted cutaneous vessels to deeper vessels, where more favorable heat transfer takes place. However, research indicates that although adrenergic involvement is of major importance, other neurotransmitters and peptides play perhaps more important and modulatory roles in cold-induced peripheral vasoconstriction.

Cold-Induced Ischemia

As previously mentioned, a prevalent view of NFCI is that it is a vascular neuropathy brought about primarily by intense and prolonged cold-induced peripheral vasoconstriction. Although numerous nonvascular components are obviously involved in the development and symptomatology of NFCI, alteration in peripheral blood flow in the cutaneous circulation is commonly a salient feature.[2,11,12] Landmark clinical descriptions of NFCI by Ungley, Channell, Richards, and Blackwood,[11,12] based on numerous observations, recognized the major role of cold on peripheral blood vessels in the development of NFCI. In addition to their observations indicating that intense peripheral vasoconstriction is of prime importance in the etiology of NFCI, these investigators also concluded that some of the most severe damage to minute cutaneous blood vessels occurs during the time of cold exposure. Their findings indicated that although a complex sequence of events may follow, initial vascular involvement may be paramount in the onset of NFCI.

In addition to the insults precipitated by reduced blood flow due to vasoconstriction, tissue damage is also associated with the cold-induced vasoconstriction. Recent studies[17] have observed prominent endothelial injury during cold exposure, particularly the opening of junctions between adjacent endothelial cells. Also observed during cold exposure is the adhesion of leucocytes to the small blood vessel walls and occlusion of blood vessels due to the accumulation of blood cells.[17] The pronounced microvessel constriction and reduced blood flow lead eventually to local tissue hypoxia, which, in turn, complicates the injury.

Cold-Induced Vasodilation

If, during initial exposure of an extremity to cold, a predominant response is restriction of peripheral blood flow due to sympathetic vasoconstriction, then we might expect peripheral vasoconstriction to continue—even after the temperature of the tissue reaches that of the surrounding environment. Instead, however, a complex and dynamic vascular response usually occurs. As the exposed extremity continues to cool, the peripheral circulation usually passes through a phase of cold-induced vasodilation (the CIVD response),[24] which is believed to be a protective mechanism against damage to peripheral tissues. The observed vasoconstriction-vasodilation oscillation of blood flow in the extremities exposed to cold may continue for several hours.[25] Experimental evidence is consistent with the hypothesis that the initial cold-induced vasoconstriction is produced by an enhanced smooth muscle responsiveness to norepinephrine, whereas CIVD is related to a cessation of the transmitter release from adrenergic nerve endings.[26] There is an alternative claim, however: that the vasodilation results from a cold-induced relaxation of vascular smooth muscle.[5] It is generally considered that intense cold-induced vasoconstriction results in a reduction of blood flow to the distal tissues. As the temperature of the tissues drops, sympathetic nerve conduction is interrupted and vasodilation occurs owing to the cessation of norepinephrine release. The resultant return of blood flow rewarms the tissue, nerve conduction is reestablished, norepinephrine is released, and renewed vasoconstriction occurs.[27]

As important as CIVD appears to be as a protective mechanism against damage to peripheral tissues, it is equally important that the absence of the CIVD response may be a significant component in the development of NFCI. CIVD may be considerably delayed in onset or completely abolished in the presence of high levels of vasoconstrictor tone.[28] The diminution of the CIVD response has been suggested as potentially involved in the development of NFCI.[1,2] In an animal model of NFCI following a cold injury exposure, CIVD was found to be completely absent and remained absent for several weeks, suggesting that CIVD loss is indeed associated with the development of NFCI.[16] The observed elimination of the vascular response after prolonged cold exposure may indicate the profound effect that prolonged peripheral vasoconstriction can play in the development of NFCI. Direct microvascular observations[17] have also indicated rapid loss of CIVD during cold exposure.

Neural Pathology

Clinical and experimental studies alike have indicated that cold exposure can be a direct cause of peripheral nerve injury. Nerve dysfunction and later prolonged damage to peripheral nerves and to sympathetic fibers are common components of severe NFCI. A widespread, predominantly distal degeneration of axons has been documented as a result of exposure to cold.[29] Historically, there has been general disagreement on the pathological changes in neural tissues as a consequence of NFCI, particularly evidence regarding degeneration of nerves. Early observations[30] suggested that small and unmyelinated nerve fibers were most affected by cold, and that large myelinated fibers tended to survive cold exposure. Other observers[31,32] made the opposite suggestion; that small myelinated and unmyelinated fibers tended to survive cold exposure, and large myelinated fibers have the greatest susceptibility to cold. In numerous studies, no relationship was obtained between the size of the nerve fiber and its susceptibility to cold damage. Certainly, research protocols and species differences contributed to the contradictory observations regarding cold-induced nerve injury. The weight of more-recent research[1,33] has tended to demonstrate that thick, myelinated fibers appear to be more susceptible to damage by cold. However, the full extent of fiber involvement probably depends, ultimately, on the actual duration and severity of the cold exposure. Additionally, there is consistency in studies that indicates that cold exposure results in nerve conduction blockage and associated cessation of axoplasmic transport.[33] Often nerve edema is present, and passive leakage through damaged endoneurial capillaries occurs. Although the mechanisms directly responsible for cold-induced nerve damage are far from clear, it is considered likely that ischemia plays an important role in the etiology of the nerve injury.

Some research using animal models has shown changes in muscle and blood vessels following cold exposure,[30,34] suggesting that NFCI may be related to changes in supporting vasculature leading to nerve ischemia, whereas other studies[29,35,36] have shown peripheral nerve degeneration without apparent damage to blood vessels. It appears that the mechanism of nerve damage by NFCI is complex and may depend upon the general procedure used to induce the cold injury. For example, in those experiments mentioned above,[29,35,36] in which extremities were cooled and no changes in blood vessels were observed, the animals were anesthetized during cold exposure. In other studies[30,34,37] in which blood vessel damage was reported, the animals were conscious during cold exposure. It is possible that the added stress associated with the cold exposure in awake, conscious animals may provide a component to NFCI that leads to blood vessel changes and nerve ischemia. It is important to note that nerve fiber damage is often located in proximity to vascular elements rather than to peripheral nerve areas. This may indicate that neural pathology is not always due to direct cold effects on neural tissue, per se, but can be confined to areas of cold-induced vascular dysfunction.

Stress and Nonfreezing Cold Injury

Although NFCI is clearly related to sustained exposure of extremities to a cold environment, mere cold exposure of an extremity often does not appear to be sufficient to produce the clinical symptomatology and pathology in humans. This might distinguish NFCI from other forms of local cold injury (eg, frostbite). The pathogenesis of NFCI includes not only exposure of the extremities to cold for an extended period, but often a confluence of factors that, together with cold exposure, appear to facilitate the formation of the distinctive pathology.

Evidence collected from military cold research indicates that cold injury appears to occur more

often among military personnel in combat situations than in civilians or in noncombat military personnel exposed to similar or even more severe cold weather.[4,6,7] This differential occurrence suggests that the conditions existing in warfare environments somehow interact with exposure to cold weather to potentiate NFCI. Numerous studies conducted during World War II and the Korean War suggest that combat stress was a significant contributing factor in cases of cold injury.[7,38–41] The physiological changes incurred in stressful combat conditions may alter peripheral thermoregulatory mechanisms during cold exposure and thereby promote the formation of cold injury in the extremities.[1,7] Although there is a very strong suggestion that stress exposure may influence the susceptibility to NFCI, little attention has been given to the manner in which stressful stimuli may modify physiological responses to cold.

Behavioral stressors such as cold exposure can also profoundly affect vasomotor tone.[42–44] Acute and prolonged stress resulting from exposure to combat environments results in significant peripheral vasoconstriction, particularly in the extremities.[1] Moreover, physiological reactions associated with combat stress, such as sweating, combined with intense vasomotor constriction can lead to temperature reduction, especially in the distal portions of the hands and feet—the parts of the body that are most susceptible to NFCI. Behavioral stress dramatically affects the peripheral circulation in a manner similar to that observed when an extremity is exposed to a cold environment. Changes in peripheral circulation that accompany or result from either an acute or a prolonged stress-inducing environment may worsen the physiological conditions in an exposed limb and facilitate the conditions for the development of NFCI.

Reperfusion Injury

In recent years, there has been considerable interest in molecular mechanisms of injury that occur during the reperfusion of vascular beds that have been ischemic for prolonged periods. Although the vast majority of such studies have focused on other tissues and forms of insult, Das, Iyengar, Jones, Lu, and Maity[45] have provided good evidence that the administration of agents that inhibit the generation of toxic oxygen substances can attenuate the damage caused by NFCI. This is in accord with the rationale behind slow rewarming (discussed below in Treatment of Nonfreezing Cold Injury), which is based on the observation that rapid rewarming of NFCI exacerbates the injury. Although it is fair to conclude that reperfusion injury is an important component of the etiology and pathogenesis of NFCI, it is not yet possible to compare its importance with other components.

Ischemic-Hyperemic-Sensitization Disorders

Clinically, NFCI appears to be a somewhat indistinct member of a family of disorders in which there are different combinations of ischemia followed by hyperemia and subsequent sensitization to temperature disturbance. Other conditions in this group may be barely distinguishable from NFCI and have obvious common etiological factors, or they may appear to be more distinct. For example, "shelter limb" occurred in civilians who were forced to take shelter in the deep tunnels of the London Underground during the blitz in World War II.[46] Clinical descriptions of shelter limb are indistinguishable from the characteristic stages of NFCI, so it appears to be closely related. Another condition that resembles NFCI is "paddy foot," which occurs in military personnel whose feet are immersed for prolonged periods in much warmer water (typically warmer than 24°C).[47] Remarkably, some casualties who suffered NFCI in the Falkland War went on to suffer 100% incidence of paddy foot when they were later sent to train in tropical jungles. Another interesting condition that is possibly identical to NFCI is "pink disease," in which children who sleep with their hands and feet exposed to cold room air present as apparently stage-three NFCI. It has been suggested that childhood pink disease may account for some of those fresh military recruits who seem unusually susceptible to the cold.

More-distant relatives of NFCI may include another apparently protean group of conditions now known as the reflex sympathetic dystrophies (RSDs). Although extremely variable in presentation, many RSDs follow blunt tissue trauma such as that sustained in automobile accidents, in which prolonged reduction in local nutrient flow could have occurred. Later, these patients may complain of swelling, pain, and sensitivity to temperature (warm, cold, or both). Attempts to treat RSD by sympathectomy are also variable in success. One promise of continued research into NFCI is that the results may also be of benefit in addressing these other, possibly related, conditions.

EXPERIMENTAL AND RESEARCH APPROACHES TO NONFREEZING COLD INJURY

Laboratory animal studies of NFCI have been performed in order to better understand the development and pathophysiology of the injury, which in turn, might ultimately lead to more successful prevention. Clinical human research has focused on the development of technologies to investigate salient characteristics of NFCI.

Animal Models

Numerous laboratory animal studies have focused on the constellation of dysfunctions associated with exposures to nonfreezing cold. Briefly detailed here are selected animal studies that highlight various focuses of research that have been considered central in attempting to understand the etiology of NFCI.

In one of the earliest research papers, Smith, Ritchie, and Dawson[48] exposed rabbits to wet and dry cold. These investigators reported that primary damage immediately after cold exposure was to capillary vessels, with no observed damage to nerves or muscles of the extremities. Later, an extremely influential experimental investigation of NFCI was performed by Blackwood and Russell,[30] using the rat tail as the research model. In this study there was an attempt to expose the animals to wet, cold conditions comparable in many respects to those under which NFCI develops in humans. Caged animals were systematically exposed for 24 to 96 hours to 3°C to 4°C air temperature, with 4°C to 5°C water running through and filling the cages to a depth of 1 in. The animals were then examined immediately and up to 60 days later. Following exposure, different animals were rewarmed at different rates. This study is of interest because it is one of the few that tried to mimic the conditions of human exposure and then followed the course of injury development over an extended period of time. These investigators found damage to nerve and muscle tissue after exposures of 48 hours, and the intensity of the damage increased with longer exposures. In animals examined 2 months after exposure, the nerve and muscle tissue still showed evidence of degeneration. It was observed in this study that the primary injury appeared to occur during the exposure, and that variations on the rewarming process had no systematic effect on the ultimate pathology.

Lange, Weiner, and Boyd[49] exposed the hind leg of rabbits to 3°C to 5°C cold water for 3 to 4 days. They, like Blackwood and Russell, also observed dysfunction mainly in muscular and nervous tissue. Capillary permeability increased and severe edema formed during, rather than after, exposures.

Das and colleagues[45] studied the effects of cold exposure and focused primarily on aspects of rewarming in rabbits. The legs of rabbits were exposed to a freezing mixture of ice and salt at 0°C for 20 minutes. Cellular injury during cold exposure and rewarming was monitored by estimating the release of creatine kinase and lactate dehydrogenase in plasma samples obtained from the femoral artery. These markers showed a slight increase during cooling but a marked increase during rewarming. The injury obtained in this study was believed due to the generation of oxygen-derived free radicals that may attack membrane phospholipids during rewarming, as significant breakdown of membrane phospholipids was observed. Interestingly, administration of quinacrine, a phospholipase inhibitor, preserved membrane phospholipids and reduced the release of the intracellular markers of injury.

Many of the research studies relating to NFCI have focused experimentally on the particulars of the neuropathies resulting from cold exposure. Denny-Brown and colleagues[31] investigated the effects of 0.5°C to 8°C cold exposure on the sciatic nerves of cats. This was one of the first studies that clearly demonstrated that cold exposure selectively damages the peripheral nerves of the extremities, in that large myelinated fibers were found to be more susceptible to cold-induced damage than smaller ones. These investigators demonstrated that exposures as short as 30 minutes at 8°C could produce nerve damage. The similarity of the damage to that related to vascular ischemia was noted.

Peyronnard, Pedneault, and Aguayo[29] submerged rats up to hip level in 1°C cold water for 12 hours and then examined ventro-caudal nerves at varying time periods following cold immersion. The rat tail nerves examined showed a greater loss of large myelinated fibers than of myelinated fibers of lesser diameter. Unmyelinated fiber numbers remained within the range of controls.

Experimental cold injury to the sciatic nerves of rats was studied by Nukada, Pollock, and Allpress.[33] Cooling of the nerve was accomplished by circulating 3°C cold water through copper tubing on which the exposed nerve was placed for 2 hours. They found nerve conduction block and termination of axoplasmic transport. Prominent nerve edema was observed with damaged endoneurial capillaries and

a widening of endothelial junctions. Large myelinated nerve fibers were severely damaged, whereas unmyelinated fibers were not affected. The authors of this study suggested that the nerve fiber degeneration they observed may be related to cold-induced blockage of axoplasmic transport.

Gilliatt and Kennett[50] studied the effects of cold exposure in the tibial nerves of rabbits. The hind limb was immersed up to the lower thigh in 1°C cold water for 10 to 14 hours, or the exposed tibial nerve was placed in a cooling trough maintained at 5°C for 2 to 3 hours. These investigators observed local axonal injury that eventually led to Wallerian degeneration. After 3 to 4 weeks they found loss of large myelinated fibers with no effect on nonmyelinated fibers.

The effects of cold temperature on peripheral nerve function and structure were studied in rats by Shurtleff and colleagues.[37] The tails of rats were submerged in 1°C cold water for 10 to 12 hours. Nerve action potentials and muscle action potentials in the tail were altered following cold exposure. Reductions in potential amplitudes were observed immediately after cooling, with further reductions over the week following exposure. Initial nerve damage appeared to be located just below the surface of the water, and later, in the first week after exposure, Wallerian degeneration occurred. Damage was observed in large myelinated fibers and capillaries following cold exposure. The capillaries within the endoneurium showed unusual endothelial and red blood cells, and many had occluded lumina. The pattern of damage suggested a vascular component in the injury.

The effects of cold exposure on thermal sensitivity were investigated by Ahlers and colleagues.[51] Thermal sensitivity was measured in the tails of rats before and after the tail and a portion of the hind flank were exposed to 1°C to 4°C water for 1 to 9 hours. They found that exposure of the rats to cold water for 6 to 9 hours produced anesthesia at first, followed by a heightened temperature sensitivity to a heat stimulus to the tail. Although the animals initially exhibited increased latencies in tail-withdrawal responses immediately following the cold exposure, on subsequent sessions they showed an increased sensitivity to the heat stimulus, indicated by decreased latencies in tail withdrawal. The course of alterations in thermal sensitivity obtained in this study appears broadly similar to those observed in humans with NFCI.

Thomas and colleagues[16] studied alterations in cutaneous blood flow and temperature in the tails of rats immersed in 1°C cold water for 6 to 9 hours. Following cold exposure, cold-induced vasodilation was absent and remained absent for several weeks. Cold-induced disturbances of cutaneous blood flow in the rat tail consisted of a sequence of distinctive stages, analogous to those observed in human NFCI. Immediately following cold exposure of several days, reduced blood flow and thermal sensitivity were observed, followed in a week by a hyperemic stage, and later by increased vascular and thermal sensitivity.

These selectively reviewed studies, as well as others, clearly suggest that there is a significant vascular component associated with the development of NFCI, probably directly involved with cold-induced vasoconstriction or cold-induced ischemia. The research literature also seems consistent in indicating that most neural damage occurs in larger myelinated fibers, probably resulting from a complex interaction of direct cold effects and damage to supporting neural vasculature. Generally, Wallerian degeneration occurs in the more distal parts of the affected fibers at a later time.

Although the summary of these individual and collective findings brings appreciation of much information regarding the pathology of the cold injury, it should be emphasized that most of these studies have examined the effects of application of a cold environment to a specific system injury. However, with only a few exceptions, the results of these animal studies are not necessarily directly pertinent to the unique human NFCI condition, as the majority of the studies do not indicate a discernible progression of stages following cold exposure that are analogous to the syndrome as described in humans.

Human Studies

The great majority of earlier work on the pathophysiology of NFCI in humans concentrated on the gross injuries seen during the two World Wars. Although pathological examination of amputated limbs led to detailed descriptions of such severe injuries, it (*a*) has not provided useful insight into more pervasive subtle injuries and (*b*) has produced conflicting conclusions. Because it would be manifestly unethical to experimentally induce NFCI in human subjects, more recent attention has been directed at the investigation of cases during the fourth stage, principally to gain insight into the mechanism by which the combination of cold, ischemia, and reperfusion can result in prolonged cold sensitization.

Clinical observations led Francis and Golden[1] to advance the hypothesis that cold sensitization resulted from sympathetic denervation supersensitivity, which in turn, was caused by local damage to

the sympathetic fibers innervating the peripheral vasculature. As Oakley[52] pointed out, this would imply either that NFCI was selectively affecting small and unmyelinated nerve fibers, or that those fibers were being damaged along with others but were least able to regenerate successfully. His results from the measurement of thermal sensory thresholds confirmed the relative sparing of larger and myelinated fibers, in direct conflict with most observations in animal experimental models. In a later review, Francis and Oakley[3] highlighted the many conflicts raised by the hypothesis of denervation supersensitivity.

The next step was to use more careful experimental measurements of skin blood flow, obtained using modern laser Doppler rheometry, to study the altered pattern of local vascular responses. Initial results suggested that there was no evidence to support the existence of sympathetic denervation supersensitivity, but that there may have been an underlying abnormality of the local vascular endothelium.[48] However, most recent work has shown that responses to a wide range of maneuvers are normal despite gross cold sensitization; this makes it extremely unlikely that there is any significant lasting nerve damage, and rules out Francis and Golden's hypothesis. The only significant and consistent abnormalities found in the microcirculation of those with stage-four NFCI were (*a*) greatly reduced resting flows and (*b*) exaggerated responses to a cold stress. Both "on" and "off" responses to maneuvers believed to be modulated by local sympathetic action were unaffected by injury. Future studies are intended to observe the effects of iontophoresis of various vasoactive agents into the distal extremities of those with cold sensitization.

Synthesis of these findings is difficult, particularly as such a profound vascular consequence appears to have little physical evidence to account for it. There are two additional mechanisms that have not yet been studied in humans or animals. The first is that early sympathetic and endothelial disruption is followed by regeneration and peripheral recovery, but that higher control mechanisms are altered during this period to produce the characteristic sensitivity to cold. The second is that early responses to the injury result in a transient physiological sensitivity to the cold, possibly as a result of sympathetic injury, which results in a reduction in the number of vessels in the microcirculation—the opposite of the vascular proliferation seen during physical training and heat exposure. Either process could then be self-reinforcing through Francis and Golden's vicious circle of cooling.[1]

A range of different techniques have been used to investigate fourth-stage NFCI, of which infrared thermographic assessment of sensitivity to the cold and the measurement of thermal sensory thresholds appear to be the most useful. Infrared thermography was introduced in the United Kingdom by Golden, during his studies of veterans of the Falkland War in 1982, and was later taken up in the United States by Hamlet. Two completely different protocols have developed as a result. In the United Kingdom, patients are subjected to a local standardized mild cold stress (currently, 2-min immersion in a waterbath at 15°C) after the rest of the body has become accustomed to a warm air temperature (30°C). Thermograms taken immediately prior to, immediately following, and 5 minutes after removal of the hand or foot from the water are then used to assess resting limb temperature and the speed of recovery of that temperature (see Figure 15-8). By contrast, in the United States, the protocol attempts to assess CIVD in a limb exposed to more-prolonged and -severe cold exposure. British experience with the latter approach was discouraging, owing to the difficulty in reproducing CIVD responses even in ostensibly normal subjects.

More recently, Oakley and Lloyd[53] started using the Middlesex Hospital Thermal Testing System, a simple automated process designed to measure warm and cool sensory thresholds in patients with diabetic neuropathy.[52] They found that unrecordably high warm sensory thresholds, more than six Centigrade degrees above the skin temperature of the affected body part, were extremely common in patients with NFCI although never found in healthy controls. Although not a directly relevant assessment of cold sensitization, unlike thermographic assessment, this automated process is now in routine use as a discriminatory test. Oakley[54] has also measured skin blood flow in affected limbs using laser Doppler systems, but this is time-consuming and normally contributes little that could not be obtained from thermography.

TREATMENT OF NONFREEZING COLD INJURY

Currently, treatment consists of the application of fairly obvious and simple interventions. Research may yet provide leads to more specific, no doubt pharmacological, interventions.

Hypothermia

If an individual who has developed NFCI has been exposed to a cold environment long enough, there is always the possible complication of hypothermia. The appearance of hypothermia is a medical emergency and may make the diagnosis and treatment of local cold injury more difficult. A major concern if hypothermia is present is modulation of the treatment relating to rewarming of the individual. NFCI requires a very gradual rewarming of the injured extremities, while hypothermia may require rewarming of the body as rapidly as is possible. Owing to the potential life-threatening aspects of hypothermia, treatment of that condition takes precedence over the problem of NFCI, of course, although the extremities should be spared if NFCI is suspected.

Warming

In cases of NFCI, affected extremities should be allowed to rewarm slowly. In fact, rapid rewarming may actually intensify the injury. Often, it may be preferable during earlier stages of NFCI, if appropriate, to transfer the individual to a more comfortable rewarming site, without attempting to rewarm in the immediate cold environment.

Dehydration

If an individual has been exposed to cold for more than a day, it can usually be assumed that dehydration is present, a consequence primarily of cold diuresis. Individuals should be rehydrated as soon as possible if evidence of dehydration is apparent. Rehydration with warm liquids is an essential aspect of any rewarming regimen, and is also a major contributor to the stabilization of normal physiological processes and the modulation of critical cold-induced fluid shifts within the body. Rehydration will also contribute to more adequate blood flow in the extremities of the cold-exposed individual.

Tissue Freezing Complications

Clinical evidence suggests that in severe cold exposures, the tissues of an individual may have passed through cycles of frostbite in addition to exposure to conditions that induce NFCI. In such cases, careful diagnosis as well as attention to the specifics of combined injury treatment are required.

Drug Treatment

At present, no known medications have been specifically identified as appropriate for the treatment of NFCI once the critical exposure duration has been reached. Because of the known involvement of α-adrenergic receptors in the control of peripheral limb circulation and the observed noradrenergic sensitization of individuals with NFCI,[1] it was believed previously that administration of α-adrenergic blocking agents or general vasodilators would be beneficial. However, this approach to treating NFCI is of little help. Presently, the most that pharmacological intervention can offer a casualty with NFCI is a reduction of the symptoms related to persistent pain.

Physical Therapy

If the individual's condition warrants such treatment and the facilities are available, physical therapy can be beneficial in the treatment of NFCI. Physical therapy during the early stages of NFCI can help maintain adequate joint articulation in cold-exposed limbs. Exercises of the legs, ankles, and feet may be of immense treatment support. If physical therapy is started during the early stages of NFCI, then it is usually indicated that it be continued through the later stages.

Pain Treatment

Pain is often one of the most enduring features associated with NFCI and can persist throughout the later stages. Unfortunately, the treatment of this pain remains controversial. As described earlier, conventional approaches have been singularly unsuccessful, and cannot be recommended. In a pilot study in 1980 and 1981, Oakley used quinine salts (200–300 mg, given at night), which appeared more successful than regular analgesics, although others since have denied that they are of use. Since 1982, the standard treatment in the armed forces of the United Kingdom, first proposed by Riddell,[55] has been amitriptyline hydrochloride, in doses of 50 or 100 mg given at bedtime. Incremental increases in dosage may be required with both drugs if pain "breaks through" after initial relief.[3]

Sympathectomy

Surgical or pharmacological disruption of the regional sympathetic nerve supply has long

been advocated for the treatment of increased sensitivity to the cold and persistent pain. However, the rationale behind this remains elusive, and there is a dearth of reported studies that use controls or follow treated groups for sufficient periods of time. In recent experience, sympathectomy may give short-term improvement in local perfusion and attenuate increased sensitivity to the cold. Some subjects also report impressive pain relief. But by 6 months later, these signs and symptoms have usually returned to their previous severity, if not actually worsened. No form of sympathectomy can therefore be recommended as having any lasting clinical merit, and clinicians should avoid becoming enthused by initial responses to trials of nerve blocks.

PHARMACOLOGICAL RESEARCH CONSIDERATIONS

Although at present there are no pharmacological interventions that are directly applicable to the prevention of NFCI, recent research related to vascular and neural pharmacological considerations certainly suggest areas that appear promising for concentrated research efforts.

Vascular Considerations

The evidence presented here supports a view of the importance of prolonged cold-induced peripheral vasoconstriction in the etiology of NFCI. The critical events that initiate the injury appear to occur during the time of cold exposure rather than following it, although there are obvious complications in the injury development from reperfusion following ischemia and from the direct effects of cold on neural function. The existing clinical and experimental observations on NFCI indicate that the magnitude and duration of cold-induced vasoconstriction are often far more intense than might be accounted for by sympathetic nerve activity, as reflected by adrenergic activation. As discussed earlier, adrenergic blocking agents and vasodilators have had little success in the prevention or treatment of NFCI. It is apparent that other major factors contribute to profound cold-induced vasoconstriction besides the vasoactivity mediated by known adrenergic involvement.

In this regard, several vasoactive peptides have been identified in the peripheral nervous system; they possess (*a*) some of the most profound vasoconstrictor activity known in humans and (*b*) the capability to interact with adrenergic activity. The vasoconstrictor activity of these vasoactive peptides occurs through their own receptors as well as through synergistic interactions with the adrenergic system. That is, the vascular activity induced by norepinephrine may be potentiated by enhanced circulating or local concentrations of these peptides. More important, these peptides are now known to be released in response not only to cold environments but also to other stressful environments.

Neuropeptide Y is a 36-amino acid peptide neurotransmitter[56] with a wide distribution in the peripheral nervous system.[57–59] The actions of neuropeptide Y at sympathetic neuroeffector junctions have occasioned numerous studies, owing to the prevalence of the peptide in sympathetic nerves and its coexistence with norepinephrine in these nerves.[58] At least three important effects of neuropeptide Y have been demonstrated at sympathetic neuroeffector junctions[60–62]:

1. a direct postjunction response, such as that of vasoconstriction;
2. a postjunctional potentiating effect of norepinephrine-evoked vasoconstriction; and
3. a prejunctional suppression of stimulated norepinephrine release.

It has been suggested that the vasoconstrictor activity of neuropeptide Y becomes more important during situations of high sympathetic nerve activity, such as exposure to cold.[63] Increased sympathetic nerve activity, such as that due to cold, is accompanied by both adrenergic desensitization and increased neuropeptide Y release.[63,64] Thus, neuropeptide Y may restore lost responsiveness to norepinephrine, and neuropeptide Y may also become a more efficacious vasoconstrictor agent. Indeed, neuropeptide Y is capable of constricting small arteries enough to produce total ischemia.[65]

In a study in which rats were exposed to 4°C water for 10 minutes, circulating neuropeptide Y levels were increased by 300% and remained elevated for 30 minutes following the rats' removal from the cold.[66] This study demonstrates the long-lasting effects of neuropeptide Y that have been associated with its extended vasoconstrictor response. More importantly, this study found that during chronic exposure, neuropeptide Y effects did not become habituated, as is usually found for catecholamine responses, but actually increased in magnitude. Additionally, it has been observed that central administration of neuropeptide Y can induce profound peripheral vasoconstriction, demonstrat-

ing that central neuropeptide Y release may be an important contributor to the stress-induced enhancement of NFCI.

Endothelin 1, one of the isoforms of endothelin, has been shown[67,68] to be a highly effective vasoconstrictor that evokes potent and long-lasting constriction effects. Other forms of the endothelin peptide are now known to possess vasoactive activity; however, the vasoconstriction potency of endothelin 1 is reported to be 20-fold greater than that of norepinephrine, making it one of the most potent human vasoconstrictors known.[69] Relevant to the development of NFCI, increased release of endothelin 1 has been observed in humans during cold stress.[70] Importantly, endothelin 1 has been demonstrated to interact synergistically with neuropeptide Y to induce rapid, intense, and long-lasting vasoconstriction.[71] More relevant to the etiology of NFCI, combinations of increased sympathetic tone, increased circulating endothelin 1, and increased circulating neuropeptide Y levels can promote increased expression of prolonged α-adrenergic receptor and neuropeptide Y-mediated regional vasoconstriction in altered conditions, such as those induced by cold.[71]

In addition, an important role in vasoconstriction induced by cold exposure has been identified for 5-hydroxytryptamine (ie, seratonin).[72] In particular, the vasoactive response of 5-hydroxytryptamine to cooling appears to be mediated by the 5-hydroxytryptamine$_2$ receptor.[72,73] The role of 5-hydroxytryptamine$_2$ receptors in cold-induced peripheral contractions has been clearly identified in humans.[72] The contribution of the release of 5-hydroxytryptamine during cold exposure is potentially even more important in cold-induced vasoconstriction, because 5-hydroxytryptamine and neuropeptide Y have been shown to interact synergistically to induce significant vasoconstriction.[71] Also, like endothelin 1, 5-hydroxytryptamine can induce vasoconstriction by neuropeptide Y with concentrations of neuropeptide Y that alone do not produce constriction.[71]

One of the most important sequence of events in the etiology of NFCI appears to be related to the initial changes induced by intense and long-lasting peripheral vasoactivity during cold exposure. It would appear that one of the most appropriate places to try to break the vicious circle of cooling and vasoconstriction described by Francis and Golden[1] is at the initiation of cold-induced vasoconstriction. Such an approach would be consistent with evidence from both clinical and experimental studies indicating the paramount importance of severe peripheral vasoconstriction in NFCI; this approach also indicates that biotechnologies that focus on the alleviation of intense cold-induced vasoconstriction could be important in preventing NFCI. An understanding of (*a*) the role of the vasoactive peptides, such as those discussed above, that are released during cold exposure; and (*b*) their vasoconstrictor effects as mediated through their own receptors, as well as through synergistic interactions among themselves and with the adrenergic system, may lead to the development of technologies that prevent NFCI.

Relevant to this consideration, specific antagonists for these potent cold-induced vasoactive peptides are now available, making pharmacological intervention possible that would completely or partially block cold-induced vasoconstriction activity. These antagonists offer a promising class of agents to be examined for potential prevention and treatment of NFCI. Current knowledge suggests that such antagonists could, in theory, modulate the role of the powerful endogenous vasoconstrictors that are released during stressful environmental conditions and, in turn, control their potential contribution to the development of NFCI.

Neural Considerations

In view of the cold-induced neuropathies consistently observed in NFCI, it would be of benefit to military medicine to attempt to develop prevention or therapy for maximal neuronal recovery. Among many current candidates for investigation, recent research has identified new neurotrophic factors related to the endogenous polypeptide, nerve growth factor, that have shown significant promise in promoting survival of damaged neurons that have been put at risk. Nerve growth factor and other related factors are members of a class of polypeptide neurotrophic factors that are involved in the development, survival, and maintenance of neuronal tissues. Consideration should be given to this class of factors, as nerve growth factor has been used successfully in treating a variety of neuropathies, including toxic drug-induced neuropathies due to taxol and cisplatin (two antitumor drugs) as well as diabetic neuropathy.[74,75] While not all neuropathies are alike, critical elements found in diabetic and drug-induced neuropathies may be similar to those found in NFCI-induced neuropathy. These similarities include impaired axonal transport, decreased sensory amplitudes, and modified thermal sensitivity. As NFCI shares many characteristics with these successfully treated neuropathies, it is possible that research with nerve growth factor may offer a viable means to prevent or ameliorate its associated neural dysfunction.

Nerve growth factor appears to be important in keeping neurons healthy and provides a first line of defense against environmental insult.[76] Critical to this function is the binding of nerve growth factor to receptors that promote phosphorylation of cellular proteins.[77,78] Nerve growth factor may play a vital role in ensuring that microtubules remain in proper assembled configuration for axonal transport, considered as a related dysfunction in NFCI, by interacting with microtubule-associated proteins. Nerve growth factor might also be potentially applicable to the prevention or treatment of NFCI because of its abilities to counteract the generation of free radicals that may occur during reperfusion and to maintain proper ion gradients across the cell membrane. As neurotrophic factors are intricately involved in the initial events of nerve regeneration, they appear to be a promising class of pharmacological agents for further consideration for potential treatment of NFCI.

PROTECTION AGAINST AND PREVENTION OF NONFREEZING COLD INJURY

Protection against exposure to cold, wet, stressful environments is surely the first step in preventing nonfreezing cold injuries. So simple a remedy is not necessarily possible during military campaigns, however, as constrictive clothing and lack of protective equipment, sleep, and nutritious food, combined with the tactical situation and a cold, wet, inhospitable environment, can predispose susceptible combatants to debilitating injury. Campaigns may have been lost before, and could be again, not because of enemy action but because of attrition from NFCI.

Protection Against Cold, Wet, and Stress

As prolonged exposure to cold, with or without concurrent wet or stressful environments, is an essential condition for induction of NFCI, it is obvious that any practice that limits exposure of an individual to the cold environment will reduce the probability of developing NFCI-or any other cold-induced injury.

Adequate thermal protective clothing is necessary for maintaining general body warmth, however, as it is the extremities that are most susceptible to NFCI; extra care must be taken to maintain local thermal protection, and in the more proximal sections of limbs through which arterial blood must flow to reach the distal extremities. As adequate maintenance of local blood flow is of prime importance in protecting against NFCI, protective gear should be not only of appropriate thermal insulation value but also nonconstrictive. This is particularly true for footwear, as constriction of blood flow in the feet certainly can hasten the development of NFCI. In this regard, it is also important that an individual remain mobile to ensure optimal circulation in the extremities.

There are extreme individual differences in the susceptibility to cold injury that are related to an individual's vascular reactions to the cold environment. Individuals capable of maintaining higher local blood flow and skin temperatures are often less susceptible to cold injury, given the same environmental conditions, than those with lower peripheral blood flow and skin temperatures. Consequently, great caution must be exercised when reexposing casualties who have had prior cold injuries, especially if there is any evidence that they suffer from residual cold sensitization.

The onset of NFCI can be hastened if individuals are malnourished and even moderately dehydrated while they are exposed to severe cold environments. Fatigue is also frequently a precipitating factor promoting onset of NFCI. Military personnel, especially commanders, should continually be aware of the ongoing physical stresses imposed by the surrounding terrain, particularly when it begins to induce a state of fatigue.

Finally, in consideration of factors that may promote the development of NFCI, medical officers should recognize that the stress associated with military operations and the vasoconstrictive effects that such stress induces—particularly stresses related to combat environments—seem to be among the more significant etiological factors. In this regard, it is important that education and training for troops to be deployed into cold operational environments recognize the constellation of precipitating factors that may lead to the onset of NFCI. Preventive education and training for military personnel will help preclude the onset of cold-induced injuries by making troops more aware of cold-related symptoms and the factors that are important in protecting against such problems. Medical personnel training for recognition of signs and symptoms of NFCI is important, as is doctrine relating to the care and rewarming of extremities. Medical personnel should be specifically targeted for intensive training in cold-related symptom identification and treatment.

EXHIBIT 15-2

A NEAR MISS IN THE FALKLANDS

The Falkland Islands are an isolated and bleak archipelago of peat and rock outcrops situated in the South Atlantic ocean between Tierra del Fuego, the stormy and cold extreme of South America, and the solitary near-Antarctic wilderness of South Georgia. Almost devoid of trees, roads, and human population, their climate is cold, wet, and windy; their weather ever-changing. As the British Task Force sailed south in 1982, planners back in the United Kingdom and medical officers embarked with the troops were only too aware that nonfreezing cold injury (NFCI) was likely to be a major problem.

Following the initial naval units, which secured sea access to the islands and engaged the Fuerza Aerea Argentina (Argentinian Air Force), the first wave of land forces consisted of three Royal Marines commando regiments and two Parachute battalions, all of whom were landed in a huge fjord-like inlet on 21 May 1982. The best-trained infantry units in the United Kingdom, many had only recently returned from arctic-warfare training in Norway, and all were considered to be highly experienced and well equipped, with high standards of fieldcraft. For many, the slippery slope to cold injury started on D day, when they became soaked to the waist as their landing craft were unable to reach dry land in the difficult beaches around San Carlos Water.

Once ashore, they discovered that digging defensive positions in the sodden peat only ensured that their feet would remain permanently wet. Whether they remained in those positions guarding the beachhead, or moved forward over the coming days as the advance built toward Stanley, most found that their footwear would not dry out again. They were using a wide variety of different boots: most had the traditional, short direct molded sole (DMS) model, of thin, lightly proofed leather (then the standard infantry boot of the United Kingdom); one commando had high-quality trial versions of its immediate successor, the combat high boot, which ran higher up the leg and was constructed to be more waterproof; others tried traditional reversed-grain leather mountaineering boots, which they had been using in Norway; there were also modern two-component plastic mountaineering boots, cross-country skiing boots, rubber Wellingtons, and more. Some wore a single pair of tough nylon socks, others two pairs of thick loopstitch "Arctic" socks, and clothing drawn from the full United Kingdom military range of the time, with many civilian enhancements.

By the second day after D day, casualties started to arrive at the 2nd- and 3rd-echelon medical support facility at Ajax Bay with a primary initial diagnosis of NFCI. All cases among United Kingdom troops were mild, with no overt tissue damage, but most were unable to sleep because of the pain, or to don their boots again because of the swelling. Most casualties had carried out routine foot care but had neither been resupplied with dry socks nor rotated into locations where they could dry out.

Although only 109 casualties reached 2nd- and 3rd-echelon medical support, they accounted for 14% of all British nonfatal casualties, and later examination of some of those returning to the United Kingdom revealed that 98% of the troops at the front line—the rifle or fighting companies—had suffered NFCI during the 4 weeks that they had been ashore. In contrast, incidence rates among logistical-support and rear-echelon personnel were almost 0%. Casualties with NFCI had clogged the evacuation chain, requiring scarce helicopters for their return to receive medical care. Once back there, they filled precious accommodation in the tiny settlements of the Falklands. Some were billeted on board logistical support vessels, and a few were further injured on board RFA *Sir Galahad* when she was hit off Fitzroy. Although they had not lost limbs or been otherwise mutilated by war, no more than a handful became fit enough to return to the frontline during the conflict.

The arrival of the Gurkhas, Scots, and Welsh Guards on 30 May 1982 still did not provide sufficient manpower for Divisional Command to start rotating troops. Concern grew that so many of those at the front line were unable to wear their boots, and that the steady trickle of cases becoming *hors de combat* was decimating field strength to the point that the final assault on Stanley could not be mounted. If the Argentine surrender had been delayed by several weeks, it is entirely possible that attrition of the British force due to NFCI would have been so great that a successful conclusion to the Falklands War would have been impossible. Among other factors, it was the operational cancer of NFCI that forced the pace toward the end of the conflict. Some 2,000 fit, well-trained, well-equipped infantry soldiers had succumbed to the same condition that had crippled their antecedents on the Somme in the fall of 1916. The poor bloody infantry had again rediscovered NFCI.

PREVENTION OF NONFREEZING COLD INJURIES IN MILITARY OPERATIONAL ENVIRONMENTS

At present, the most effective prevention of NFCI is simply to prevent prolonged exposure to cold. However, because of the complexities of military operations, and the confluence of factors that may lead to NFCI, such limitation to environmental exposure may be impossible to achieve. Additionally, operational considerations may preclude certain limitations on cold exposure. At the least, however, systematic rotation of personnel out of exposure environments or out of combat environments should be made a high priority in the planning of military operations. Undoubtedly, limitations on the duration of personnel exposure remain the main effective measure against NFCI.[4]

Efforts related to the development of protective equipment (such as vapor barrier boots) and concentrated training of personnel in hygienic foot procedures has helped reduce the incidence of NFCI.[4] However, footwear designed to eliminate the ingress of water can act as a double-edged sword by preventing the egress of liquid from the boot, where the latter is generated as sweat or results from leaks. Unless this moisture can be removed and dry socks donned, impervious footwear such as vapor barrier boots and vapor permeable layers worn within the boot can actually induce NFCI. The use of such highly protective assemblies therefore requires that wearers are able to carry out frequent foot-care routines and change into dry socks. These are often not possible. Related to foot hygiene are requirements that feet be kept warm and dry and not be allowed to remain motionless for lengthy periods. Constricting footwear, owing to its restrictive effects on blood flow within the foot, can also contribute to the development of NFCI.

Although strict adherence to these measures can be useful, in the realities of military operations and of combat in cold environments, application of comprehensive preventive measures has proven to be quite difficult, resulting in significant occurrence of cold injury.[79] For example, due to circumstances of combat, many of the British NFCI casualties in the Falkland War were Royal Marines who were arctic trained and maintained as high standards of foot care as were possible (Exhibit 15-2).[4]

Consequences of Nonfreezing Cold Injury as a Learned Disorder

An anecdotal account of the effects of military reassignment of patients with NFCI has suggested that cold sensitization, and possibly the other problems encountered in stage four, is extremely unusual in those who are sent to live in a tropical location soon after the end of stage three. Oakley[80] suggested that cold sensitization and other sequelae may be maladaptive responses in fundamentally normal neurovascular beds; coupled with that, he proposed that relocation to the tropics be made part of the formal treatment regime. There is already supporting evidence from a trial of conditioning[81] that this proposal is worth investigating. Even if it does not prove successful as a means of treating those with established NFCI, by preventing casualties from sustaining further damaging cold injury, it can only be beneficial.

SUMMARY

NFCI remains a significant medical problem in military operations performed in cold weather. When military operations must be carried out in cold and wet environments for more than a very limited time period, the probability is high that NFCI will occur. Practical operational considerations often do and will continue to preclude optimal prevention efforts based on limiting exposure of personnel. In more than half a century of research related to the occurrence of this injury in military environments, no other proven prevention strategies for NFCI have been found.

Over the past decades our understanding of the mechanisms and systems associated with the pathophysiology of vascular damage, neural dysfunction, combat stress, and reperfusion injury has been greatly enhanced through research in areas not directly related to cold-induced injuries. However, medical research focused on the sequential progression of events involved in cold-induced modulation of those mechanisms and systems is lacking. To safeguard the health, safety, and ability of military personnel to successfully complete their missions while working in cold environments, it is imperative that more attention be focused on elucidating pharmacological and physiological strategies for breaking the core of the vicious circle of peripheral cooling, prolonged sympathetic tone, and cold-induced vasoconstriction. It is also crucial that we identify effective treatment regimens for casualties that occur when prevention efforts fail, particularly for the problems of cold sensitization and residual pain.

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Chapter 16

TREATMENT OF ACCIDENTAL HYPOTHERMIA

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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

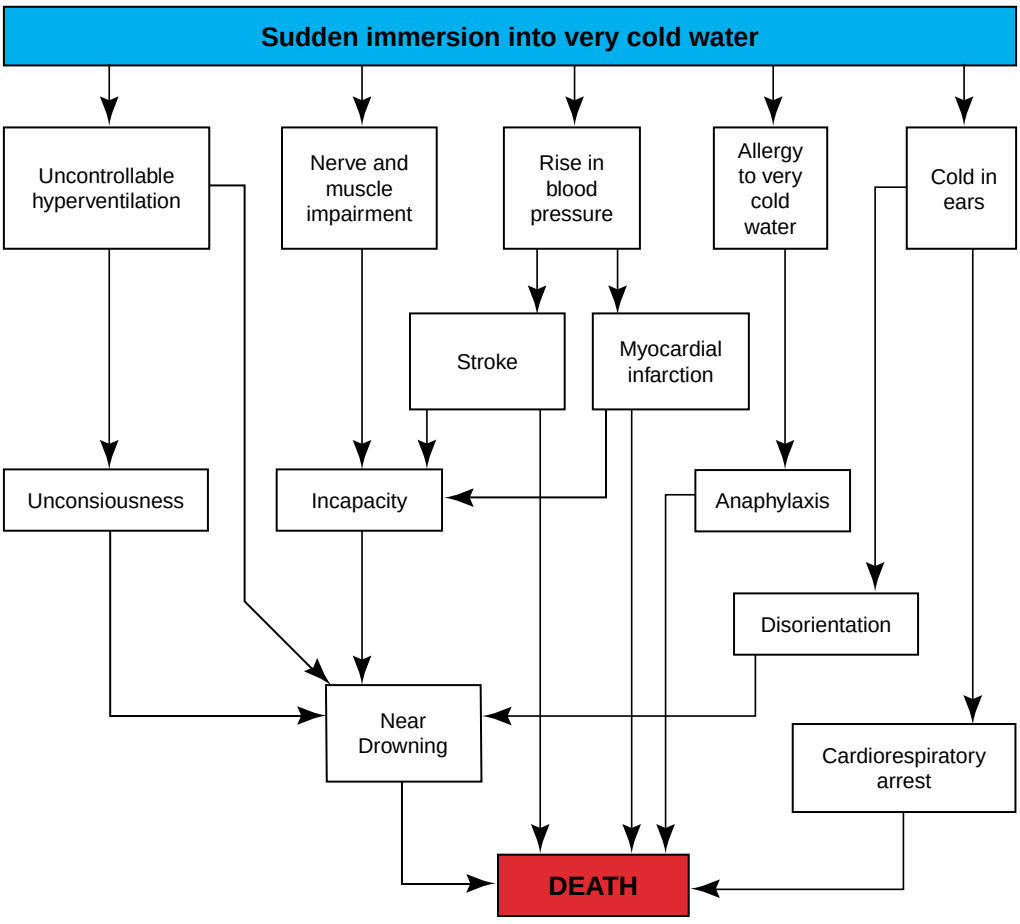

Fig. 16-1. Sudden immersion into very cold water initiates processes that can lead to death. Adapted with permission from Lloyd EL. ABC of sports medicine: Temperature and performance in cold. *Br Med J.* 1994;309:532

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

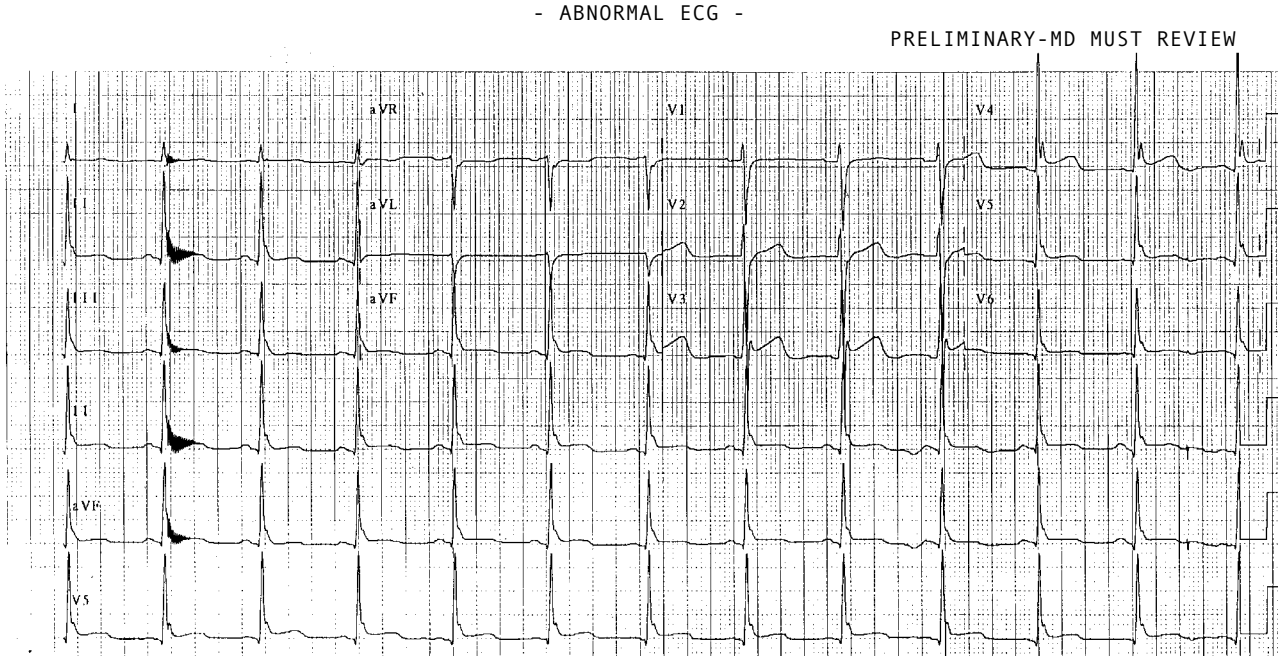

Fig. 16-2. A 12-lead electrocardiogram displaying excellent J waves (also called Osborn waves) on V4. The patient was a 32-year-old man whose core temperature at the time this electrocardiogram was made was 31°C. Note that although the computer recognized the reading as abnormal, it was not set to diagnose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

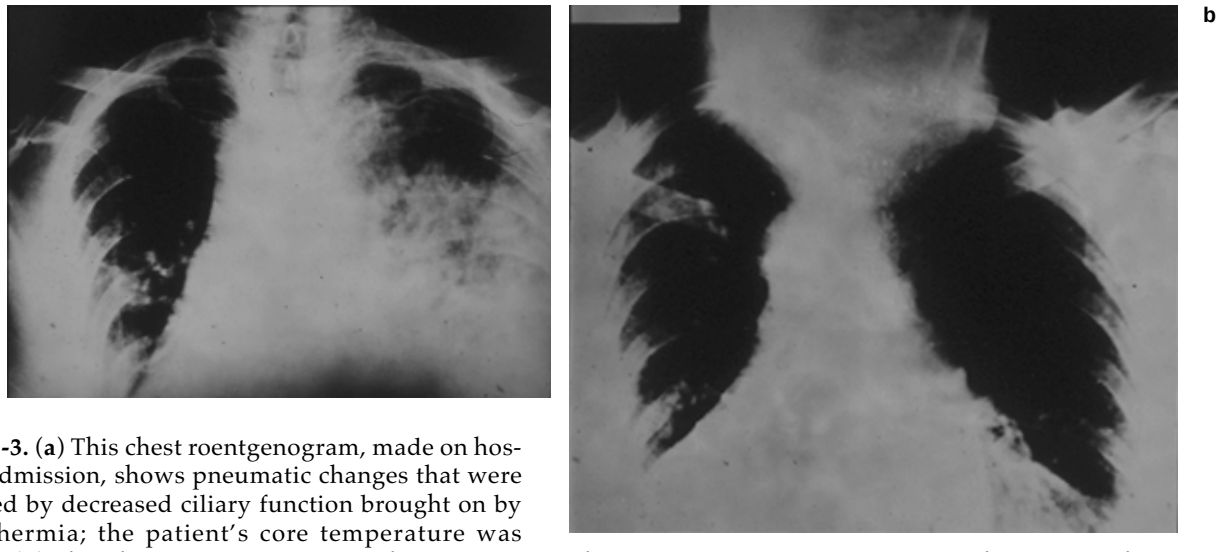

Fig. 16-3. (**a**) This chest roentgenogram, made on hospital admission, shows pneumatic changes that were induced by decreased ciliary function brought on by hypothermia; the patient's core temperature was 32.5°C. (**b**) This chest roentgenogram of the same patient shows marked improvement on reaching 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

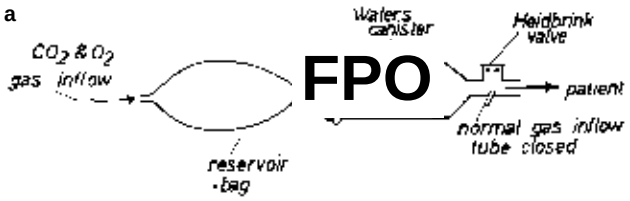

Fig. 16-4. (a) Simple circuit for providing airway warming from an anesthetic machine. The patient may be connected by face mask or endotracheal tube as appropriate, and ventilation assisted if required. INSTRUCTIONS FOR USE: The carbon dioxide is allowed in as a flow rate of 3 to 5 L/min, and oxygen 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

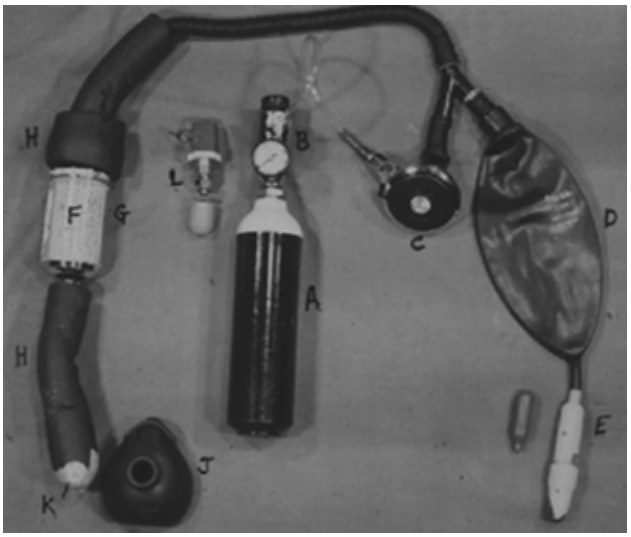

Fig. 16-5. Lightweight Lloyd portable airway warming equipment. (**A**) Oxygen cylinder. (**B**) First-stage reducing valve and gauge. (**C**) Demand-reducing valve with manual override to allow 2-L reservoir bag (**D**) to be filled if ventilation assistance is required. The demand valve may be replaced 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.

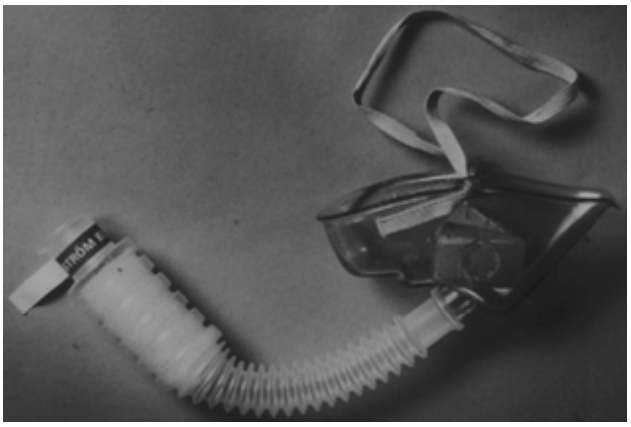

Fig. 16-6. Condenser humidifier with face mask attached. This airway rewarming equipment can be used in the emergency treatment of accidental hypothermia. The end of the humidifier should be placed under the clothing next to the skin, and the whole device, including the mask, should be covered (eg, with 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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Chapter 17

COLD WATER IMMERSION

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INTRODUCTION

The body core temperature is maintained daily within a narrow range, despite wide variations in environmental (ambient) conditions and activity levels.[1] In fact, the body core temperature is so consistent that for centuries physicians have used deviation from this normal temperature as an indication of a departure from the healthy state.[2] Maintenance of a normal temperature and the normal variations of the circadian and lunar rhythms are achieved by changes in all physiological systems, one of the most important of which is alteration in skin blood flow.[3] In response to the perception of skin temperature, behavioral adjustments are made in clothing, environment, or both, to avoid sweating or shivering (ie, to maintain the core level). When exposed to more extreme environmental conditions, powerful physiological effector mechanisms are called into play to defend against hyperthermia and hypothermia. Active vasodilation of blood vessels of the skin and secretion of sweat keep body temperature within a few degrees of normal even in severe conditions of increased ambient temperature, such as marathon running on a hot day.[4] On the other hand, the defenses against hypothermia are less effective. Individuals exposed to severe cold are unable to maintain thermal steady states for extended periods despite maximum vasoconstriction and high rates of shivering.

REGULATION OF BODY HEAT CONTENT

The physics of heat loss following cold water immersion dictate a more rapid drop in core temperature than from exposure to a cold air environment.[5] Figure 17-1 illustrates the dramatic difference in body (core) temperature drop when the same individual was exposed alternately to 0°C air and immersed in 10°C water. Magnitude of heat loss and eventual decrease in core temperature depends to a great extent on how much of the body is actually immersed.[6] In the following discussion, *immersion* is defined as submerged to the neck.

When a body is immersed in water it is obvious that evaporative and radiant heat exchange can no longer occur between the subject and the environment. The major heat exchange in water occurs by means of conduction with the surrounding water. The exceptions to this are the nonimmersed body parts, in most cases the head. The head can represent a significant site of heat loss to the environment owing to its minimal insulation and lack of vasoconstriction.[7] For example, when the ambient temperature is 4°C, the uncovered head could be responsible for heat loss representing 50% of resting heat production, increasing to 75% at an ambient temperature of –15°C.[8]

The heat dissipation capacity of water is considerably greater than that of air, as the ratio of heat conductivity of water to air is approximately 24:1.[9] A body immersed in calm water is surrounded by a relatively stable "boundary layer" of fluid. Heat is conducted from the skin into this boundary layer as a function of the temperature gradient, heat transfer coefficient, and surface area of contact,[10] as Equation 1 demonstrates:

$$H = K A (T_w - T_{sk}) \quad (1)$$

where H represents heat transported, K the heat transfer coefficient, A the surface area of contact between skin and water, T_w the temperature of water, and T_{sk} the temperature of the skin.

Conservation of body heat and maintenance of core temperature during immersion depends on the temperature of the water. If there is no net heat loss or gain by the body during immersion, the water temperature is within the thermoneutral zone (also called the neutral zone or the vasomotor zone). The

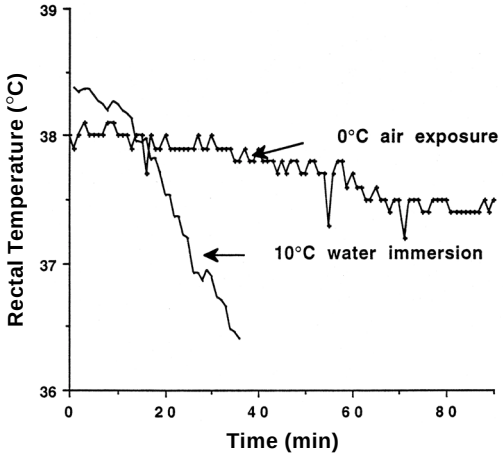

Fig. 17-1. Rectal temperature versus time relationship in one human volunteer under two types of cold stress: 0°C air and 10°C water. The subject is a young white man of average build, fat content, and physical fitness. He is dressed the same for both sessions. Note that even though the water temperature is 10°C higher than the air temperature, the decrease in core temperature is much more rapid in the water immersion, owing to the increased conductivity of the water compared with that of air.

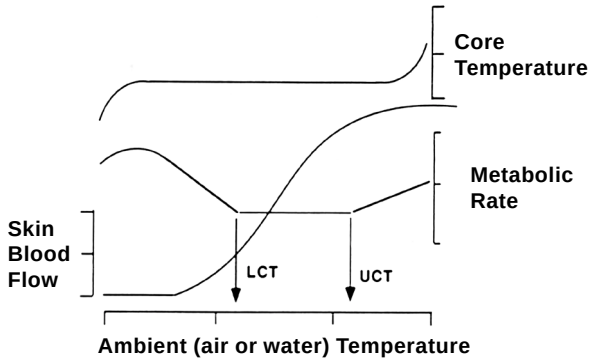

Fig. 17-2. The relationships between core temperature, metabolic rate, and skin blood flow as a function of ambient temperature. The thermoneutral zone is represented by the horizontal segment of the metabolic rate curve and is bounded by the upper critical temperature (UCT) and the lower critical temperature (LCT). In the thermoneutral zone the core temperature is maintained constant by alterations in heat exchange at the surface, represented here by changes in skin blood flow.
Adapted with permission from Eckert R, Randall D, Augustine G. *Animal Physiology: Mechanisms and Adaptations.* New York, NY: WH Freeman; 1978: 580.

ambient temperature defining the thermoneutral zone is a very narrow range around 35°C.[11,12] Within this limited range of ambient temperatures, nude subjects maintained their core temperature nearly constant for at least 1 hour without shivering or sweating. This ability to maintain a steady state in core temperature was attributed to changes in skin blood flow; that is, vasomotor control.[12,13] In the thermoneutral zone, skin blood flow is under the reflex influence of skin temperature. An increase in skin temperature results in a rapid increase in skin blood flow and vice versa. It is from this thermoneutral zone that the body moves when cold water immersion occurs, calling into play powerful compensatory mechanisms. The concept of the thermoneutral zone and temperature regulation is summarized in Figure 17-2.

As the water temperature falls below the lower critical temperature, peripheral vasoconstriction can no longer effectively prevent heat loss. Therefore, heat loss exceeds the heat produced by the basal metabolic processes, and heat production must be increased to maintain the core temperature. The lower critical temperature is a function of subcutaneous fat thickness and ranges between 30°C and 34°C.[14] Increased heat production can be accomplished by an increase in metabolic rate,[5] voluntary muscle work (exercise), or shivering, in any combination. Although these three mechanisms will contribute to the maintenance of core temperature, in a cold water environment, the added physical activity associated with exercise will tend to disrupt (ie, to mix) the boundary layer and, to some extent, facilitate heat loss. These mechanisms (increased heat production and maximal vasoconstriction) may be able to stabilize core temperature for some time. However, if the cold stress is of sufficient magnitude and is continued for a long enough time, heat loss will eventually exceed heat production, leading to a decrease in core temperature and eventually to hypothermia. As the core temperature declines past a critical level, the rate of both tissue metabolism and shivering will be depressed, resulting in an accentuated drop in core temperature due to this depression of heat-production mechanisms.

The problem of maintaining core temperature during cold water immersion can be modeled as a simple two-compartment system in which the body core is separated from the surrounding environment by an insulating shell (Figure 17-3). Heat can move from the core through the shell by conduction, essentially a diffusive process, but this mechanism is a very slow transport process, owing to the distances involved. The major pathway for heat movement out of, or heat exchange with, the core and into the shell and subsequently facilitating transfer

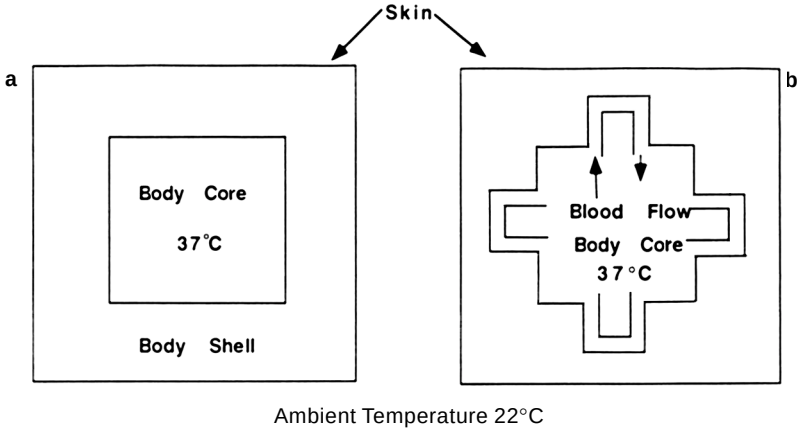

Fig. 17-3. A summary of the Shell–Core model of heat flow in the body. (**a**) If the blood flow to the shell is minimal, it will provide the largest insulating capacity and therefore retain heat and maintain core temperature. (**b**) When vasodilation in the shell occurs, the insulating capacity of the shell drops and heat is lost from the core.
Adapted with permission from Vander A, Sherman J, Luciano D. *Human Physiology: The Mechanisms of Body Function.* 7th ed. Boston, Mass: WCB McGraw-Hill; 1997: 628.

EXHIBIT 17-1

RANGER TRAINING INCIDENT REPORT

On 15–16 February 1995, three students in training to become US Army Rangers, ranging in age from 23 to 27 years, died of hypothermia during a routine patrol at the Florida Ranger camp. Failure in supervision and judgment, lack of situational awareness, and lack of control placed Ranger students in water too cold, too deep, and too long for safe operations.

The Ranger Training Brigade trains about 3,000 Ranger students each year. This training takes place in Fort Benning, Georgia, the mountains of northern Georgia, and in the swamps of Florida. The tragedy that occurred to class 3-1995 was a result of unfortunate human errors. However, it does bring to light the need to validate Army water exposure guidance tables.

These Ranger students died during the swamp phase of their training. This is the third and final phase of Ranger School, conducted at Camp Rudder, Florida. By this point in the ranger training process, the philosophy is to allow the student chain of command to make their own decisions involving navigation, judgment, and leadership. The Ranger Instructors (RIs) will only intervene when student errors have made the mission impossible, or make the training unsafe.

According to Ranger training standard operating procedures in preparation for the swamp field training exercise (FTX), water levels are checked 10 days and then again 2 days prior to the FTX. On the initial check, water levels were noted to be 12 to 18 inches lower than those encountered during the last FTX. However, during the 2-day prior check the water level had risen 18 to 24 inches. Data from the National River Forecasting Agency indicated that the river was rising and would reach flood stage around 15 February. This information was not used by the Florida Ranger Training Brigade. The typical way water depth was measured was by visually checking various landmarks in the area rather than through a quantified, repeatable process.

The general forecast for the FTX was partly cloudy with temperatures ranging from 60°F to 70°F. Winds were from the south at 10 to 15 mph, with a 30% chance of rain. From the site recon on 14 February, water depth was noted as knee to mid-thigh deep. Water temperature on 15 February was 52°F, but rose to around 54°F to 59°F during the day.

The general mission for Class 3-1995 was to move 8 to 10 km downriver via a Zodiac boat, move a short distance (700–800 m) through the swamp, and move the remaining few kilometers over ground to their objective. On 15 February the FTX was given the go ahead. The Ranger students embarked downriver in their Zodiac boats.

At approximately 1400 hours, C Company's student platoon leader missed his drop site. Because of the Ranger Training philosophy described above, the RI allowed him to continue downriver. B Company followed C Company downriver. A Company found their drop site and debarked their boats onto dry land. The completed their mission as planned. The decision to allow the student chain of command to continue downriver placed the RIs of B and C companies in an unfamiliar area of the swamp. This is one of the critical factors of this FTX.

Current Ranger standards allow students to be in chest-deep water of 50°F to 55°F for a maximum of 3 hours. If the water temperature is from 56°C to 60°F, the time limit increases to 5 hours. If the whole body is submerged, the time limit is no more than 5 minutes for either condition.

B and C companies found a drop site and reported that the water levels were much higher than briefed. Because of the current it was impossible to move back upriver to a different drop site. The Ranger students decided to move through the swamp away from the river, the general idea being that the water level would have to drop as they approached high ground. The first man entered the water at 1600 hours. Movement through the swamp was much more difficult than anticipated. After the students had been in the water for 1.5 hours, early signs of hypothermia were becoming evident. At this point the RIs took administrative control of the patrol and called for the first of several medical evacuation (medevac) requests.

(**Exhibit 17-1** *continues*)

Exhibit 17-1 *continued*

The RIs decided the best way to reach high ground and get out of the water was by constructing rope bridges to cross the deep water. Because this contingency had not been planned for, it took about 30 to 45 minutes to complete the rope bridges and move all of the students across the water obstacle. During this time the students waited in the chest- to neck-deep water, causing their bodies to continue to cool. After crossing these water obstacles the companies were forced to continue their movement through the swamp.

The medevac helicopter was over the Ranger students by 1740 hours. It was not until 1840 hours that the medevac helicopter departed with the hypothermic students. This delay was caused by the confusion of the current situation as well as problems with extracting the three students from the swamp. All of these students survived.

Once the helicopter departed, the RIs attempted to get the students moving in an organized fashion toward higher ground. The students were reluctant to respond. Their legs were cramping and most were beginning to feel better by staying in one place. During the medevac, the rotor wash of the helicopter combined with the current of the river had caused a group of students to drift away from the main element. These students attempted to push on to high ground on their own. One RI organized a small group to move ahead of the main element. This group was to build a fire to help the students in the main element to rewarm, as well as to guide the lost group to their location.

By approximately 2100 hours, most of the students had reached high ground. However, several students were still missing in the swamp. RIs began searching for the missing Ranger students as individuals at the fire ignited flares to help guide the search. At this point another medevac helicopter arrived. The helicopter was called for seven students in critical need of medical attention. However, because of limited room aboard the helicopter, only five of these students were able to be evacuated. This medevac attempt was greatly hampered by all the confusion on the ground. In panic, the "lost" Ranger students had removed the red lenses from their flashlights. This caused problems for the pilot and crew because the signal for the medevac pickup point is a flashing white light. As the medevac attempt continued, RIs resumed their search. At 2200 hours, two students remained missing in the swamp. All other medevacs were called off because of the weather and the lack of logistical planning. The search was called off at 0340 hours to prevent any further casualties.

Of the soldiers who were successfully medevaced, one died in the emergency room. The two soldiers left behind were carried to the road and picked up by a forward line ambulance and transported to the emergency room. Both of these men were pronounced dead at approximately 0200 hours. At 0530 hours the search resumed for the last missing Ranger student. He was found at approximately 0730 hours lying on a log in the swamp less than 75 feet from high ground. This soldier was pronounced dead at 0853 hours.

The subsequent investigation of this incident prompted the Ranger Training Brigade to task the US Army Research Institute of Environmental Medicine to validate their water safety tables.

Exhibit compiled from material provided by the Public Affairs Office, US Army Infantry Center, Fort Benning, Georgia; 27 March 1995.

into the environment, is by way of blood flow between the two compartments. The size or thickness of the shell is in fact determined by (*a*) the blood flow to it and (*b*) the effective area available for heat transport. An increase in blood flow to a body region effectively decreases the size of the shell, resulting in an increase in heat loss. Peripheral vasoconstriction, on the other hand, increases the shell by increasing its thickness and decreasing the effective area for heat transport. This results in an increased insulating capacity, restricting heat loss and maintaining core temperature.

Hypothermia can be insidious, as exemplified by the deaths of four soldiers (15-16 Feb 1995) who were undergoing the rigorous training to become US Army Rangers (Exhibit 17-1). From a physiological perspective, the students suffered from secondary hypothermia. They were in the ninth day of the final phase of their training at the Ranger School, Camp Rudder, Florida. The training was intense, and as part of that training the men were allowed three meals over 2 days. More than likely, the dif-

ference between the environmental air temperature (60°F–70°F) and the water temperature (52°F) played a role in their deaths. The warm air temperature may have given the false impression that the water was also warm. The men were moving in water at 52°F for a minimum of 1.5 hours. Thus, insufficient calories and sleep coupled with intense, prolonged exercise in cold water led to the deaths of four soldiers. As a consequence, the US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts, developed new water exposure tables for the Rangers, the particulars of which are privileged information.[15]

All body regions do not respond with the same magnitude of peripheral vasoconstriction when placed under a cold stress. The trunk and head show minimal vasoconstriction, with the areas of maximal heat loss being the neck, lateral thorax, upper chest, and groin.[16] Vasoconstriction of the fingers and toes may cause discomfort significant enough to jeopardize military operations, even though the core temperature is normal. Insulating capability of the shell is a function not only of blood flow but also of fat content. The more body fat contained in the shell, the less the reduction in core temperature for a given immersion water temperature,[17] or the greater likelihood that the core temperature will stabilize at a given water temperature.[18] The importance of the subcutaneous fat layer is illustrated by the successful English Channel swimmers, all of whom are well endowed with this insulation layer.[9]

PHYSIOLOGICAL RESPONSES TO COLD WATER IMMERSION

The body's response to cold water immersion progresses from an initial stress situation to eventual hypothermia and death, depending on time and intervention. During the initial phases of immersion and the stress of cold water exposure, hypothermia is *not* the major concern; other stresses on the body's function are more life threatening. Tipton, Golden, and Hervey[19,20] have approached the various physiological changes associated with water immersion in a stepwise fashion (Table 17-1). Based on their four stages—initial immersion, short-term im—mersion, long-term immersion, and postimmersion-and depending on the temperature of the water, it is unlikely that hypothermia would become a problem until some point in stage 3, long-term immersion. The magnitude (severity) of the physiological changes in each of the stages will depend on a number of factors: water temperature, circulation (ie, water current) or body movement or both, body size, body fat content, and the presence or absence of protective gear (clothing). Stage 4, postimmersion, encompasses changes in core temperature (afterdrop) that may occur following the immersion incident. Note that treatment for hypothermia (rewarming methodology and postimmersion collapse) will not be covered in this chapter, but interested readers can find the topic discussed in Chapter 16, Treatment of Accidental Hypothermia, and in publications by researchers in Great Britain.[21,22]

TABLE 17-1

STAGES OF WATER IMMERSION

Stage	Description	Immersion Time (min)
1	Initial immersion	0–3
2	Short-term immersion	3–15
3	Long-term immersion	≥ 30
4	Postimmersion	Core temperature may fall, but after the incident (ie< afterdrop)

Source: Tipton MJ. The concept of an "integrated survival system" for protection against the responses associated with immersion in cold water. *J R Nav Med Serv.* 1993;79:11–14.

The initial response to immersion (stage 1) has a respiratory and cardiovascular component. Respirations may become uncontrolled, with reflex gasping and hyperventilation. This panic response will decrease the breath-holding time and may lead to aspiration with subsequent drowning.[23] Hyperventilation, possibly reaching five times resting values, is associated with an increase in both tidal volume and breathing frequency. The increased ventilation will result in a decrease in the partial pressure of carbon dioxide in the alveolar gas ($PACO_2$) and subsequently in arterial blood ($PaCO_2$), an acute respiratory alkalosis leading to cerebral vasoconstriction (with its effects on mental function), and possibly tetanic convulsions.[24] As the immersion time progresses, the minute ventilation decreases toward normal, the tidal volume remains increased, and the $PACO_2$ remains somewhat depressed.[25]

Heart rate response to cold water exposure depends on the length of exposure time and whether or not complete submersion has occurred. If the

victim's face is immersed while he is breath-holding, then heart rate may decrease (bradycardia), an example of one component of the "diving response" or "diving reflex."[26,27] The complete diving reflex consists of the following components:

1. decrease in heart rate,
2. increase in total peripheral resistance,
3. decrease in cardiac output (ie, stroke volume), and
4. increase in mean arterial pressure.

In humans this cardiovascular reflex will occur during face-only immersion[28] as well as in total body immersion.[29] The predominant neural drive input to the myocardium associated with the diving reflex is parasympathetic in origin. However, the general cold stimulus also activates the sympathetic neural pathway to the myocardium and drives the systemic changes in total peripheral resistance, resulting in the redistribution of organ (tissue) blood flow.[30] This competition between augmented sympathetic and parasympathetic activity may be responsible for arrhythmias that occur at the end of—or just following—face immersion.[31]

If total submersion or face immersion is absent or if breath-holding face immersion has ended, the remaining cardiovascular adjustments are a result of the initial cold stress. There is a dramatic increase in heart rate, which subsides after a few minutes but remains above preimmersion levels; the colder the water, the higher the heart rate. Other cardiovascular sequelae of cold-induced sympathetic activation are also evident: increased systemic arterial pressure, increased total peripheral resistance, and increased cardiac output. During this early sympathetic discharge phase, myocardial conduction abnormalities may be observed, such as atrial and ventricular extrasystoles and sinus arrhythmias.[32]

The initial cardiovascular responses to sympathetic activity will eventually subside as the core temperature falls. Heart rate, cardiac output, and systemic arterial pressure will fall as a function of decreasing core temperature.[33] Individual organ (heart, liver, kidney, and brain) blood flow will also progressively decrease at the rate of approximately 5% per degree Centigrade in core temperature drop (estimated from data in Blair[34]).

The kinetics and conductance of ion channels in the myocardium are temperature dependent. Hypothermia will result in alterations in electrophysiological mechanisms, prolonged action potentials, and refractory period and progressive bradycardia. At temperatures between 28°C and 30°C, the myocardium becomes susceptible to ventricular tachyarrhythmias.[35]

The initial cold immersion insult may result in death from drowning, cardiovascular collapse, or both, but *not* from hypothermia. If the initial entry into the cold water is gradual (staged) or is a common occurrence for the individual (eg, cold water swimmers and divers), then the cardiovascular and respiratory changes may be significantly attenuated.[23] If the victim survives the initial insult, the hyperventilation will subside, reducing the ventilation to match the metabolic needs. Heart rate will decrease toward preimmersion levels, with the final value depending on the water temperature. Peripheral vasoconstriction will persist during stages 2 and 3 as a means of preventing heat loss and maintaining core temperature.

The peripheral circulatory beds, such as fingers, toes, and ears, demonstrate a paradoxical response to cold exposure.[36,37] The initial vasoconstriction induced by the cold exposure is followed in 5 to 15 minutes by a vasodilation of variable duration and magnitude (cold-induced vasodilation, CIVD). This is followed by a cyclic vasoconstriction-dilation phenomenon with a frequency of 3 to 5 per hour.[38,39] This CIVD will result in an increase in skin temperature and may contribute to an increase in body heat loss. The magnitude, and therefore the importance, of the CIVD decreases as cold exposure time is prolonged and body heat content and core temperature decrease (CIVD is also discussed in Chapter 15, Nonfreezing Cold Injury).

In stages 2 and 3, shivering will begin in an attempt to increase heat production. The onset of shivering occurs at a skin temperature of 26°C.[40] As the core temperature continues to fall, shivering will increase, reaching a maximum at a core temperature of approximately 35°C.[41] A continued decrease in core temperature will be accompanied by suppression of shivering, with final cessation at a core temperature around 30°C.[42]

The further physiological changes associated with cold water immersion in stage 3 will depend on both the water temperature and the exposure time. For the purposes of this discussion it is assumed that the victim is minimally clothed and in a negative heat balance; that is, losing more heat to the environment than can be produced by metabolic processes, shivering, and muscular activity. The rate of core temperature drop will be a function of the water temperature. Figure 17-4 is a compilation of rates of core temperature cooling from 17 sets of published data.[40,43–59] The subjects in these experiments cannot be considered matched for all con-

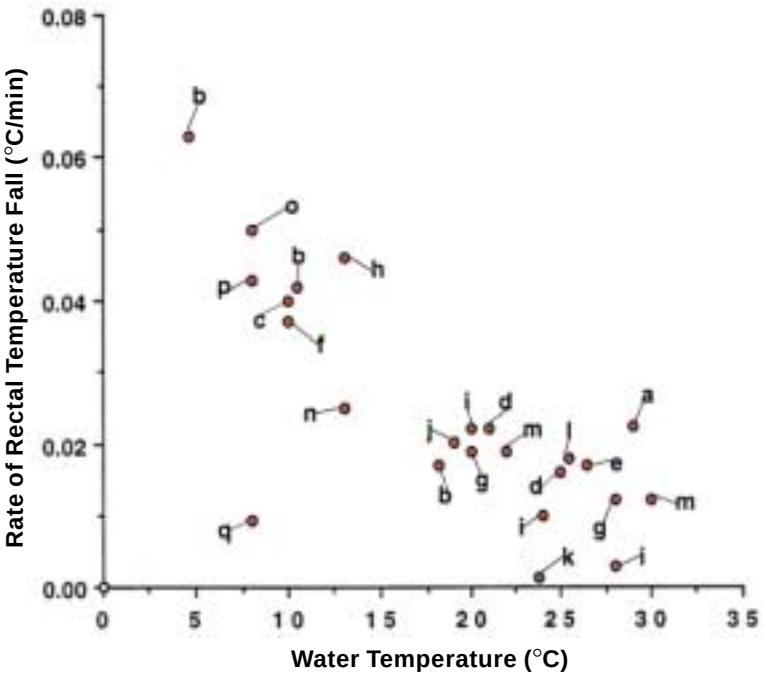

Fig. 17-4. The rate of core cooling data as a function of water temperature was extracted from 17 separate published reports (sources a–q, below) on experiments employing human volunteers. For the most part, the subjects were young white men of average build, fat content, and physical fitness. The subjects were not wearing any clothing or employing any devices that were designed to protect against cold exposure.

Data sources: (a) Hayward MG, Keatinge WR. Progressive symptomless hypothermia in water: Possible cause of diving accidents. *Br Med J.* 1979;1:1182. (b) Hayward JS, Eckerson JD, Collis ML. Thermal balance and survival time prediction of man in cold water. *Can J Physiol Pharmacol.* 1975;53:21–32. (c) Hayward JS, Eckerson JD, Collis ML. Thermoregulatory heat production in man: Prediction equations based on skin and core temperature. *J Appl Physiol.* 1977;43(2):377–384. (d) Israel DJ, Heydn KM, Edlich RF, Pozos RS, Wittmers LE. Core temperature response to immersed bicycle ergometer exercise and water temperatures of 21°, 25°, and 29°. *J Burn Care Rehabil.* 1989;10(4):336–345. (e) Veicsteinas A, Rennie DW. Thermal insulation and shivering threshold in Greek sponge divers. *J Appl Physiol.* 1982;52(4):845–850. (f) Wittmers LE, Pozos RS. In situ hypothermia testing. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold*. Bethesda, Md: Undersea Medical Society Inc; 1983: 153–168. (g) McArdle WD, Toner MM, Magel JR, Spina RJ, Pandolf KB. Thermal responses of men and women during cold-water immersion: Influence of exercise intensity. *Eur J Appl Physiol.* 1992;65:265–270. (h) Martin S, Diewold RJ, Cooper KE. Alcohol, respirations, skin and body temperature during cold water immersion. *J Appl Physiol.* 1977;43(2):211–215. (i) McArdle WD, Magel JR, Gergley TJ, Spina RJ, Toner MM. Thermal adjustments to cold-water exposure in resting men and women. *J Appl Physiol.* 1984;56:1565–1571. (j) Rochelle RD, Horvath SM. Thermoregulation in surfers and nonsurfers immersed in cold water. *Undersea Biomed Res.* 1978;5(4):377–390. (k) Beckman EL, Reves E. Physiological implications as a survival during immersion in water at 75°F. *Aerospace Med.* 1966;37(11):1136–1142. (l) Weihl AC, Langworthy HC, Manalaysay AR, Layton RP. Metabolic responses of resting man immersed in 25.5°C and 33°C water. *Aviat Space Environ Med.* 1981;52(2):88–91. (m) Martin S, Cooper KE. Alcohol and respiratory and body temperature changes during tepid water immersion. *J Appl Physiol.* 1978;44(5):683–689. (n) Graham T, Baulk K. Effect of alcohol ingestion on man's thermoregulatory responses during cold water immersion. *Aviat Space Environ Med.* 1980;51(2):155–159. (o) Giesbrecht GG, Sessler DI, Mekjavie IB, Schroeder M, Bristow GK. Treatment of mild immersion hypothermia by direct body-to-body contact. *J Appl Physiol.* 1994;76(6):2373–2379. (p) Bristow GK Sessler DI, Giesbrecht GG. Leg temperature and heat content in humans during immersion hypothermia and rewarming. *Aviat Space Environ Med.* 1994;65:220–226. (q) Bourdon L, Jacobs I, Bell D, Ducharme MB. Effect of triazolam on responses to a cold-water immersion in humans. *Aviat Space Environ Med.* 1995;66(7):651–655.

tributing variables. They were, however, for the most part, young, fit men of average body type not participating in any type of exercise or wearing any special clothing to protect against heat loss during the time of immersion. The data demonstrate the magnitude of core temperature drop as a function of water temperature. These data illustrate the variability from experiment to experiment when evaluating core temperature changes as a function of cold stress.

In emergencies, physiological compensatory mechanisms such as shivering, pulse, and loss of reflexes can be used to obtain a rough estimate of core temperature. Table 17-2 summarizes the relationship between core temperature and signs and symptoms of hypothermia. The data are divided into three ranges of core temperature: mild hypothermia, 37°C to 33°C; moderate hypothermia, 32°C to 27°C; and severe hypothermia, 26°C to 18°C.

Military research into human performance in extreme environments is conducted by many nations. In 1996, Commander Wolf J. Haenert, MD, PhD, Medical Corps, German Navy, visited the Naval Health Research Center, San Diego, California, and subsequently submitted to Robert S. Pozos, PhD, then head of the Department of Applied Physiology there, a translated report on the German experience with survivors of shipwrecks during World War II. The report, written by Dr. V. Hartmann, a

TABLE 17-2

SIGNS, SYMPTOMS, AND PHYSIOLOGICAL CHANGES ASSOCIATED WITH PROGRESSIVE HYPOTHERMIA

Stage of Hypothermia	Core Temperature Rectal, °C (°F)	Signs and Symptoms
	37 (98.6)	Normal rectal temperature
Mild	36	Increased metabolic rate due to exercise and shivering
	35	Maximum shivering thermogenesis
	34	Amnesia, dysarthria, and judgment problems
	33	Ataxia and apathy
Moderate	32	Stupor; oxygen consumption 75% of normal
	31	Shivering stops
	30	Possible cardiac arrhythmias; pulse and cardiac output 66% of normal
	29	Decreasing consciousness, pulse, and respirations; dilated pupils
	28	Increased sensitivity to ventricular fibrillation; pulse and oxygen consumption 50% of normal
	27 (80.6)	Loss of voluntary motion and reflexes
Severe	26	Major acid–base problems; no response to pain
	25	Cerebral blood flow 30% of normal; possible pulmonary edema
	23	Loss of corneal and oculocephalic reflexes
	22	Maximum risk of ventricular fibrillation; oxygen consumption 25% of normal
	20	Pulse 20% of normal
	19 (66.2)	Electroencephalogram flat
	18	Asystole

Adapted with permission from Danzl DF, Pozos RS. Accidental hypothermia. *N Engl J Med*. 1994;331(26):1757.

medical officer in the German Navy, and Dr. Haenert (who also translated the report), is presented in toto in the Attachment at the end of this chapter. It contains a key observation about afterdrop and documents the rewarming of victims of hypothermia with hot water. In addition, it mentions rewarming victims of hypothermia with a fire. In field situations, many different methods are used to rewarm victims of hypothermia. However, rewarming with hot water is not possible except aboard ship, and even then, caution should be exercised by monitoring heart rate and blood pH. Warming with fire has disadvantages in that it promotes peripheral vasodilation, which can promote afterdrop. Even in 1944, the advantages of protective clothing were noted.

GENDER DIFFERENCES IN THERMOREGULATION

Differences in thermal regulation between men and women were reported in the early 1940s.[60] However, only since the late 1970s have the scientific community and funding agencies turned their efforts to address these gender differences.[61] The data are at some points confusing and, to say the least, incomplete.

Body Characteristics

Some of the differences in temperature regulation observed between men and women may be attributed to anthropomorphic characteristics. Compared to men, women tend to be of smaller stature, with resultant larger surface area-to-body mass ratio and lower total thermal mass; these contribute to a more rapid heat loss and decrease in core temperature when exposed to cold stress.[62] The lower muscle mass of women (vs men) will produce less metabolic heat from both exercise and shivering, leading to a decreased ability to supply heat to the core. The thicker subcutaneous fat layer in women will provide more insulating capacity in the shell and to some extent retard heat loss. However, the net result for women is a faster cooling rate when exposed to an environmental cold stress.[63]

Temperature Regulation and the Menstrual Cycle

The reproductive system has an important effect on female thermoregulation.[63] In the follicular phase of the menstrual cycle (the first half of the cycle, beginning with the onset of menses and ending with ovulation), women thermoregulate similarly to men if both genders are at comparable fitness levels.[64] However, during the luteal phase of the menstrual cycle (the second half, from ovulation to the onset of menses), hormonal and physiological changes associated with ovulation significantly affect thermoregulation. Resting core temperature is elevated,[65] and onset of sweating occurs at a higher temperature, suggesting that the set point for temperature regulation has been elevated.[66]

During the luteal phase, finger blood flow shows a greater cold-induced vasoconstriction and a slower recovery when compared with the follicular phase of the menstrual cycle.[67] It has also been observed that there are menstrual cycle changes in the blood flow of the forearm, leg, and calf.[68] Regulation of cutaneous blood flow in response to cold exposure is primarily under sympathetic control, and studies have demonstrated the influence of estrogen on the sympathetic nervous activity. Evidence from animal studies indicates that estrogen induces an up-regulation (vasoconstriction) of α_2-adrenoreceptors.[69,70] Although much less investigated, the same effects seem to be true for progesterone.[71]

Cold-induced vasospastic disorders such as Raynaud's phenomenon are more common in women, with the female-to-male ratio ranging from 2:1 to 9:1.[72] The onset of these vasospastic attacks occurs with menarche[73]; the attacks subside after menopause.[74] The frequency and severity of cold-induced vasospastic episodes also vary with the phase of the menstrual cycle and subside during pregnancy.[75,76]

Behavioral differences are also associated with the menstrual cycle. In the luteal phase, women sense changes in skin temperature more quickly than during the follicular phase.[77] In addition, women have a higher skin temperature preference in the luteal phase.[78] Both these modifications are consistent with an increase in the temperature set point.

Differences in Response to Cold Water Immersion

Data suggest that women exhibit a more rapid drop in core temperature than men when immersed in cold water.[43,79] This increased rate of core temperature fall is present if both genders are matched for body fat content. Increased body fat in women does provide insulation during water immersion; however, the larger surface area-to-mass ratio and the lower thermal mass contributing to heat production will result in women's faster initial cooling rate during water immersion, compared with that of men.[61]

As mentioned earlier, exercise during cold water immersion may produce sufficient heat to retard the drop in core temperature. For men and women of the same body fat composition, decreases in core temperature are greater for women in the resting state or when performing light exercise; however, with more intense exercise, women maintain a higher core temperature than men.[44] If men and women exercise at the same absolute work intensity, there is no difference in cooling rate.[43,79] Under these conditions the women are exercising at a higher percentage of their maximal aerobic power and are therefore producing a greater amount of heat per unit thermal mass. Women exercising at the same percentage of their maximal aerobic power as men will cool at a faster rate, illustrating the imbalance between heat loss and production.[45]

PROTECTIVE CLOTHING

Two types of protection can be provided for survival in the case of cold water immersion: flotation devices and hypothermia protection. Therefore, it is beneficial during stage 1 of immersion to be equipped with some type of personal flotation device. The minimal function of this device is to float the victim in such a manner as to keep the face out of the water. The victim will not have to expend energy to stay afloat and will be protected from drowning if or when hypothermia progresses to the point that voluntary efforts to maintain flotation are no longer possible.

Numerous garments have been designed for hypothermia protection during accidental cold water immersion. One such garment is constructed to function as workable clothing in air, and will provide both flotation and hypothermia protection if the individual falls into cold water (Figure 17-5).

Fig. 17-5. (**a**) These are examples of a flotation jacket with hypothermia protection capabilities. Note the groin protection, which minimizes the flow of water through the jacket. (**b**) This deck suit has hypothermia protection capabilities. Core temperature data from both garment types and a control dressed in street attire are presented in Figure 17-6.

These suits and jackets do not keep the victim dry; water enters the garment and is trapped between the victim's body and the suit. Body heat, provided in part by shivering, warms the trapped water, which in this case represents the "boundary layer," and the suit's insulating capacity minimizes the heat loss to the surrounding environment. Figure 17-6 plots data from a subject who was immersed in 10°C water on three different occasions, dressed as follows[46]:

1. As a control, the subject wore street clothes (cotton underwear, shirt, jeans, socks, and tennis shoes) with flotation provided only by a neck collar.
2. The subject wore a jacket equipped with a groin flap to minimize water movement through the jacket.
3. Later, the subject also wore a flotation full-body suit.

The subject's core (rectal) temperature reached 35.6°C by 30 minutes in the control condition, by 60 minutes when clothed in the jacket, and remained above this level for 3 hours when clothed in the full body suit.

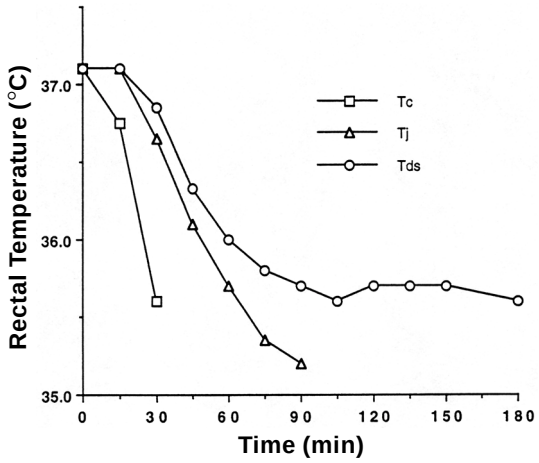

Fig. 17-6. Data representing the rectal temperature versus time plots of a single volunteer undergoing three testing conditions. The subject is a young white man of average build, fat content, and physical fitness. The three water immersions took place at least one week apart, and the water temperature each time was 10°C. In the control condition (T_c) the subject was clothed in regular street attire; in the other two conditions the subject wore either a jacket (T_j) or a deck suit (T_{ds}) designed for hypothermia protection.

Fig. 17-7. This flotation survival suit has hypothermia protection capabilities. Note that the gloves, feet (not shown), and hood are part of the suit, and are intended to keep water entry down to a few cubic centimeters. The subject is entering a tank with the water temperature set at 0°C, in a 2-phase system. Core temperature data from this garment are presented in Figure 17-8.

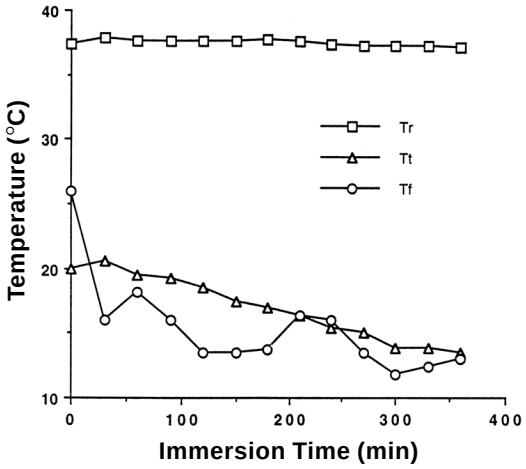

Fig. 17-8. A summary of a volunteer's 6-hour immersion in ice water (temperature range 1°C–3°C). The protective device worn was a survival suit of the dry type, as depicted in Figure 17-7. T_r represents rectal temperature, T_f represents skin temperature on the ventral aspect of index finger, and T_t represents skin temperature on the ventral aspect of the large toe. Adapted with permission from Wittmers LE, Pozos RS. In situ hypothermia testing. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold*. Bethesda, Md: Undersea Medical Society Inc; 1983: 166.

The efficiency of long-term survival suits (Figure 17-7), which are designed to keep the victim dry and provide hypothermia protection, is illustrated in the data plotted in Figure 17-8. These one-piece, full-body suits allow little or no water into the garment when the subject falls into the water. In this test, the subject was immersed in ice water for 6 hours, and his core temperature remained above 37°C for the entire exposure.

In general, as these data show (see Figures 17-6 and 17-8), some type of flotation device and protective clothing are essential for survival when the victim is in danger of long-term exposure to cold water immersion.

Many military operations require clandestine operations involving Special Forces or US Navy SEALs, who travel to their targets in open minisubmarines and subsequently swim to shore. These operations require scuba diving gear and, in cold water, various kinds of insulating suits. In most operations of this nature, the personnel experience severely cold feet or hands rather than hypothermia. Although the personnel have adequate oxygen and insulation for their truncal regions, pronounced vasoconstriction of the digits—even though they are wearing special gloves and footwear—remains a major challenge for the execution of cold water submersible operations.

NEAR-DROWNING: HYPOTHERMIA AND THE DIVING RESPONSE

The preceding discussion has for the most part been limited to immersion cold stress under conditions in which the head of the victim remains out of the water. If, however, submersion results in hypoxia due to aspiration or laryngospasm, the accident is classified as a drowning. On the other hand, if the victim is recovered, successfully resuscitated, and lives for "some time" (usually 24 h), the event is classified as a near-drowning.[80,81]

The cases of near-drowning most often reported in the popular press involve victims who are young,[82] have fallen into cold water (= ≤ 10°C), at the time of recovery are profoundly hypothermic, and have been submerged for a relatively long time. The maximum reported immersion time of a survivor is 66 minutes.[83] The question most often asked is *why* do these individuals survive when others do not. Which scientific explanation best explains the

why remains controversial and the reason for this is best expressed by Gooden,[84] who believes that a major obstacle in the rigorous scientific study of survival from near-drowning is the paucity of objective data from the time the victim enters the water to arrival at the hospital.

Theories for the survival from near-drowning include the following:

1. externally induced hypothermia, resulting in brain and body cooling,[47] or internal and external hypothermia induced by drowning;
2. the diving response, resulting in oxygen conservation; and
3. a combination of the first two.

Published approaches to near-drowning survival either support mechanisms that combine both the diving response and hypothermia or discount the diving response as irrelevant and attribute survival to a rapid-cooling phenomenon.

If we assume that the diving response is of minimal or no importance with respect to preservation of life in the near-drowning scenario, and that the entire beneficial effect resides in the decrease in body temperature, then the water must be extremely cold and heat must be lost at a very rapid rate. Observations in subjects undergoing head-out water immersion (see Figure 17-4) show that even in very cold water (eg, 5°C), the rate of core temperature drop is only 0.06°C per minute. It must be pointed out that the body has very effective measures to counter the onset of early hypothermia (ie, peripheral vascular constriction to conserve heat and shivering thermogenesis to produce more heat). The decrease in core temperature may not be rapid enough to provide sole protection for the central nervous tissue. Hypothermia can also be induced by aspirating cold water, which if combined with external cooling, would cool the core very fast.

The diving response has been described in some detail earlier in this chapter. It is present in children, is more pronounced if stimulated by cold water, and is increased under conditions of excessive stress. Face immersion and apnea are accompanied by bradycardia, a decreased limb (ie, peripheral) blood flow, decreased kidney and splanchnic blood flow, and a decreased heat loss. The decrease in organ blood flow would tend to minimize oxygen utilization by those tissues. Also, the decreased heart rate and cardiac output will further reduce cardiac work and therefore oxygen consumption by the myocardium. The overall result is a decrease in metabolism, providing an oxygen-conserving function.[82] In real life, possibly, drowning and the diving response work together to cause a drop in core temperature.

ACCLIMATIZATION

To be consistent with the definitions given by Young,[85] cold *acclimatization* (or acclimation) means in this chapter "physiological adjustments in response to chronic cold stress."[85(p419)] On the other hand, cold *adaptation* refers to "genetic effects manifested as a result of natural selection."[85(p419)] The problem in the evaluation of cold acclimatization arises from the wide range in exposure intensity. The magnitude of the intensity is determined by surface area of exposure, ambient temperature, and environmental media (air or water). Because this chapter discusses immersion situations, acclimatization will be limited to cold water exposure.

Depending on the severity of the cold exposure, acclimatization can occur with or without an associated decrease in core temperature. For example, commercial fishermen are required to use their hands in extremely cold water for extended periods while the remainder of their body is relatively well protected from the elements.[86,87] Under these conditions, the subjects show a reduced vasoconstrictor response to cold stress. An example of this reduced vasoconstriction on exposure to cold is seen in arctic explorers (Figure 17-9). This alteration of the physiological response to cold will supply warm blood to the extremities, preventing frostbite and freezing injuries as well as prolonging dexterity. However, owing to the fact that the total area of exposure is small and the remainder of the body is protected, there will be no decrease in core temperature.

The next level of exposure would consist of repeated total body immersion for short periods of time. Under these conditions there is little or no change in core temperature; however, there is a delayed onset of shivering.[88] Because even a short immersion in cold water is a major stress, longer exposures magnify the problem and make it very difficult to measure physiological changes that may reflect acclimatization. To minimize these difficulties and evaluate acclimatization, the subject's response to cold air exposure, a much lesser stress, is evaluated following repeated cold water immersion. This approach was applied by Young and colleagues[89] and Bittel.[90] Both studies showed a de-

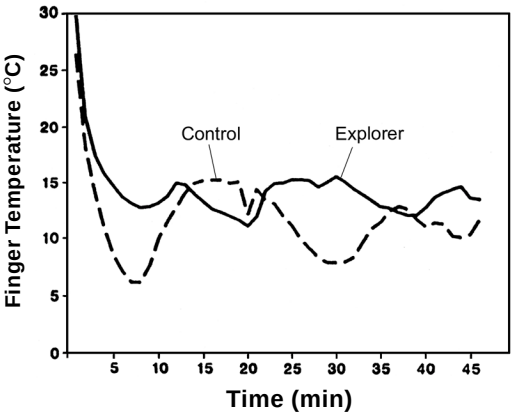

Fig. 17-9. The data represent the skin temperature of the subjects' right index finger immersed in ice water (0°C) as a function of time. The group of arctic explorers is compared with a group of controls matched for gender, age, and body type. Skin temperature is a function of skin blood flow. The explorers show much less rapid vasoconstriction when exposed to the ice water, and the magnitude of the drop is less than that seen in the control group. Adapted with permission from Hoffman RG, Wittmers LE. Cold vasodilatation, pain and acclimatization in arctic explorers. *J Wilderness Med*. 1990;1:230.

layed onset of shivering and a lower mean skin temperature in cold water-acclimatized subjects. The rectal temperature results did not match; Bittel's data showed little difference between acclimatized and nonacclimatized subjects, whereas Young's showed a lower rectal temperature at the start and a larger drop in rectal temperature in the acclimatized subjects.

The decreased skin temperature in the acclimatized subjects reduces the driving force (ie, the temperature gradient) for movement of heat from the skin into the environment (see Equation 1). This decrease in skin temperature results in a larger gradient between core and shell; therefore, a redistribution of heat internally with a decrease in shell thickness. However, the end result is an increase in effective insulation and thus, conservation of heat.

SUMMARY

The information presented in this chapter has focused on the physiological and behavioral responses to cold water immersion. When compared with cold air exposure, cold water is a much greater stress, drawing heat away from the body approx imately 20-fold faster than air of the same temperature. Protection against heat loss in cold water environments is possible, as long as personnel wear protective garments (ie, jackets and suits) as discussed. However, the suddenness of cold water accidents may preclude the opportunity to wear cold water protective gear. In such cases, the onset of hypothermia and drowning is swift and lethal.

Initially, cold stress results in peripheral vasoconstriction. This vascular response provides a method of conserving heat, essentially limiting heat delivery from the body core and increasing the external insulating layer. As the cold exposure continues, heat loss is compensated for by increased heat production, either from shivering or from muscle contraction. However, when heat loss exceeds heat production, core temperature starts to drop and the subjects proceed toward hypothermia. Changes in the individual's physiology and behavior will signal the magnitude of the core temperature drop and indicate the precautions to be taken or the treatment to be given.

It should be emphasized that cold water immersion, even when the water temperature is relatively mild (but below core temperature) has a great potential for producing the life-threatening condition of hypothermia. In addition, cold water may cause cardiovascular collapse, or drowning, or both.

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Chapter 17: ATTACHMENT

SHIPWRECK AND HYPOTHERMIA: EXPERIENCES GAINED BY THE GERMAN NAVY, 1939–1945

As a result of the loss of documents at the end of World War II, the German Naval Medical Service has not been able as yet to prepare a comprehensive medical report, but only managed to present an overview obtained by individual existing reports. Thus, it is also no longer possible to determine the number of naval personnel who died as a result of hypothermia or fatigue or the number of civilians killed during the great ship foundering in the course of the evacuation of the East in 1945. According to estimates, however, tens of thousands of people died in the icy floods.

Like the states of the western allies, the German Naval Medical Service was inadequately prepared for hypothermia problems of shipwrecked persons at the beginning of the war. Although many members of the Navy had been shipwrecked during World War I, the rescue capabilities at the beginning of the century were limited to such an extent that most persons had already died when they were recovered, and the cause of death was simply considered "drowning." Thus, an original clinical picture of hypothermia was neither assumed nor scientifically examined until the outbreak of World War II.

The 1940 publications of *The Medical Advisor for Submarines* and the *Bulletin on Medical Measures to be Taken in the Case of Submarine Accidents* did not yet deal with issues concerning hypothermia or cold water rescue. Only the improved rescue capabilities, and in particular, the events during the occupation of Norway in spring 1940, with a large number of shipwrecked persons in the cold waters of the Skagerrak and North Sea as well as the now frequent sea engagements in the northern area, forced those in command to intensively work on medical issues concerning distress at sea. The Army and Air Force only started to deal with similar problems after the mass occurrence of cold injuries during the winter battle near Moscow in 1941, and because of the large number of ditched aircraft crews suffering from cold injuries. At this point, it should be specifically pointed out that the Naval Medical Service was in no way involved in the hypothermia experiments in concentration camps, although it profited passively by the results of these experiments.

For the invasion operation, almost the entire German fleet was standing in Scandinavian waters in the morning hours of 9 April 1940. There were various sea engagements and several thousands of shipwrecked German forces. The available reports give evidence of the amazingly inadequate preparation and of the ignorance of the crews and embarked personnel, as well as of the poor medical facilities in view of the risks imposed by shipwreck and hypothermia. What alarmed the helpers again and again was the frequently observed phenomenon of the "afterdrop." After the foundering of a troop carrier, 79 members of the Army were drifting in the Skagerrak at a water temperature of 1°C. Thirty of the crew had taken off their clothes to enable them to swim better; the rest wore protective clothing. None of those who remained clothed died; after they were rescued and had changed into dry clothes they were fit for duty again. Those who had removed their clothes lay shivering in the berths of the destroyer crew after their rescue. Subjectively, they felt well and still comprehensively discussed the individual phases of the torpedoing. Suddenly, one after the other fell silent and lifelessly sank back onto the berth. They all died within 6 hours. The ship's doctor, who was overtaxed with the treatment of the casualties, immediately administered Lobelin and cardiovascular drugs to the collapsed patients, but without success (Tidow, 1960, p38).

At the same time, the cruiser *Blücher,* with a crew of 1,400 men and approximately 650 embarked Army soldiers, was torpedoed in the Oslo Fjord. The water temperature in this area was 2°C, the air temperature 0°C. The distance from the foundering ship to the dry land was only 300 to 400 meters, or about 20 minutes' swimming. Still, hundreds of soldiers died from fatigue or hypothermia. The surviving third ship's doctor, naval medical assistant Dr Pietsch, reported:

> On the smaller islands many numb persons were lying who refused to jump into the water despite shouts from the shore to swim the last meters to the shore. Upon that, I jumped into the water, reached some islands and was able ... to take 6 men ashore. In one case resuscitative measures were unsuccessful. All others were recovered in front of lighted fires some faster, others slower (Medical Action Report, pp116–117).

Only after June 1941 did the Naval Medical Office in the German Naval Supreme Command for ships and boats without doctors issue preliminary directives for the treatment of rescued shipwrecked persons. The directives demanded the *slow* warming of patients suffering from severe hypothermia. The patients were initially to be kept in cool rooms, rubbed, and packed in dry clothes. These directives, however, taking individual circumstances into account, were already slightly modified to the following:

> The decision as to which rescued persons are to be treated as patients suffering from hypothermia cannot alone be made on the basis of duration of stay in the water and the prevailing water temperature. The degree of hypothermia varies from individual to individual. Therefore, the objective impression must be also taken into consideration.

This directive attached particular importance to the careful transport of patients suffering from hypothermia on the receiving ship.

By 1942, the principles of treatment had changed. Now the *rapid* warming therapy was propagated as the best treatment method for patients suffering from severe hypothermia. This opinion was retained until the end of the war. Thus, the *Medical Advisor for Submarines* formulated in 1944:

a. Shipwrecked persons suffering from hypothermia must in any case be subjected to an immediate intensive warming. This rewarming is accomplished by means of
   - hot baths at about 45°C, or
   - hot showers at about 45°C, or
   - repeated pouring over with fairly hot water.

b. As an additional measure, cold compresses are applied to the forehead.
c. Cardiac stimulants are to be slowly injected after the body has warmed internally, otherwise there is a risk of shock.
d. The administration of alcohol has is to be avoided until circulation, respirations and consciousness are completely normal again (*Medical Advisor for Submarines,* 1944, p32).

In addition, the regulation pointed out for the first time that in cases of immersions, unnecessary swimming movements should be avoided and only should rely on the floating capacity of the life jacket.

In the course of time, the positive effect of an appropriate and adequate clothing of the shipwrecked person in the water became more and more evident and led to changes in training. The crew members who during the first years of the war had taken off their warming clothing before jumping into the water in order to be able to swim better were now "repeatedly reminded to keep all clothes on the body, whenever possible" (Missler, 1981, p19). The success of such measures became evident during the foundering of a destroyer in the polar sea in May 1942. Thirty-eight crew members, most of the personnel from the upper deck with life jackets underneath their protective clothing reached—after a longer period of swimming at a water temperature of 1°C and an air temperature of –10°C and a wind force of 5—floating parts of the wreck that were constantly flooded by the sea. Six hours after the sinking, a submarine took in the shipwrecked persons. During the examination, the rescue personnel noticed that the upper bodies were in parts still dry while the outer clothing had turned into icy armor, which had to be cut open with knives and saws. Therapeutically, the rapid rewarming was performed; however, three boiler men wearing lighter clothing died after a short time.

Various reports also point out the important role of a stable psychical constitution and a strict discipline of shipwrecked persons suffering from hypothermia. For example, in autumn 1942, a lifeboat with 4 survivors and 20 corpses reached the coast of northern Norway after a cruise of almost 9 days. At wind force 7, a sea disturbance of 5–6, with only a small supply of food and inadequate clothing, the passengers were completely soaked and suffering from hypothermia. The chief coxswain had the command in the boat and held the control fin in his arms with his last ounce of strength, because his hands were frostbitten and he could no longer use them. During the cruise many of the passengers suffered from states of delusion and confusion and force had to be used to prevent them from jumping overboard. Discipline was good until the end.

In another case, a rescued shipwrecked person in the wintry Baltic Sea in 1945 reported on a person who was shipwrecked for the second time and who, in an almost hopeless situation, motivated a group of swimmers to hold out until their rescue (compare Missler, 1981).

The extraordinarily different circumstances in the area of submarine warfare in relation to shipwreck and hypothermia is illustrated by the following report on the last mission of the submarine U 550 at the East Coast of the United States on 16 April 1944 (compare Haenert, 1994). It shows in an exemplary manner how shipwrecked submarine crews were not always taken in by enemy vessels but were sometimes abandoned to their fates:

> During a convoy battle, the type IX C submarine with a crew of 57 men was pursued with depth charges by superior US screen units and forced to surface. When the upper hatch was opened, three destroyers lying in a circle around the submarine fired at it with their internal guns. Resistance was no longer possible amidst the hail of shells; rather, the U 550 quickly foundered so that the crew left the submarine in two groups and swam away from the wreck. The only rescue facilities on board the submarine were divers and several one-man inflatable lifeboats. As protective clothing, parts of the crew wore a leather combination; others had not found the time to put on warmer clothing. The embarked naval medical assistant, Dr Torge, did not have the opportunity to take any additional lifesaving measures. The larger group of about 40 persons under the command of the first officer on watch swam toward the nearest US destroyer, as ordered. The destroyer, however, suddenly pulled away and did not make any rescue attempt. All of the soldiers died of hypothermia. The second group, 13 members of the crew, swam in the 5°C cold water for about 15 minutes to the USS *Joyce* drifting the vicinity, which took them aboard. On board, medical treatment was provided to those who suffered from moderate hypothermia or gunshot injuries.

With the activation of the small combat elements of the German Navy late in 1944, hypothermia problems gained a new significance. The crew members of these very different surface and subsurface vessels were particularly exposed to the cold. Therefore, the Navy tested cold protection measures in September 1944 for the first time. The first tests were performed on the foam suits of the Air Force, during which the soldiers noticed not only the significant insulation provided but also the good buoyancy and horizontal position in the water that the foam suits allowed. Later, the wool-in-plush underwear of the frogmen was also improved (compare Hartmann and Noeldeke, 1994).

Irrespective of all efforts aimed at improving hypothermia therapy and prophylaxis, the effects of hypothermia reached a sad climax during the disastrous foundering in the wintry Baltic Sea in the course of the evacuation of the eastern German territories. Expert prophylaxis and treatment were impossible from the very beginning, in the view of the large number of refugees. As examples of the terrible death toll at the end of the war stand two overcrowded refugee ships, the *Wilhelm Gustloff* and the *Goya*, which were torpedoed on 30 January and 17 April 1945, respectively, carrying about 6,000 people each, mainly women and children. Both ships sank and their passengers died of exposure to the cold.

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Chapter 18

MILITARY MEDICAL OPERATIONS IN COLD ENVIRONMENTS

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INTRODUCTION

Extreme cold weather poses the ultimate environmental challenge to military operations and adds special challenges to those providing medical support. Historically, harsh winter conditions have had a major impact on the outcome of military campaigns. Napoleon's Grande Armée lost all but 10,000 of its remaining 100,000 soldiers during the retreat from Russia in the winter of 1812. Although a high percentage of these injuries were secondary to frostbite and hypothermia,[1] the cold undoubtedly lessened the chances for survival among the wounded. It was during the Napoleonic Wars, however, that the concept of rapid evacuation by "flying" ambulances, or *volantes*, was developed to expedite removal of the sick and wounded.[2]

During World War I, the US Army had a total of 2,061 hospital admissions for trench foot, with more than 91,000 lost man-days secondary to cold-related injuries.[3] Cold weather injuries were also a serious concern during World War II. On Attu Island, Alaska, in 1943, 1,200 cold injuries (80 of 1,000 soldiers, or 8%) were reported during the Aleutians campaign. Afteraction reports indicated that poor equipment and poor training were major reasons for the high casualty rate.[1] The European theater also had heavy cold weather casualties. General Omar Bradley[4] described the severely crippling effect that trench foot had on the 12th Army Group as they moved toward the Rhine River in late 1944. More than 12,000 riflemen, 19% of the total casualties from this campaign, were evacuated. The decision to favor gasoline and ammunition shipments over winter clothing had been a bad choice, but alert commanders who provided drying tents for soldiers to change socks daily had fewer trench foot injuries, Bradley noted. More than 91,000 cold injuries among US Army personnel serving in all theaters of operation were officially reported by the end of World War II.[3]

Between World War I and World War II, major advances were made in resuscitative care and prevention of shock. Although the adverse effects of cold on the wounded with blood loss was appreciated during World War II, the need for external warmth to treat or prevent hypothermia in wounded patients was not universally recognized.[5] During the Korean War, 6,300 cases of cold injury were reported among US Army soldiers and Marines, 90% of which were frostbite of the feet.[3] More recently, the Falkland War (1982) produced a significant number of casualties among British Royal Marines and sailors. Nonfreezing cold injuries were a significant problem, with 70 patients with severe injuries transferred to a hospital ship. Hypothermia caused the deaths of many Argentine sailors who were forced to abandon ship.[6] In addition, management of human waste was a noted problem during the Falkland War, where the ground froze and latrines overflowed with excreta. Some cases of gastroenteritis were attributed to this lack of sanitation.[6] A more detailed discussion of cold weather operations may be found in Chapter 10, Cold, Casualties, and Conquests: The Effects of Cold on Warfare.

Successful military operations in cold climates require reliable equipment, warm and durable clothing that can be worn in layers, and motivated and extensively trained personnel who can cope efficiently with environmental conditions. Medical personnel must not only master the skills to take care of themselves in a cold climate, they must also be able to effectively treat casualties disabled by trauma or illness without allowing those patients to become hypothermic or frostbitten.

PREVENTIVE MEDICINE IN COLD WEATHER

Medical officers assigned to operational units must understand the organizational structure of the brigade and division to which they are assigned. They must also be fully knowledgeable about the organization's capabilities and mission, and be thoroughly familiar with the capabilities and limitations of the medical assets available. Specific planning for a combat mission will require the medical officer to know the current status of medical supplies, the type of terrain to be encountered, the expected weather conditions, the availability of air and ground support for patient evacuation, and the estimated numbers and kinds of casualties that are expected.

Medical officers must also ensure that they fully utilize their preventive medicine assets to assess field sanitation, including the provision of a safe and adequate quantity of potable water, adequate and properly prepared rations, and adequate latrine structures. Maintaining strict standards for field sanitation is vital to maintaining the health of any deployed force. During cold weather operations, military personnel are inclined to neglect good sanitation practices unless enforced by the command. Latrines should be downwind and 100 m from food service sites and at least 30 m from water points.[7] If the ground is frozen, burn-out latrines or plastic bags

("bag and drag" latrines) may have to be used. Latrines should be in a tent or other shelter and will need to be heated for sustained operations in extreme cold.

Proper water treatment using standard water purification methods is as essential in cold weather as it is in warmer temperatures. Bacteria and parasites may still be present in surface waters. Melted snow should not be considered safe to drink without boiling, using iodine tablets, or other appropriate treatment by trained individuals. Personal hygiene must not be neglected. For sustained operations, enough hot water should be produced to allow personnel to wash the hands after using the latrine; and to wash the feet, face, groin, and armpits daily. For personnel on the move, the disposable hand towelette found in the Meal, Ready-To-Eat (MRE) packets are an alternative to water, but only if they are kept warm. Monitoring foot care is vitally important.

When briefing the commander, the medical officer must be able to describe succinctly the following points, the first of which is the most important:

- the level of readiness of the medical units, including their proficiency to function in a harsh, cold environment;
- the health assessment of the soldiers in the command, including their physical and mental ability to conduct the mission, and the adequacy of preventive medicine measures;
- the current status of medical supplies, including blood supply and the ability to keep casualties warm and stable while awaiting evacuation; and
- the patient evacuation plan using transportation assets expected to be available, including patient-exchange points.

SURVIVAL IN THE COLD

Most newly recruited US military personnel are not accustomed to the rigors necessary to stay healthy and warm in a cold environment. Quality training, motivation, and attention to detail are essential to prevent cold-related injuries.

Keeping Warm

Medical operations in a cold weather environment can be exhausting and mentally draining. However, with proper training and equipment, medical personnel can develop the skills and confidence to perform their mission. All medical personnel must be aware of the signs and symptoms of injuries and illnesses common to a cold weather environment and the necessary measures to prevent them. Cold-related medical problems can be divided into four major areas:

1. cold injuries such as frostbite and trench foot;
2. hypothermia;
3. altitude illnesses, including acute mountain sickness, high-altitude pulmonary edema, and high-altitude cerebral edema; and
4. miscellaneous medical problems, such as snow blindness, carbon monoxide poisoning, and dehydration.

Cold weather can be characterized as two types: cold–wet and cold–dry. Cold–wet conditions occur where temperatures are near freezing, and variations in the day and night temperatures cause alternate freezing and thawing. These conditions are often accompanied by wet snow and rain, which cause the ground to become soft and muddy. With these conditions, service members require clothing that consists of a waterproof or water-repellent, wind-resistant outer layer, and an inner layer with sufficient insulation to provide protection in moderately cold weather, –10°C (14°F), or warmer. Waterproof boots and good foot care are essential to prevent nonfreezing cold injury (eg, trench foot, a term that came into use during World War I but is no longer used in the US military). Cold–dry conditions occur when average temperatures are lower than –10°C, when the ground is usually frozen and the snow is dry. These low temperatures, plus wind, increase the need for protection of the entire body. For these conditions, service members may require clothing that will protect them at a windchill factor of –62°C (–80°F). The inner layers of clothing must provide good insulation; the outer layer must be wind resistant and water repellent, yet allow for ventilation to prevent moisture buildup. The acronym COLD (*c*lean, *o*verheating, *l*oose and *l*ayered, *d*ry) can help service members remember some of the basic principles of cold weather clothing[7]:

C: Keep clothing *c*lean. Clothing keeps one warm by trapping warm air against the body and in the pores of the clothing itself. However, if these pores are filled with dirt, sweat, or other grime, the clothing will not be able to do its job as efficiently.

O: Avoid *o*verheating. Military personnel newly assigned to a cold climate tend to wear too

much clothing and overheat. Military personnel should be taught to be comfortably cool and to avoid sweating. Proper ventilation and removal or addition of clothing layers according to temperature and activity level are essential to cold weather survival.

L: Wear *l*oose and *l*ayered clothing. For comfort and to forestall some freezing injuries, clothes should be loose. Tight clothing restricts circulation and prevents the trapping of warm air between the body and clothing. Layering is another important principle for staying warm in the cold. Several thin layers are usually more efficient than one thick layer. Different types of layers are used in the Extended Cold Weather Clothing System (ECWCS), which is currently issued to military personnel assigned to cold climates.[7]

The ECWCS was developed to provide a lighter-weight, less-bulky clothing system that was better suited to the modern cold weather battlefield. This system uses synthetic materials to provide warmth and handle moisture more efficiently than the older standard clothing system. The vapor-transmission layer of the ECWCS is the sweat-transfer layer. This innermost layer soaks up body moisture and draws it away from the body to keep the wearer dry. Significant progress has been made with synthetics such as polypropylene, which draws water away from the body but stays dry. The next layer is an insulating layer. This is the layer that holds the warm layer of air around the body. Polyester pile or wool works well. Cotton should not be used, as it does not retain its insulating factor if it becomes wet. The outside layer is the protective layer, which protects the insulating layer not only from getting dirty but also from getting wet. It consists of a wind-resistant, water-repellent parka and trousers made of GORE-TEX (manufactured by WC Gore and Associates, Newark, Del)

D: Keep *d*ry. The conductive heat loss from wet clothing will rapidly reduce the body's core temperature and lead to hypothermia. The wearing of wet boots and clothing must never be permitted.

Not only proper footwear itself but also the proper *use* of the footwear are critical for all military personnel operating in cold climates. Insulated leather boots, commonly known as mountain boots, are usually adequate for active individuals down to temperatures of –18°C to –23°C (0°F to –10°F), provided that the boots have been properly treated with a waterproofing compound. Military-issue mukluks are lightweight canvas boots with a rubber sole that is lined with wool felt. These boots are excellent for vehicle drivers in a cold–dry environment, but mukluks do not provide the support needed for wearing skis. The vapor barrier (VB) boot, on the other hand, is designed to be worn both on the march and with skis. The VB boot is made of an inner and an outer layer of rubber and is filled with either wool fleece or closed-cell foam insulation. It is designed to be worn with one pair of issue socks, which are thick and insulating. VB boots are worn in a cold–dry environment and will usually protect the feet down to –46°C (–50°F) (Figure 18-1). When using these boots, it is essential to change socks whenever they are wet with sweat. The Ski March Boot System consists of several layers, including vapor transmission socks, insulating socks, vapor barrier socks, the VB boot itself, and several different overboot designs. Other boot systems have been developed and have been used on a limited basis by deploying military units. Boots should be dried whenever possible, but the wearer must be careful not to use open flames or extreme heat, which could cause damage.

Just as important as adequate boots is proper foot

Fig. 18-1. The vapor barrier (VB) boot is standard issue for military personnel assigned or deployed to arctic environments. Often nicknamed the "Mickey Mouse" boot, it provides excellent protection from cold under most conditions down to –50°F. Air trapped between two layers of rubber and a thick felt sole pad provide the insulation. A release valve (not shown) is present to relieve pressure when traveling in unpressurized aircraft. VB boots should be inspected at least annually to ensure that the trapped air layer is tightly sealed. Photograph: Courtesy of US Army Northern Warfare Training Center, Fort Greeley, Alaska.

care. A thin, moisture-transmitting sock should be worn next to the foot, with the thick, insulating issue sock worn over that. Socks may need to be changed several times daily to keep feet dry. Sweating can be reduced by using an aluminum hydroxide deodorant on the feet. Using foot powder to absorb excess moisture works well, but with boots designed to breathe (eg, mountain boots), excessive use of foot power can clog the fibrous parts of the boot and reduce their effectiveness. Gaiters, which are leggings worn over boots, may be worn for extra warmth and prevent snow from getting into boots. It is very important that the wearer not restrict circulation in the feet by lacing the cold weather boots too tightly. For sedentary individuals, it is equally important to keep some form of insulation between the ground and the footwear. Personnel working inside a heated tent can still sustain a frostbite injury to their feet if their boots are in direct contact with ice or frozen ground instead of floor boards or other suitable floor covering.

Protecting the hands is critical to cold weather survival and is the most problematic issue with regard to preventing frostbite. Accomplishing a mission in extreme climates often requires a balance between the need for dexterity and preventing cold injury. Insulated gloves are normally adequate for temperatures above –12°C (10°F) if an individual is moderately active. Trigger-finger mittens with wool inserts usually provide good protection between –12°C and –29°C (10°F and –20°F). Arctic mittens are necessary at colder temperatures. At temperatures below –18°C (0°F), contact gloves (thin, snug-fitting gloves made of synthetic fiber) should also be worn, to allow the wearer to remove his mittens for short periods when dexterity is needed to perform tasks. Mittens should always be attached to lanyards, to allow them to be removed without being lost. When not in use, arctic mittens should be carried inside the parka to keep them warm.

Keeping warm involves wearing enough clothing to keep from losing body heat and removing layers as necessary to avoid overheating. Service members must be taught to take immediate action to warm their extremities at the first sign of cold-induced pain or numbness. Changing socks, running in place, or both, will warm feet that are just beginning to get too cold. Likewise, increasing activity, doing "windmill exercises" with the arms, or putting on warm mittens will rewarm hands just beginning to get numb. Chemical heating pads, available commercially, are useful for rewarming feet and hands but should not be used as a substitute for adequate clothing.

Sleeping in an unheated structure in a cold climate requires a fair amount of skill to keep warm. Good insulation between the sleeping bag and the ground is essential to prevent the rapid loss of body heat. Closed-cell foam pads, air mattresses, or small branches from a conifer will provide adequate insulation; take care that plenty of insulation is placed between the feet and the ground to prevent frostbitten feet. Remember that the body heats the sleeping bag; the bag only insulates. On top of the sleeping bag, any waterproof cover will hold the heat in the bag. Sleeping bags should be shaken to ensure maximum loft of the insulating material. The flap should be pulled over the zipper to prevent freezing and heat loss. Service personnel should never sleep in the clothes that were worn all day (cold weather clothing changes are accomplished inside a heated tent or the individual's sleeping bag). Clothes damp with sweat will soon cool the body. Clothes and boots should be placed inside or directly underneath the sleeping bag. A balaclava (a knit cold weather hat) should be worn while sleeping to reduce heat loss from the head. A carbohydrate meal eaten just before bedtime will provide metabolic fuel during the night.

Nutrition and Hydration

Good nutrition and hydration are as essential as warm clothing to functioning well in cold weather. The average person living and working in cold weather needs 25% to 50% more calories than their normal intake, depending on activity level. The average male soldier requires 4,500 calories per day during cold weather field training, and the average female soldier requires 3,500 calories per day under the same conditions.[8] The cold weather ration currently issued to US military personnel provides these needed calories. As often as possible, meals should be served hot, which provides additional warmth as well as improves morale. Tent stoves, cook stoves, or the chemical heater issued with the MRE packets work well under most conditions. In addition to regular meals, frequent high-calorie snacks and hot, non-caffeinated beverages or soups should be provided. High-fat diets are an efficient way to provide needed calories, but carbohydrate sources of calories may be better tolerated by the average service member.[8]

Adequate hydration can be problematic in arctic and subarctic conditions. Service members often do not feel thirsty even though they are operating in an atmosphere as dehydrating as any desert. Depending on activity level, each person will need at least 6 liters of fluid per day. For units operating away from motorized vehicles, obtaining water and preventing it from freezing are difficult chores. Water containers transported on a squad sled

known as an *ahkio* (from the Finnish, pronounced ah-kee-oh) soon freeze, as does water in the standard-issue arctic canteen. Many experienced military units operating in cold environments use nonstandard thermos containers or water storage vessels that may be worn inside a parka. Canteens, if used, should be filled two-thirds full and then wrapped in a pack or kept in the sleeping bag. Just before going to sleep, service personnel can fill their canteens with hot water and place them inside the bag. This will not only keep the canteens from freezing, but will also warm the bag.

Shelter and Heaters

The Arctic 10-Man Tent is currently the standard tent used by most US military forces deploying to extremely cold regions. This tent can be managed by a squad, has a liner, and can be warmed effectively by a variety of stoves. It can be carried by an ahkio. The tent has ventilation openings and accommodates a stove pipe. To avoid a fire hazard, safety must be the utmost concern when using a stove inside the tent. The sides of the tent must not be staked down to allow rapid egress in the event a stove flares up. Good ventilation is essential to prevent carbon monoxide poisoning or loss of oxygen. Stoves should never be operating when all tent occupants are asleep.

Another shelter system currently used is the Modular Command Post System. This special tent is designed to use a heating system that will allow temperature-sensitive equipment to operate. This system can also be used to provide medical care.

The stove most commonly used by US military units in cold climates is the M1950 Yukon Stove. This simple but effective stove will burn wood or liquid fuel dripped onto a burner. Diesel fuel, kerosene, or gasoline may be used, but gasoline has proven to be the most efficient fuel. Gasoline is also the most dangerous fuel, especially when it is not handled properly. The US Army Research, Development, and Engineering Center, Natick, Massachusetts, has developed the Family of Space Heaters (FOSH) (Figure 18-2). These stoves are designed to operate without external electric power, use all types of liquid or solid fuel, and work at temperatures as low as –51°C (–60°F). Because they control the flow of fuel through a regulated vaporization step, these heaters are designed to be safer than the M1950 Yukon Stove. A thermoelectric fan powered by the heat of the stove helps improve efficiency by circulating warm air.[9]

Mobility

Traveling over snow and ice can be challenging for military personnel who have never participated in outdoor winter activities. Medical personnel will have to master basic snowshoe and skiing skills to conduct their mission in snow country. Lightweight, tracked vehicles designed for off-road movement in snow greatly enhance both military operations and the ability to evacuate casualties.

Fig. 18-2. The Family of Space Heaters (FOSH) was developed to provide safer, more efficient heaters for field use. Each stove is capable of using multiple fuels including diesel, JP8, JP5, kerosene, wood, and coal. (**a**) The Space Heater, Convective (SHC) is a 35-kBTU, 67-lb heater that provides forced hot air to heat tents. It can be operated inside or outside the shelter. The SHC has a thermoelectric heater that uses waste heat to generate enough electricity to power the blowers, pumps, ignition system, safety system, and controls. (**b**) The Space Heater, Arctic (SHA) is a 28-kBTU heater designed to heat the Arctic 10-Man Tent and other tents with 100 to 200 ft^2 of floor space. Weighing 35 lb, it was designed to be mobile and easy to assemble. (**c**) The Space Heater, Small (SHS) is a 12-kBTU heater designed to heat the Soldier Crew Tent (5-man tent) and other tents with 80 to 100 ft^2 of floor space. It weighs 19 lb, including an integral fuel tank. (**d**) The Space Heater, Medium (SHM, or H-45) is designed for general purpose and TEMPER (*t*ents, *e*xtendable, *m*odular, *per*sonnel) tents. It replaces the older M-41 heater, which had operational and safety problems. The H-45 weighs 70 lb. The SHA and SHM heaters utilize an attached thermoelectric fan (shown atop the SHA, b) that circulates heat down to the tent floor, improving heat distribution and providing fuel savings. Photograph: Mr Joe Mackoul, Project Engineer, US Army Soldier Systems Center, Natick, Mass.

Fig. 18-3. The standard issue snowshoe is lightweight, versatile, and easy to learn to use. This soldier is wearing vapor barrier (VB) boots (see Fig. 18-1) and the Extended Cold Weather Clothing System (ECWCS), with winter overwhites for camouflage. Photograph: Courtesy of US Army Northern Warfare Training Center, Fort Greeley, Alaska.

Snowshoes

Personnel can master the necessary skills to walk in snowshoes in a short period of time (Figure 18-3). Snowshoes require very little maintenance and allow movement in densely wooded terrain. They are particularly useful for individuals working in confined areas, such as around bivouac sites and supply dumps. However, they are not an efficient means to travel long distances because they are slow and require a great expenditure of energy.

Although the types of snowshoes vary, the concept remains the same. The magnesium snowshoe commonly issued to US military personnel is light and durable. The nylon binding used with this snowshoe is adaptable to all types of issued footwear. The magnesium snowshoe also has "teeth" on the bottom, which aid in traction. An additional feature in a survival situation is that magnesium shavings from the snowshoe frame make an excellent fire starter.

Skiing

Skis, once mastered, are much faster and more efficient than snowshoes in open terrain. Military personnel unfamiliar with their use, however, may require several weeks of training to be proficient with them. The backcountry or mountaineering ski commonly issued to arctic units is a cross between Nordic (cross-country) and alpine-type (downhill) skis. They have a metal edge and cable bindings that allow free movement of the heel. This binding is designed to fit the VB boot (Figure 18-4). The mountaineering ski functions adequately under a variety of conditions and can be used in steep terrain by a skilled individual.

a

b

Fig. 18-4. (**a**) The military ski is a metal-edged mountaineering ski suitable for downhill and cross-country skiing. The soldier in this photograph is making a telemark turn. (**b**) The detachable fabric "skins" seen on these military skis provide traction for steep uphill climbs. Photographs: Courtesy of US Army Northern Warfare Training Center, Fort Greeley, Alaska.

Fig. 18-5. These US Marines are extending their range by skijoring behind a Small Unit Support Vehicle (SUSV): the tracked vehicle is towing them. Skijoring requires balance, but the technique is easily mastered. Unit members attach themselves by wrapping one turn of the towrope around the ski poles, which are held under the arm. For safety, skiers are not directly attached to the towrope.

Nordic skiing uses a "kick and glide" action. The skis have a bow, called a camber, in the center portion. When fully weighted on one ski, this center portion flattens to allow the ski to grip the snow, thus allowing the kick. The tips and the tails of the skis provide the glide. Skis must be waxed, using the appropriate wax for the temperature and snow conditions. For ascending steep inclines, a fabric "skin" can be attached to the entire length of the ski bottom to provide grip.

Unit mobility over long distances can be enhanced by towing ski-mounted service members from a vehicle. This technique, called *skijoring*, works well with seasoned troops, but care must be taken to prevent frostbite caused by the windchill (Figure 18-5).

Ground Vehicles

Requirements for ground vehicles will vary depending on the type of operation and the terrain. Wheeled vehicles are frequently limited to paved roads. Chains are frequently required, even on vehicles with four-wheel drive. Tracked vehicles are often of limited utility because of their heavy weight. The standard vehicle used by most US military units deployable to arctic and subarctic regions is the Small Unit Support Vehicle (SUSV). This lightweight, tracked vehicle can negotiate most types of terrain, pull skijoring units and supplies, or evacuate patients. Snowmobiles are quite versatile in snow country but are not standard equipment for most units. They can move rapidly and pull ahkios.

MANAGEMENT OF CASUALTIES IN THE EXTREME COLD

Managing seriously injured casualties in an extremely cold environment (≤ –20°F wind chill equivalent) is difficult, especially if there has been significant blood loss or the casualty cannot be rapidly evacuated to a warm place. The basic tenets of emergency trauma care as taught in the American College of Surgeons' Advanced Trauma Life Support[10] course remain the same, but the medical care provider may need to balance the casualty's need for medical stabilization against the risk of hypothermia or frostbite from exposure. A review of a mass casualty situation during an arctic winter illustrates this point well (Exhibit 18-1).

The detrimental effect of hypothermia on trauma victims has been noted in several studies. In a study of adult trauma patients, Jurkovich and colleagues[11] found that trauma victims with similar Injury Severity Scores had significantly higher mortality rates when their core temperature decreased compared with those whose core temperatures were normal. In this study, no trauma victim with a core temperature below 32°C survived. In a study of 94 trauma victims, Luna and colleagues[12] reached similar conclusions. Both studies indicated that an increased risk for hypothermia existed when large volumes of blood or crystalloid fluids were infused.

Locating Casualties

Casualties dressed in camouflage whites can be difficult to find in the snow. Ice fog and lack of daylight during the winter months in the extreme Northern Hemisphere (or extreme Southern) can further challenge search efforts. Successful rescue requires finding and evacuating wounded personnel rapidly. Those conducting the search should ascertain the last known position of missing service members and look for signs of equipment and tracks. Search efforts must be rapidly organized, with defined search grids for all parties involved. Good communication is vital. If a rapid systematic search fails to locate the individual, then infrared detection devices, if available, should be used—from either the ground or the air. These devices

EXHIBIT 18-1

TRAUMA AT FIFTY BELOW ZERO

In January 1989 a military C-130 aircraft crashed as it approached the runway at Fort Wainwright, Alaska. The temperature was –47°C (–51°F) with calm winds and ice fog. Visibility was approximately 200 m. Of the 18 passengers and crew, 7 were dead at the scene from multiple trauma, which included head, abdominal, and chest injuries. An eighth soldier, who sustained a subdural hematoma, died 3 days after the crash.

Rescuers required approximately 120 minutes to extract a ninth casualty, whose injuries consisted of a comminuted lower leg fracture and blunt chest trauma, from the wreckage. Attempts to keep the ninth casualty warm with blankets and a portable space heater proved unsuccessful. Fluid administration was attempted during the extraction process but proved impossible because the intravenous fluid would rapidly freeze and the plastic tubing would shatter. When he arrived at the hospital, the casualty's core temperature was 28°C (82°F). He was rewarmed with infusions of warmed intravenous fluids in addition to peritoneal lavage, and intubated and ventilated with warm air. Despite these efforts, he went into ventricular fibrillation, progressed to asystole, then died 75 minutes after resuscitation began.

A tenth casualty, found injured in a snowbank, had a skull fracture with intracerebral hemorrhages, abdominal injuries, and deep facial lacerations that bled profusely but only after the casualty was rewarmed in the emergency room. This individual, who arrived at the hospital 1 hour after the crash, was wearing arctic gear except mittens. He suffered deep frostbite to both hands, but recovered. Most of the other victims suffered mild hypothermia and frostbite in addition to their injuries from the crash.

Source: Johnson DE, Gamble WB. Trauma in the arctic: An incident report. *J Trauma.* 1991;31:1340–1346.

have proven to be highly effective for locating people.[13]

Assessing the Patient

As with any trauma victim, a rapid initial assessment must be completed on a casualty, even in extreme cold weather. The assessment should be done with minimal, if any, removal of clothing. Ensure that an airway is established and maintained with cervical spine control and that the patient is breathing adequately. Cardiopulmonary resuscitation should be rendered as necessary in the best manner possible. Severe bleeding should be stopped with a pressure bandage, or a constricting band if necessary, in the event of a traumatic amputation or severed artery. Severe extremity wounds may have little or no bleeding, however, secondary to vasoconstriction from the cold environment. Estimating blood loss by pulse, skin color, or capillary refill will not be useful after prolonged exposure to subfreezing temperatures.

If a spinal injury is a possibility, care must be taken to immobilize and lift the victim carefully. Obvious fractures should be immobilized to prevent further damage, but splinting should normally be delayed in favor of expediting evacuation, especially if the patient is to be placed on a sled or litter. Intravenous fluid replacement in the field at extremely cold temperatures is not feasible. Veins constrict, making the establishment of a patent intravenous line difficult. The exposure and delay could lead to hypothermia, and the intravenous line will quickly freeze if left exposed to the outside air. Once immediate life-threatening problems are stabilized, the casualty's best chance for survival is rapid evacuation to a warm treatment site.

Management at a Battlefield Medical Treatment Facility

Once a casualty has been transported to a battalion aid station or other battlefield medical treatment facility, a more thorough assessment can be made. Intravenous fluids will need to be warmed (to 39°C) to prevent hypothermia.[10] Normal saline may be more practical to carry and use in forward areas than Ringer's lactate. Usually the preferred replacement fluid for victims with traumatic blood loss, Ringer's lactate may precipitate after freezing; normal saline does not. In a battalion aid station, crystalloid solutions can be heated in a water bath using a stove or, preferably, an electric heater if a generator is available. In combat area support hospitals, microwave ovens can be used to warm crystalloid fluids

except glucose-containing solutions, blood, and plasma. If a patient arrives with significant hypothermia, intravenous fluids should be warmed to 39°C (43°C).[14]

In an extremely cold environment, keeping a casualty warm is an integral part of emergency management. Tents may be difficult to warm, especially when temperatures are extremely low and the wind is blowing, so casualties must be kept inside prewarmed arctic sleeping bags (Figure 18-6). Several effective heat sources for individual patients are available, but not all are commonly used by military units subject to deployment in cold regions. Chemically activated heating pads will last several hours when placed inside a sleeping bag. A surprisingly safe charcoal heater known as the Norsk Personnel Heater (Figure 18-7), a Norwegian innovation, uses a battery-operated fan to provide an individual heat source.[15] This device also comes with a humidifier. Both of these heat sources will keep an individual warm when used appropriately but are not efficient for rewarming a seriously hypothermic casualty.

Intravenous solutions, once warmed, must be kept warm. In a shelter or tent, insulation placed on the bag of infusate and the tubing may be adequate if the temperature is not too cold. Otherwise the apparatus will have to be placed inside the sleeping bag with the casualty, with a pressure cuff to maintain flow. Portable devices capable of delivering warm, humidified air to casualties in the field are available. These greatly increase the amount of heat that can be delivered. A thermometer, if used, should be placed in the airway tubing to monitor the temperature. To prevent injury to the casualty's bronchi, the temperature should be between 42°C and 46°C.

Fig. 18-6. Care of a trauma victim is extremely challenging in a cold environment. The need for exposure for wound care and intravenous access must be balanced with the risk of hypothermia.

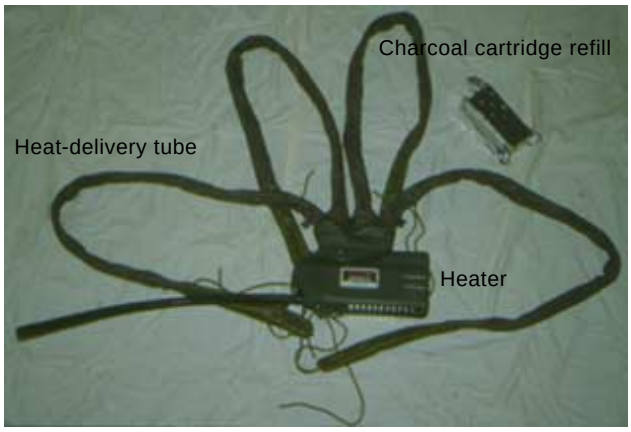

Fig. 18-7. The Norsk Personnel Heater, for individual use, is currently part of the US Army medical inventory for warming casualties. The heater, which measures approximately 9 x 5 x 2 in., provides 40 to 250 W of heat for 6 to 20 hours on one charcoal cartridge. The flexible tubes are placed around the casualty's body to circulate heat. A unique, specially designed catalytic converter converts carbon monoxide to carbon dioxide.

Establishing a medical station (US Navy) or battalion aid station (US Army) requires proper planning. Avoid avalanche zones; make sure the aid station is in a good defensive position, such as a forested area, and that it is near a good water supply. Ensure that good track discipline (ie, minimize the trail size by keeping infantry and vehicle tracks in line and in shadows or near trees) is used when traveling to the aid station to lessen the chance of enemy detection. Among units deployed to the arctic, the 10-man mountain tent is used as a battalion aid station more than any other shelter. When running an aid station out of this or other similar shelters, some form of improvised flooring such as plywood should be used, if possible, to keep feet warmer. On tundra, the ground will become wet and soggy without a floor as the tent becomes warmer. In extreme cold, the temperature at ground level will likely remain below freezing, especially near the door and edge of the tent. Therefore, it is very important to keep medications elevated to prevent damage from freezing. Table 18-1 lists the stability after freezing of commonly used medications in a deployment setting.

In highly mobile settings, it may be expedient to establish an aid station in an ambulance or other vehicle such as an SUSV, particularly if the predicted number of casualties is light. An SUSV may provide a warmer and cleaner environment with a

TABLE 18-1

STABILITY OF MEDICATIONS AFTER FREEZING

Medication	Stability After Freezing ≤ 0°C
Normal saline	Can be used upon rewarming
Ringer's lactate	Unstable
Lidocaine 1% injection	Can be used if clear
Sodium bicarbonate injection	Unstable
Naloxone hydrochloride	Unstable
Diphenhydramine injection	Can be used upon rewarming
Tetanus toxoid	Unstable
Meperidine hydrochloride	May be used if clear
Calcium chloride 10% solution	Unstable
Penicillin GK injection	Stable in powder form
Procaine penicillin G injection	Stable; potency is retained
Furosemide injection	Stable; potency is retained
5% dextrose in water, 5% dextrose in normal saline	Unstable
Morphine sulfate: injection solution or tablets	Potency is retained; insoluble particles may form
Prochloroperazine: injection, solution, tablets, capsules	Unstable
Promethazine injection, tablets, suppositories	Unstable
Antacids	Potency is probably not affected, but emulsions may separate
Epinephrine injection	Unstable
Diazepam injection	May be unstable; thaw in warm water bath; use if no precipitate is visible

Source: Auerbach PS, Geehr EC, eds. *Management of Wilderness and Environmental Emergencies.* 2nd ed. St Louis, Mo: Mosby; 1989: 356–357.

ready source of electrical power, but use as an aid station will encumber a vehicle.

Military Medical Evacuation

The method of military medical evacuation can take many forms; however, the chain of participants remains relatively constant. The infantryman is often overlooked as a member of the treatment team, but all service members are trained in "self-aid" and "buddy-aid." The hospital corpsman or medic attached to each company is the next link in the chain. They provide first aid and emergency procedures; continual observation; and care to ensure that the casualty's airway is open, bleeding has ceased, and circulatory shock and further injury are prevented. Corpsmen and medics also ensure that medical supplies are used effectively and make requests for air or ground ambulance, as appropriate. The unit medical station or the battalion aid station is the first link in the chain of evacuation where a casualty can expect to see a physician or physician assistant.

To provide the best chance for a casualty to survive evacuation to the next-higher echelon of care, all service members should be taught the following guidelines:

- Apply essential first aid to treat life-threatening conditions and stabilize the patient.
- Obtain the fastest means of evacuation available. Use aviation support whenever possible.
- Warm the casualty if necessary and keep him warm. Protect from the elements.
- Check on the casualty's condition frequently, but avoid unnecessary handling and exposure. In extreme cold, the casualty's breathing rate, mental status, and subjective reporting of pain and feeling may be the best indicators of his condition.
- If evacuation must be by ground, select the easiest route, by using scouts if necessary. If the route is long and arduous, set up relay points and warming stations using minimal medical personnel. Normal litter teams must be augmented in arduous terrain.
- Do not separate the casualty from his gear.

Fig. 18-8. The akhio is a fiberglass sled designed to carry up to 200 lb of equipment. Infantry squads usually use them to carry their 10-man tent, stove, fuel, water, and other equipment. The akhio can also be used to evacuate a casualty. The sled weighs 38 lb and measures 88 x 24 x 8 in. Three rails on the bottom help the sled track in a straight line. Maneuvering the fully loaded akhio requires the coordinated efforts of up to four individuals, especially on turns. A knotted rope, seen in this photograph, is placed beneath the akhio as a brake for downhill runs. Photograph: Courtesy of US Army Northern Warfare Training Center, Fort Greeley, Alaska.

In snow, putting the casualty on the standard ahkio and towing it is usually the easiest method of manual evacuation (Figure 18-8). The ahkio can be pulled by snowmobile or loaded into a SUSV or helicopter. The ahkio is large enough to accommodate one casualty and most, if not all, of his survival gear. To evacuate a casualty using an ahkio, pad the bottom with one or more sleeping mats. The patient may be placed in a standard issue sleeping bag or an evacuation bag, which is a larger bag that provides room for extra medical items. In extreme cold or for a lengthy evacuation, a Norsk Personnel Heater or chemical heat pad should be used to provide additional warmth. A poncho or other suitable vapor barrier should be placed on the outside of the sleeping bag to prevent heat loss. The canvas cover should then be placed over the casualty except for the face. Ths casualty should be lashed securely but not too tightly. The casualty's head should be slightly elevated and placed to the rear of the ahkio, unless the evacuation route is downhill; then the head should be placed to the front. Evacuation by stretcher can be arduous if the casualty must be carried a great distance over rough terrain. It takes up to eight individuals (squad size) rotating turns to complete an evacuation this way.

Medical evacuation by air is the ideal method to transport a critically injured casualty to the definitive care needed to preserve life. The extreme cold found in the Arctic, Antarctic, and at high altitude can place limitations on aircraft, however, and especially on their navigation systems. Visibility is often poor secondary to snow and ice fog. Aircraft may also be limited by the difficulty of finding a landing zone, the height of an obstacle, the size and topography of the landing zone, and the wind direction and velocity. Personnel on the ground must ensure that the landing zone is free of obstacles, that the snow is firmly packed down in the landing zone, and that the landing zone is appropriately marked.

When a rotary aircraft lands, care must be taken to avoid frostbite from the windchill produced by the rotating blades. As with all requests for air evacuation, establishing good communication is vital.

SUMMARY

Medical operations in cold environments require constant vigilance by medical personnel and the chain of command to prevent disease and nonbattle injury and to limit morbidity and mortality of combat casualties. Good planning, careful oversight, and thorough training are essential. Management of casualties in an extremely cold environment greatly challenges the resources and ingenuity of the medical team. Rapid assessment, stabilization, and evacuation will, in most situations, be the key to survival for casualties with significant injuries. All healthcare providers, from the soldier in the field providing basic first aid to the physician at a field hospital, must take aggressive measures to prevent hypothermia from occurring.

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Appendix

PICTORIAL ATLAS OF FREEZING COLD INJURY

WILLIAM J. MILLS, JR., MD*

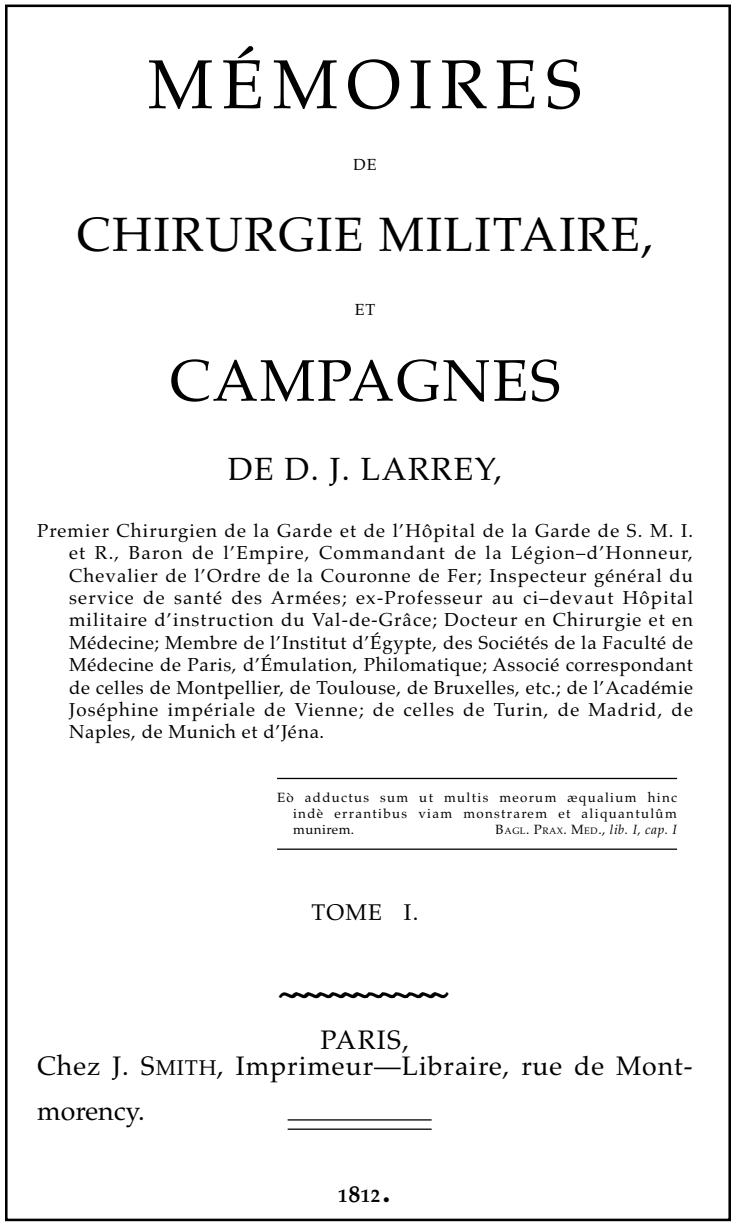
*Rear Admiral, Medical Corps, US Navy (Ret); 1544 Hidden Lane, Anchorage, Alaska 99501

HISTORY OF AND INTRODUCTION TO FREEZING COLD INJURY

a

b

Atlas Fig. 1. (**a**) One of history's oldest written accounts of an army decimated by cold was that of Xenophon, who in 400 BC led 10,000 Greek soldiers from Sardis to Babylon and back, through the mountains of Armenia, battling the hazards a retreating and disorganized army faces when pursued by the combined, unrelenting foes of severe cold weather and harassing enemy forces. Warmth was obtained by campfire heat; friction; massage of the body parts with greases, oils, and unguents; and preservation of heat by covering up in snow. Amputation and death from exposure were common. (**b**) As this chart from Xenophon's account shows, the course of his army was not far from the ancient city of Babylon, quite near the city of Baghdad, the present-day capital of Iraq. Pursuit of the 1991 Persian Gulf War past Baghdad into the mountains of Kurdistan and the ancient trail of Xehophon's Greek army might have brought US troops into a winter war that would have required quite different preparation. Reproduced from Xenophon. *The Expedition of Cyrus Into Persia, and the Retreat of Ten Thousand Greeks, 400 BC.* Spelman E, trans. 2 vols. London, England: D. Brown; 1749.



MÉMOIRES

DE

CHIRURGIE MILITAIRE,

ET

CAMPAGNES

DE D. J. LARREY,

Premier Chirurgien de la Garde et de l'Hôpital de la Garde de S. M. I. et R., Baron de l'Empire, Commandant de la Légion–d'Honneur, Chevalier de l'Ordre de la Couronne de Fer; Inspecteur général du service de santé des Armées; ex-Professeur au ci–devaut Hôpital militaire d'instruction du Val-de-Grâce; Docteur en Chirurgie et en Médecine; Membre de l'Institut d'Égypte, des Sociétés de la Faculté de Médecine de Paris, d'Émulation, Philomatique; Associé correspondant de celles de Montpellier, de Toulouse, de Bruxelles, etc.; de l'Académie Joséphine impériale de Vienne; de celles de Turin, de Madrid, de Naples, de Munich et d'Jéna.

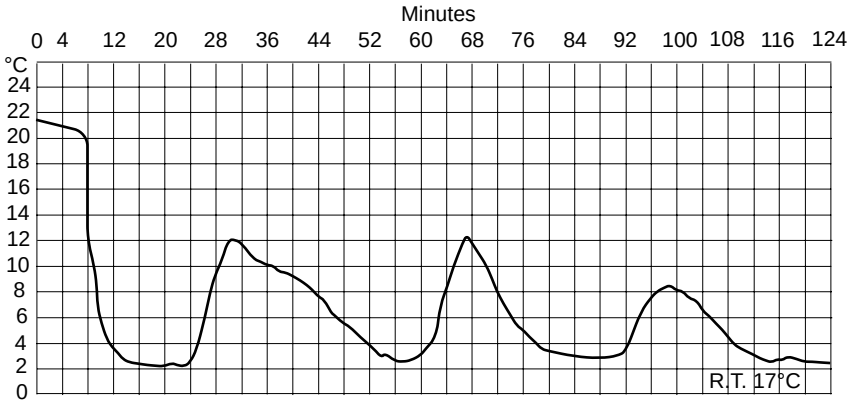
Eò adductus sum ut multis meorum æqualium hinc indè errantibus viam monstrarem et aliquantulûm munirem. BAGL. PRAX. MED., *lib. I, cap. I*

TOME I.

PARIS,
Chez J. SMITH, Imprimeur—Libraire, rue de Montmorency.

1812.

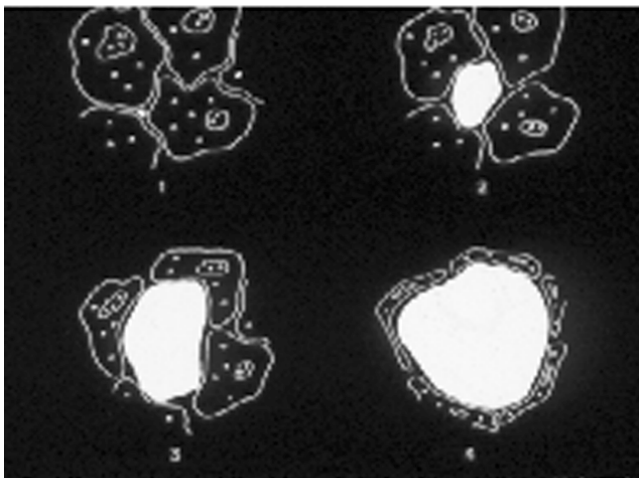
Atlas Fig. 2. A detailed, factual, and vivid description of the effects of cold on a retreating army was documented by Baron Dominique-Jean Larrey, the chief surgeon of Napoleon's Grande Armée in the retreat from Moscow in 1812/13. Larrey's *Mémoires de Chirurgie Militaire et Campagnes* gave precise description of freezing injury and its etiology, including data on general body cooling. He recommended slow rewarming, or delayed warming with ice and snow techniques, and friction massage, all out of favor now although accepted for more than 150 years after his reports. The monograph is replete with the problems of prevention and care involving massive numbers of troops, in an army in which more than 80% of its force perished from cold and cold-related problems. Much can still be gained by a study of this classic. Larrey disapproved of rapid rewarming, and his words discouraged the use of rapid thawing until the latter half of the 20th century. He did note the disastrous effect of excessive heat, apparently recognizing that frozen extremities, warmed in the close proximity of bivouac fires, sustained a second thermal injury, a burn, with disastrous effect. For the student of freezing injury, this monograph is highly recommended.



Atlas Fig. 3. Peripheral temperature curve of a finger immersed in ice water, demonstrating the "rise and fall" response, gradually decreasing, which is known as the "hunting response of Lewis." The ice water was unstirred; the room temperature was 17°C. Reprinted with permission from Lewis T. Observations upon the reactions of the vessels of the human skin to cold. *Heart*. 1930;15:183.

Atlas Fig. 4. By definition, frostbite is a result of heat loss sufficient to permit ice formation in the superficial or deep tissues. According to H. T. Meryman, one of America's premier cryobiologists:

> The single most important and fundamental concept in biological freezing is that regardless of the mysterious complexity of the biological matrix, freezing represents nothing more than the removal of pure water from solution and its isolation into biologically inert foreign bodies, the ice crystals.[1]



In Meryman's representation of the freezing process in tissue, (1) some "substance" in the fluid of the extracellular space permits a nucleus of ice to form. (2) The extracellular solution has been concentrated from freezing and the resultant high osmotic pressure in the extracellular fluid draws intracellular water through the cell membrane. (3 and 4) The continuous growth of extracellular ice permits mechanical pressure to be exerted on cells, compressing them. This results in progressive cell dehydration, a condition more damaging to the cell membrane than the mechanical effect of the ice crystals. Quotation: (1) Meryman HT. Mechanics of freezing in living cells and tissues. *Science*. 1956;Sep 21:124. Drawing: Reprinted with permission from Meryman HT. Preservation of living cells. *Federation Proc*. 1963;22:82.

ATLAS EXHIBIT 1

TYPES OF FREEZING INJURY

- Frostbite, superficial to deep
- Mixed injury (immersion followed by freezing)
- Freeze–thaw–refreeze
- High altitude, hypoxia, freezing
- Compartment compression and freezing
- Extremity fracture and freezing
- Freezing injury superimposed on severe hypothermia
- Hypothermia following any form of cold injury (trench foot, immersion injury, frostbite)

ATLAS EXHIBIT 2

CELLULAR RESPONSE TO FREEZING AND THAWING

During Freezing

1. Growth of ice crystals damages cell structure
2. Electrolyte concentration causes protein denaturation
3. Intracellular and extracellular pH becomes more acidotic
4. Cells dehydrate
5. Protein-bound water decreases

During Freezing and Thawing

6. Cell membranes rupture
7. Intracellular organs are damaged
8. Cell membrane permeability is abnormal
9. Essential enzymes are destroyed
10. Microscopic damage occurs to capillaries
11. Arterial and venous walls are injured
12. Mitochondrial damage to muscle cells is consistent

ATLAS EXHIBIT 3

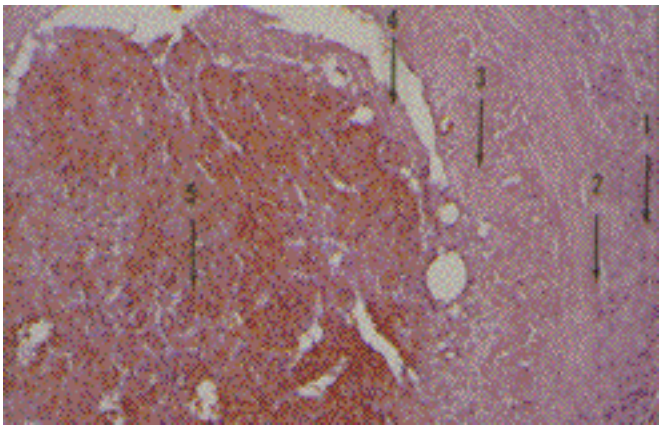
VASCULAR CHANGES DURING AND AFTER THAWING

Vascular Changes During and Soon After the Thaw

1. Circulatory stasis
2. Corpuscular aggregation
3. Venule and arterial obstruction
4. Red blood cells pile up back to the capillary bed
5. Hyaline plugs develop in the vascular tree
6. Progressive dermal ischemia, which is worsened by (or perhaps a component of which may be caused by) the activation of the arachadonic acid cascade

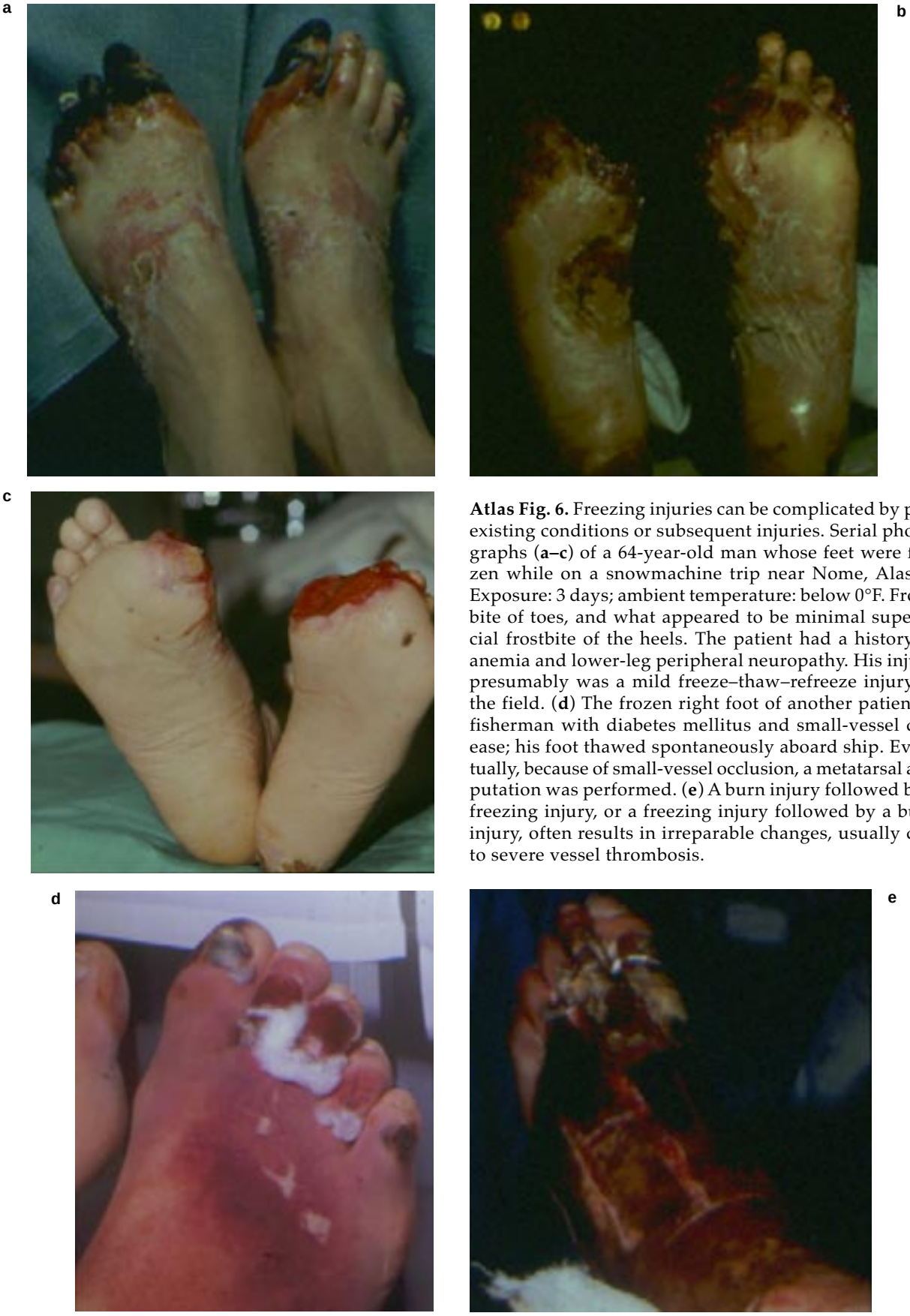
Vascular Changes After the Thaw

7. Marked tissue edema
8. Anoxia and ischemia of tissues
9. Increased tissue compartment pressure
10. Capillary and peripheral vessel collapse
11. Thrombosis, ischemia, necrosis, gangrene



Atlas Fig. 5. Long-standing wet–cold exposure, followed by freezing. This section of a small peripheral artery of the foot shows (1) adventitia demonstrating nonspecific inflammation; (2) tunica media with less obvious inflammation, with spindle-shaped muscle fibers; (3) thickened intima and circumferential muscle fibers; (4) endothelial disruption growing into a luminal clot, with endothel-ialization of thrombus; and (5) an intraluminal clot being organized, with ingrowth of fibroblasts and capillaries. This pathological pattern may be found in freezing as well as peripheral nonfreezing injury. The endothelial cell and its lining are sensitive to, and early targets of, low temperatures.

Unless otherwise noted all figures are from the professional collection of William J. Mills, Jr., MD. Some of these illustrations have also been published in *Alaska Medicine.*



Atlas Fig. 6. Freezing injuries can be complicated by preexisting conditions or subsequent injuries. Serial photographs (**a–c**) of a 64-year-old man whose feet were frozen while on a snowmachine trip near Nome, Alaska. Exposure: 3 days; ambient temperature: below 0°F. Frostbite of toes, and what appeared to be minimal superficial frostbite of the heels. The patient had a history of anemia and lower-leg peripheral neuropathy. His injury presumably was a mild freeze–thaw–refreeze injury in the field. (**d**) The frozen right foot of another patient, a fisherman with diabetes mellitus and small-vessel disease; his foot thawed spontaneously aboard ship. Eventually, because of small-vessel occlusion, a metatarsal amputation was performed. (**e**) A burn injury followed by a freezing injury, or a freezing injury followed by a burn injury, often results in irreparable changes, usually due to severe vessel thrombosis.

METHODS OF THAWING

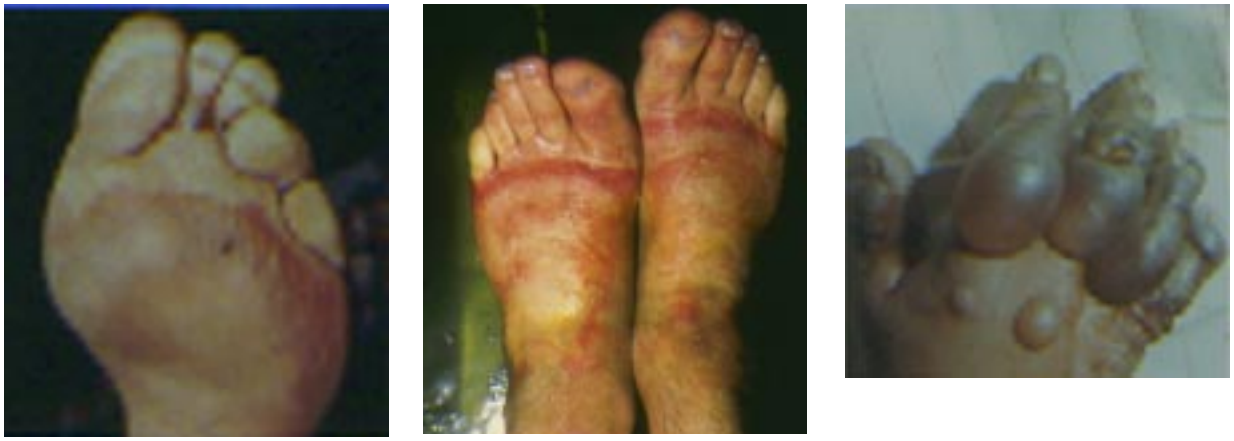
ATLAS EXHIBIT 4

METHODS OF THAWING

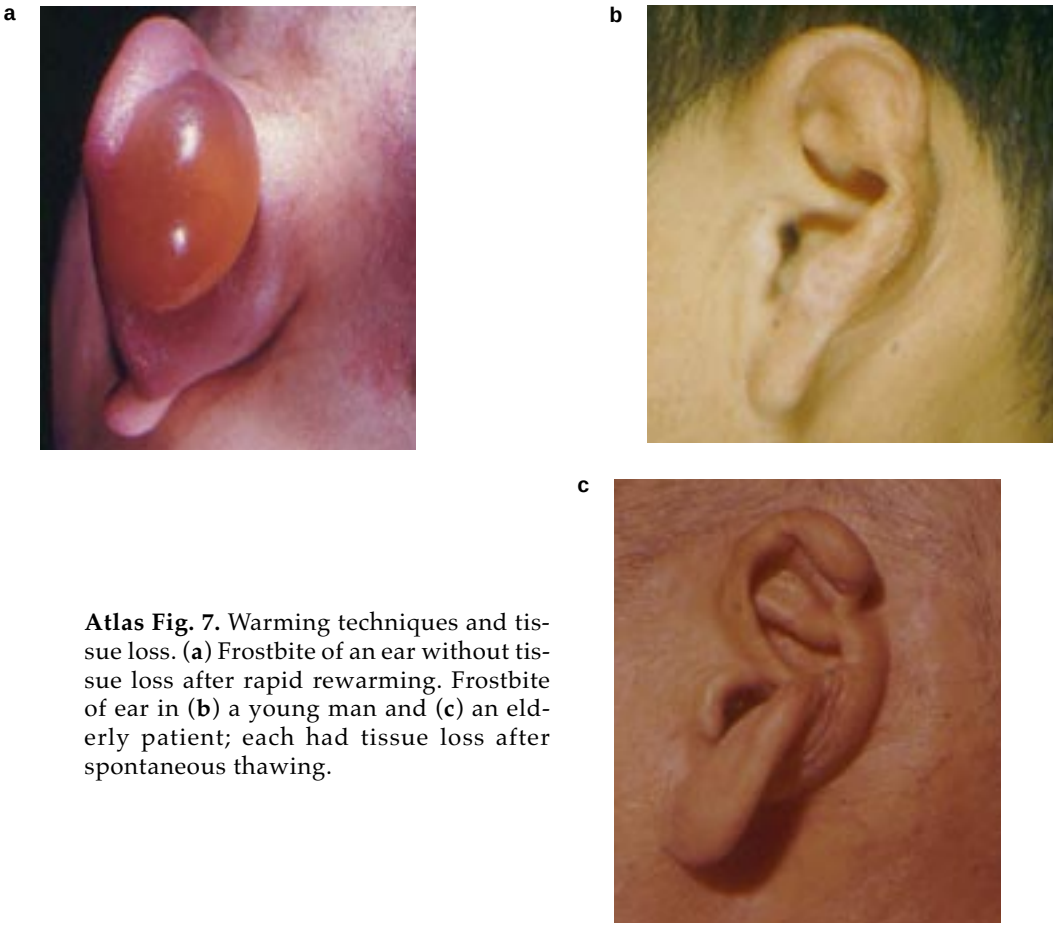
In *decreasing* order of effectiveness, the following methods of thawing are utilized by rescuers, the freezing victims themselves, and even medical personnel:

1. Rapid rewarming in water, 32.2°C to 41.1°C (90°F–106°F). (Use a tub, a whirlpool bath, or a Crane lift platform in a Hubbard tub.)
2. Spontaneous thawing at room temperature, in cabin heat, during foot travel or rescue, or in sleeping bag.
3. Delayed thawing, using ice and snow techniques, cold water, or friction massage.
4. Thawing by excessive heat, such as that from a camp fire, oven, or engine exhaust (> 48.4°C, or > 120°F).

a **b** **c**

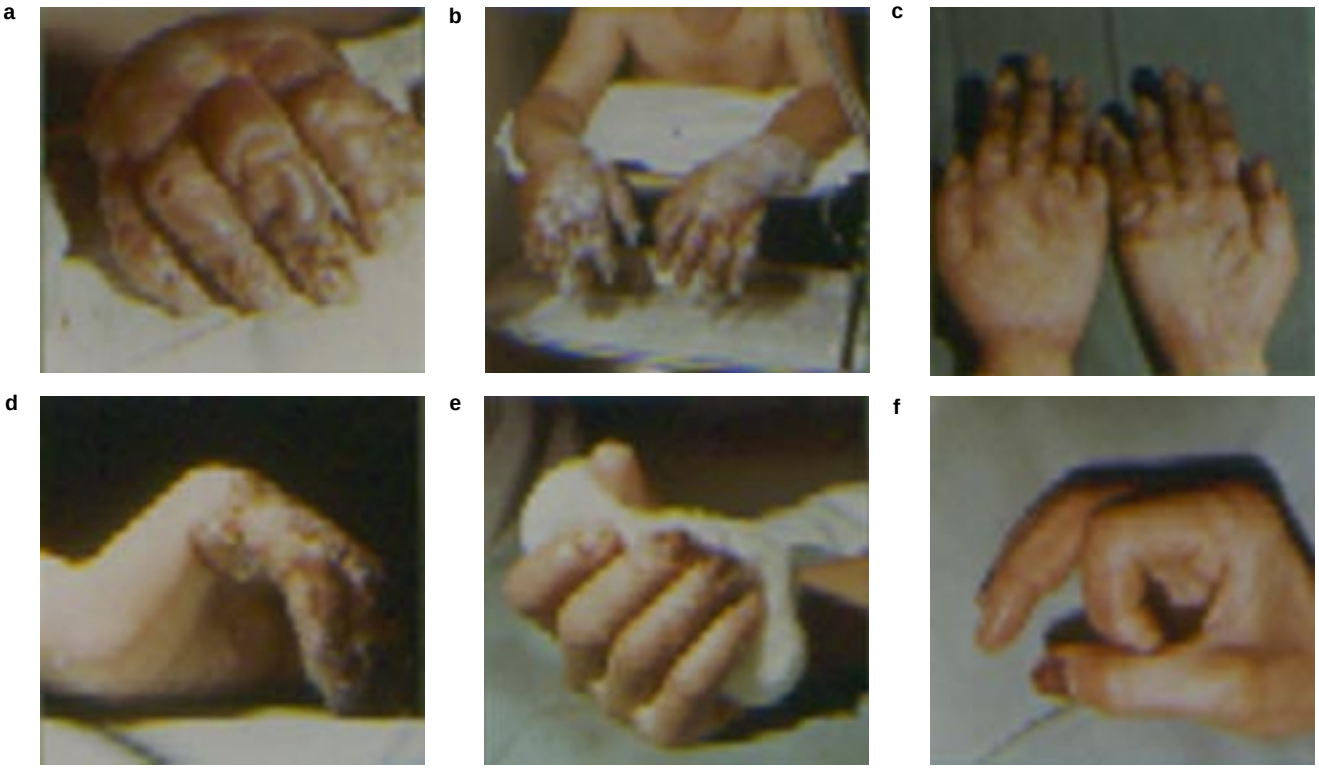


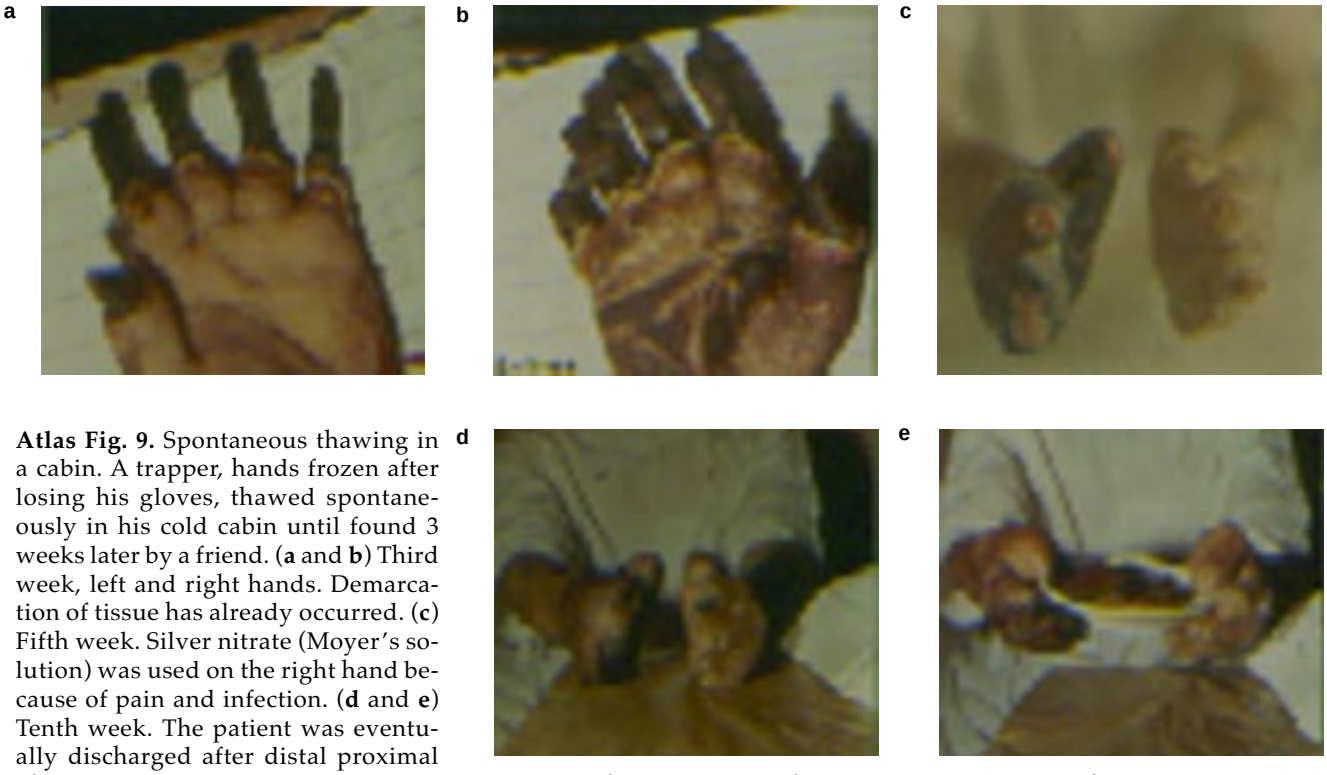
Atlas Exhibit Fig. (**a** and **b**) Before thawing, the clinical appearance of the frozen part is cold, white, or bloodless. The outer shell of skin is rigid, and the depth of freezing is difficult to determine. (**c**) After rapid thawing, the part is flushed red or pink, or has a violaceous hue. Blebs appear 1 to 24 hours after the thaw and rupture spontaneously in 4 to 10 days. The castlike eschar forms after the blebs rupture, and the eschar sheds after 21 to 30 days.



Atlas Fig. 7. Warming techniques and tissue loss. (**a**) Frostbite of an ear without tissue loss after rapid rewarming. Frostbite of ear in (**b**) a young man and (**c**) an elderly patient; each had tissue loss after spontaneous thawing.

Atlas Fig. 8.

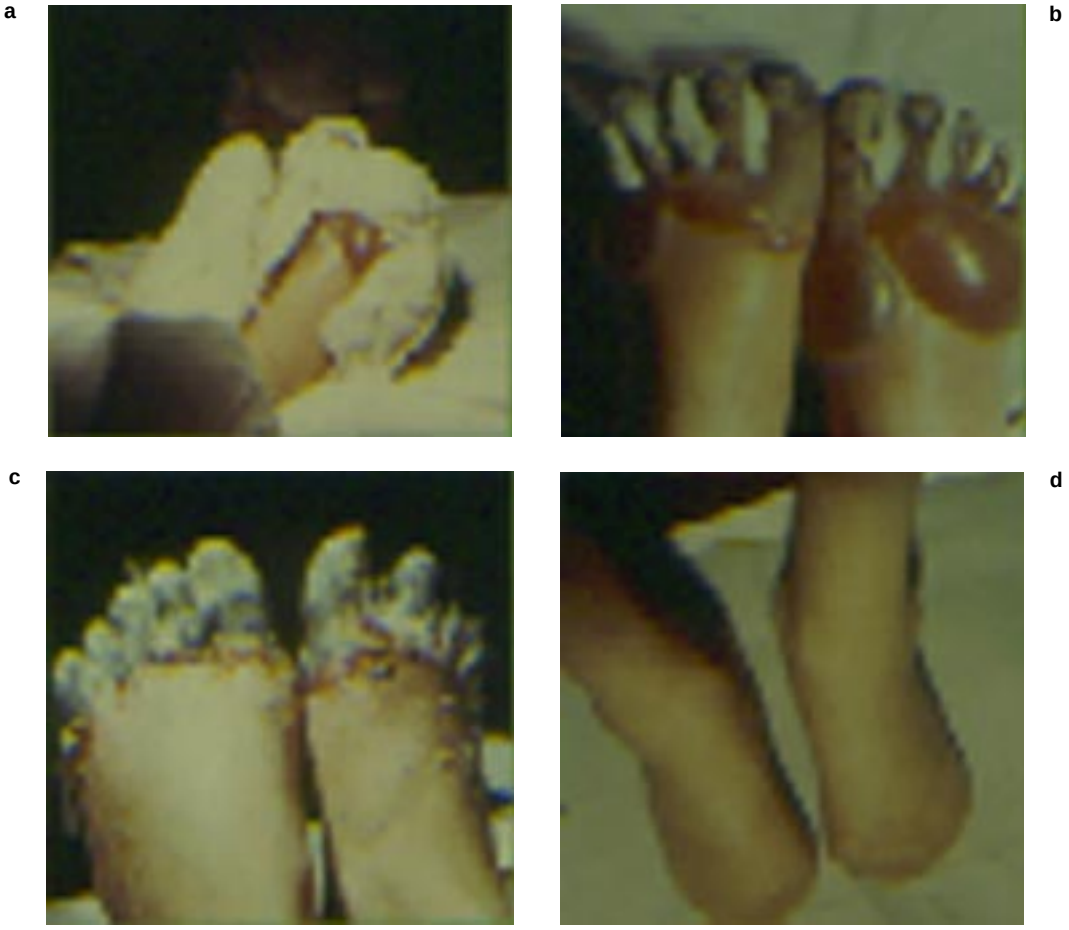




Atlas Fig. 9. Spontaneous thawing in a cabin. A trapper, hands frozen after losing his gloves, thawed spontaneously in his cold cabin until found 3 weeks later by a friend. (**a** and **b**) Third week, left and right hands. Demarcation of tissue has already occurred. (**c**) Fifth week. Silver nitrate (Moyer's solution) was used on the right hand because of pain and infection. (**d** and **e**) Tenth week. The patient was eventually discharged after distal proximal phalangeal amputations but still demonstrates gross hand function. The results of spontaneous thawing vary, due in part to the wide range of temperatures found in cabins, homes, and hospitals.

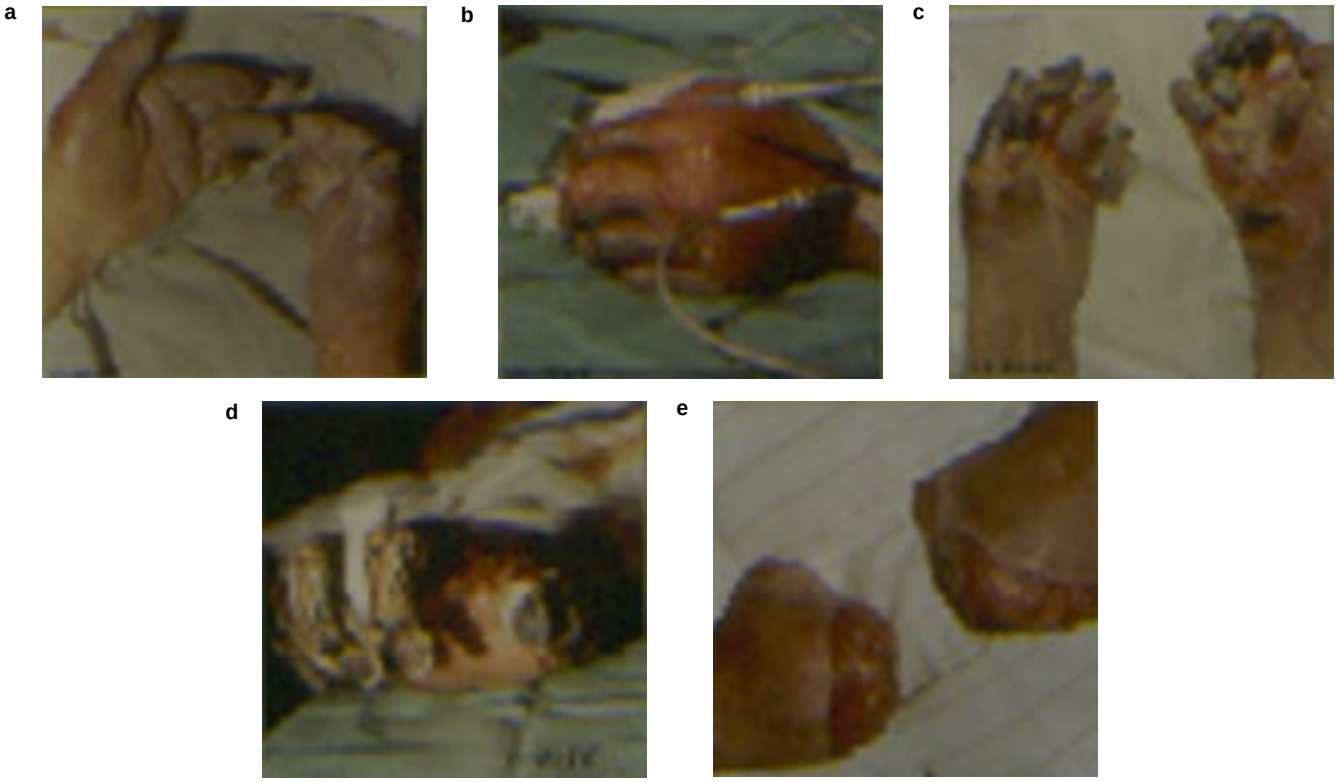
Atlas Fig. 8. Rapid rewarming in a water bath at 42°C (108°F). (**a**) First day. The patient sustained freezing of hands and feet on the Arctic Slope when marooned in the open as a result of a vehicle accident. Winds were 80 knots, ambient temperature between –20°C and –26°C. The patient lost his overboots and gloves in the accident. His entire exposure time, he states, was 15 to 20 minutes, followed by 45 minutes in the wrecked vehicle awaiting rescue. On rescue, he was warmed in water at 42°C (160°F); the warming and care were directed by radio from Anchorage, Alaska. The patient was then transferred from the Arctic Ocean shore to Anchorage by air travel at 24 hours. On arrival, the hands demonstrated large, clear, pink blebs extending to fingertips; these are excellent prognostic signs, especially that the blebs are distal and extend to the nailbeds. NOTE: Only after rapid rewarming in warm water is there return of sensation in the fingertips; this remains until blebs appear in the dermis and epidermis and separate those tissues from the deep structures. (**b**) Fourth day. Constant, twice-daily whirlpool is prescribed with digital exercises, using surgical soaps such as pHisoHex,[*] Hibiclens,[†] or Betadine.[‡] (**c**) Twenty-first day. By the third week, epidermal eschar has formed, preventing joint motion. (**d**) Fourth week. Periodically, when the tissue permits, the eschar is incised to allow joint motion. Escharotomy usually is performed from the 14th to the 31st day. (**e**) Fifth week. Digital exercises are done at frequent intervals at least four times daily, as with whirlpool and biofeedback training. By this time, loss of volar fat pad and loss of nails have occurred and hypesthesia is resolving. (**f**) Seventh week. The anatomical result is good, but volar fat pad loss and intrinsic muscle loss are obvious. The patient has considerable atrophy of the first dorsal interosseus, and also of the abductor digiti quinti.

[*]hexachloraphene detergent cleanser; mfg: Sinofi Winthrop Pharmaceuticals, New York, NY
[†]chlorhexidine gluconate; mfg: Stuart Pharmaceuticals, Wilmington, Del
[‡]povidone-iodine; mfg: Purdue Frederick, Norwalk, Conn



Atlas Fig. 10. Delayed thawing with ice and snow. The patient, a 54-year-old Alaskan Eskimo trapper and hunter, was on a trail in the high arctic when a blizzard struck. His dog team ran off, leaving him stranded without food, water, or shelter, and wearing thin gloves and uninsulated rubber boots. The ambient temperature varied between –50°F and –20°F (–45°C to –29°C). He walked for 6 days, his feet "frozen solid" by the third day. He walked at least 20 miles each day with only snow available for liquid. His feet were frozen for at least 4 days and he left them frozen, without trying to thaw, to maintain adequate walking capability and to survive. On reaching the village of Black River, Alaska, his feet were immersed in snow and ice water and thawed by this delaying method over an 8-hour period. (**a**) On arrival at the hospital in Anchorage, the feet were edematous, cyanotic, and cold. They were wrapped with soft dressing to avoid bleb rupture, protect tissue, and avoid refreezing injury. (**b**) Sixth postthaw day. The feet demonstrate a sign of very poor prognosis. The blebs are dark, moderately hemorrhagic, and proximal to the metatarsophalangeal joints. The toes and distal tissues are without blebs or blistering, and are dusky, edematous, cold; the foot is insensitive at that level. Phalangeal amputation or midfoot amputation is generally unavoidable with this pattern. The final result might be anticipated from the date of admission, as early as 24 hours after the thaw, when blebs are large, hemorrhagic, and proximal, rather than pink, large, and distal.

(**c**) Thirtieth day. The feet, on the frostbite regimen of twice-daily whirlpool baths, demonstrate superficial infection at the junction of the viable and gangrenous tissues. Infection is held in abeyance and controlled by whirlpool baths and aseptic care, permitting the self-demarcation of tissues, so that maximum length of foot is gained. Guillotine amputation is considered from this point on, once the tissue edema has subsided and there is no further tissue retraction. (**d**) Six months. Following revision amputation at the distal metatarsal level at 3 months, the patient went on to a good result and returned to his occupation of trapper and hunter. NOTE: In the very early days of treatment, many patients were hospitalized for long periods. Now, owing to increasing hospital and medical expense, patients are sent home or to a nursing home after the acute stage, where the postthaw regimen (including whirlpool therapy, all drugs required, and digital exercises and biofeedback training) is carried out under supervision.



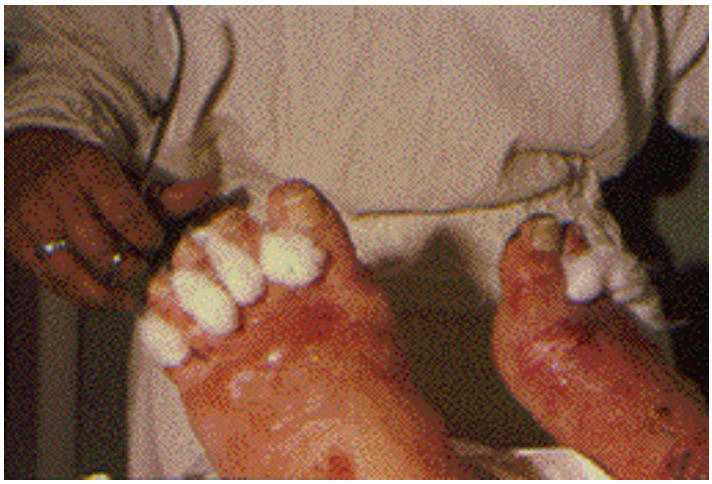
Atlas Fig. 11. Freezing injury thawed with excessive heat (scalding water from a teakettle poured over freezing hands). (**a**) The burn appearance at 24 hours after the thaw. (**b–d**) From 48 hours after the thaw through the 16th day, gradual necrosis of tissue is seen, with eventual increasing mummification. (**e**) Spontaneous digital amputation was present between the fifth and sixth weeks, without surgical interventions. As with the freeze–thaw–refreeze injury, freezing injury thawed by excessive heat usually results in higherincidence of amputation, and at higher levels, on feet and hands.

TREATMENT AFTER THAWING

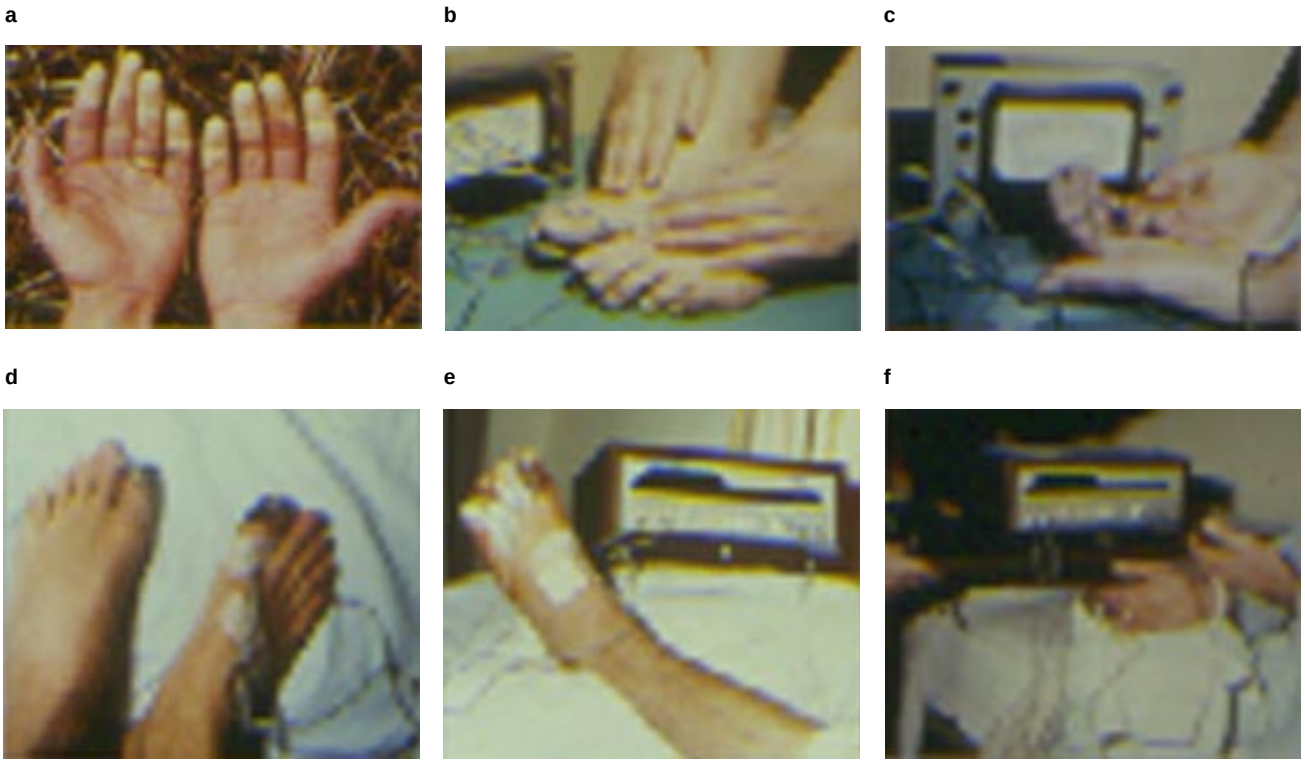
ATLAS EXHIBIT 5

TREATMENT AFTER THAWING

1. Protect the thawed part.
2. Use sterile sheets, masks, and gowns during the bleb stage.
3. Practice "open" treatment.
4. Shun macerating dressings.
5. Administer whirlpool baths with benign soap twice daily.
6. Leave blebs intact.
7. Exercise digits constantly.
8. Avoid early debridement or amputation.
9. Split constricting eschar carefully; whirlpool action will debride gently thereafter.
10. Use antibiotics only with deep or ascending infections.
11. Administer toxoid booster (recommended).
12. Place cotton pledgets between digits to prevent maceration.
13. Avoid narcotics.
14. Consider early skin grafts and late pedicle flaps.
15. Use antibiotics, vasodilators, enzymes, sympathetic blockade, or sympathectomy if needed. Alcohol can be ingested in moderation if requested; smoking should not be permitted.
16. Possibly prevent infection and pain with Moyer's method, using 0.5% $AgNO_3$.
17. Consider fasciotomy if tissue compartment pressure is evident.



Atlas Exhibit Fig. To avoid maceration, cotton pledgets are applied between toes and fingers. If severe swelling or edema occur, pledgets are discontinued to minimize digital vessel pressure. Macerating ointments and salves are avoided if they will not permit the whirlpool to wash off superficial debris and bacteria.



Atlas Fig. 12. Biofeedback, an ancillary therapeutic method for raising digital temperature. (**a**) The hand of a patient with Raynaud's phenomenon, representing the thousands of individuals who demonstrate a labile vasomotor response to cooling, even if that cooling is by a slight drop of ambient temperature (eg, 68°F–65°F). As does Raynaud's phenomenon, frostbite and other cold injuries respond to biofeedback training, or physiological self-regulation. The technique encourages patients who are concerned over the appearance of cyanosis and early tissue loss, giving them a means of contributing to their treatment. Patients practicing the technique can increase the skin temperatures of their digits from the low 70s to the high 80s (degrees Fahrenheit; ~ 21°C–32°C) or even higher; the technique prevents as well as treats frostbite or other cold injury. (**b**) Relaxation techniques and biofeedback training permit cool or cold hands and feet to be warmed by reversing the phenomenon of cold-induced vasoconstriction. (**c**) Selective cntrol of each hand by biofeedback: the left hand is cold and cyanotic, but the right, which is connected to the thermistor, is vasodilated. (**d**) A frostbite patient with necrosis of the large toe is using biofeedback to increase the blood flow in the border area of viability. (**e**) A borderline freezing injury of the foot is treated by biofeedback techniques to avoid tissue loss and increase circulation. (**f**) The technique is particularly effective for hands; in most patients, they appear to be more easily warmed than feet. The technique is helpful, too, in warming digits to avoid injury.

ATLAS EXHIBIT 6

DRUGS USED TO TREAT FROSTBITE

- Plasma volume expanders (eg, low molecular weight dextran)
- Vasodilating agents (tolazaline hydrochloride; eg, Priscoline, Vasodilan)
- Hypotensive agents (guanethidine monosulfate; eg, Reserpine)
- Hemorrheologic agents (pentoxifilene; eg, Trental)
- Calcium blocking agents (eg, Nifedipene)
- Sympatholytic agents (phenoxybenzamine hydrochloride; Dibenzaline)
- Anticoagulating agents (eg, Heparin)
- Thrombolytic enzymes (eg, streptokinase, tissue plasminogen activitor)
- Industrial solvent (dimethyl sulfoxide)
- Nonsteroidal antiinflammatory agents (acetylsalicylic acid [aspirin]; ibuprofen [eg, Motrin])

PROGNOSIS

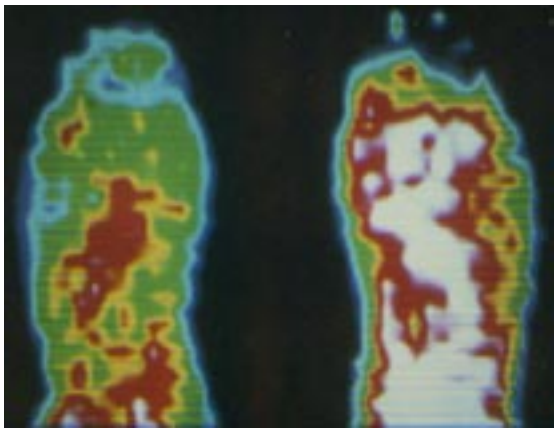
ATLAS EXHIBIT 7

FROSTBITE PROGNOSIS, I

Prognosis is *best* when

1. Freezing is of short duration and depth is superficial.
2. Freezing is not associated with hypothermia, fracture, or other trauma.
3. Thawing is by rapid rewarming.
4. Blebs develop early, pink, and large, and extend to digital tips.
5. Capillary perfusion returns rapidly.

a



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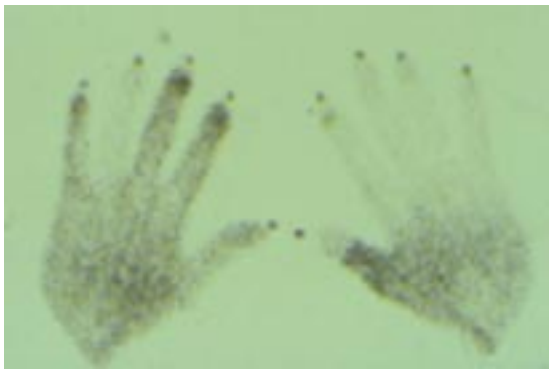

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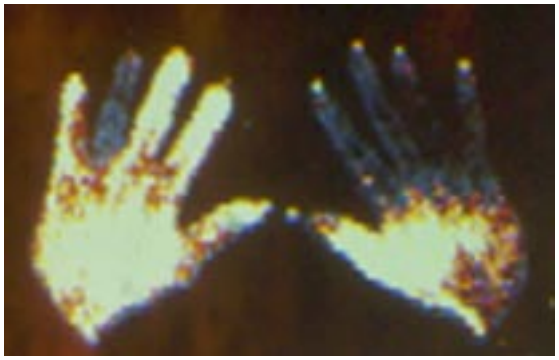
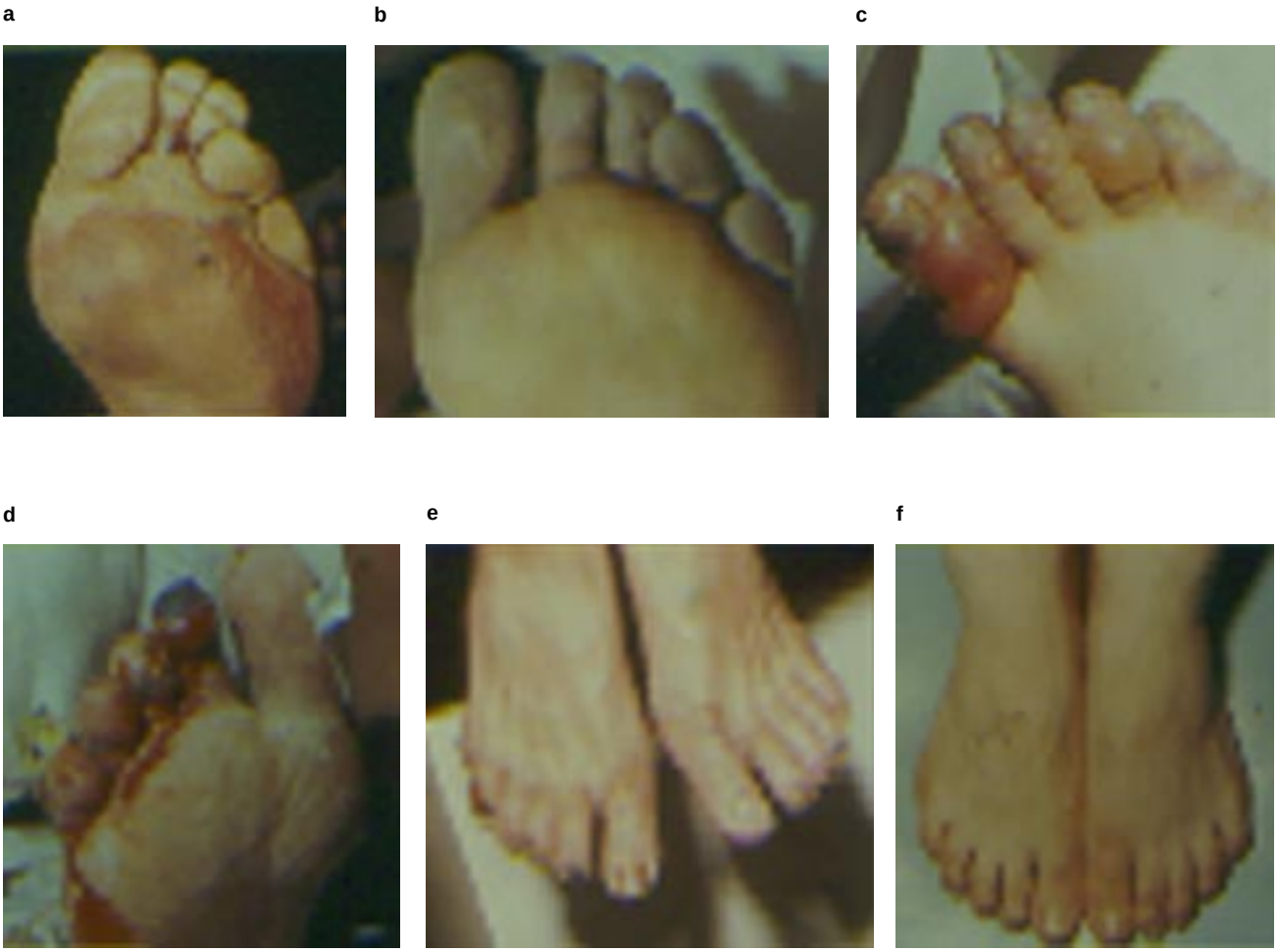
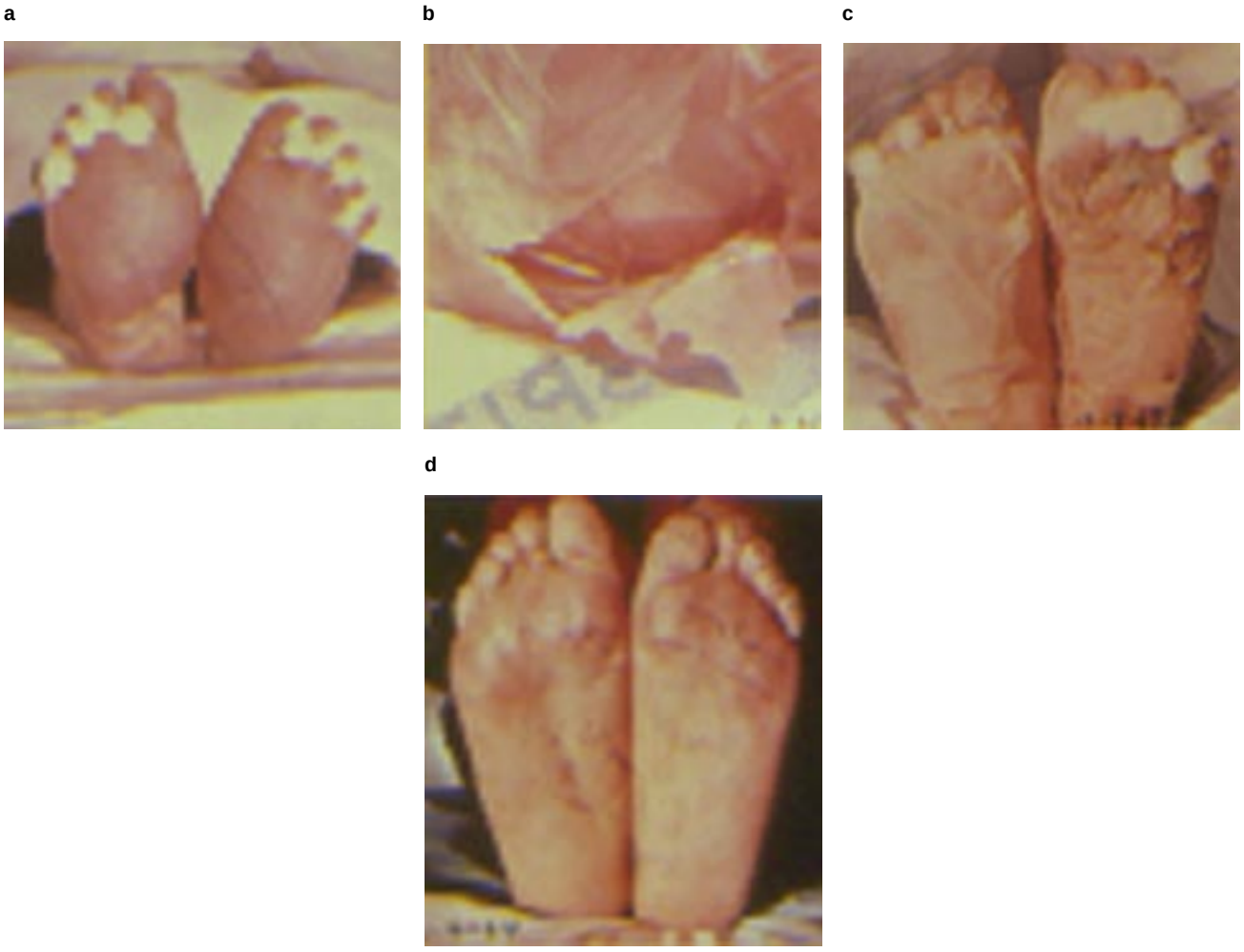


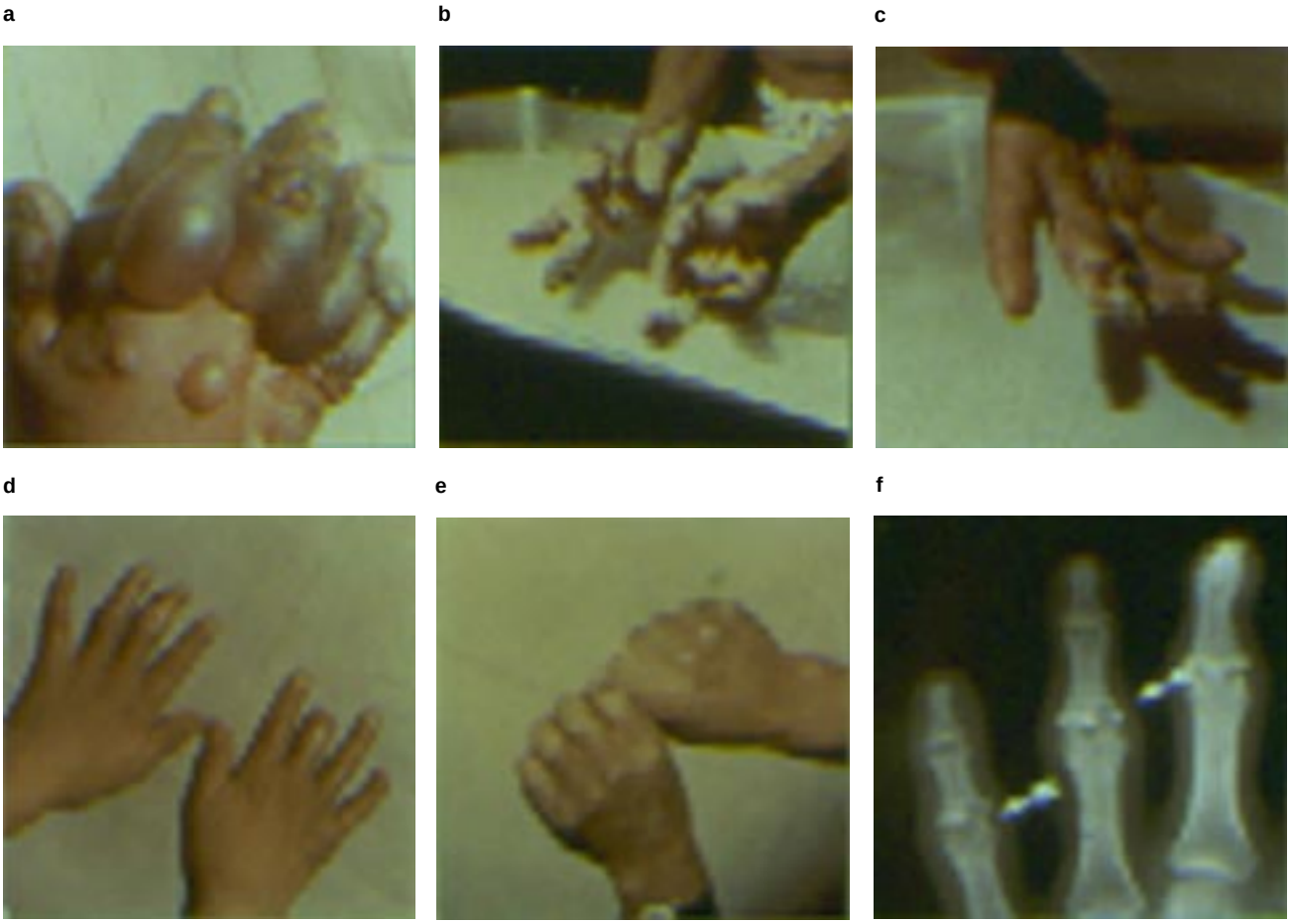
Atlas Fig. 13. For an early prognosis of the cold injury, thermography and isotope studies are helpful. Thermography permits early evaluation of the depth of freezing and adequacy of superficial perfusion, although it does not show the status of the microvascular circulation. Deeper tissues are evaluated with technetium 99m studies. Repeated evaluations with both modalities aid in determining the effectiveness of therapy. (**a**) In thermography, bright colors generally indicate good circulation, while dark colors (green, blue, black) indicate poor blood flow. (**b**) The thermogram demonstrates decreased blood flow to finger 4 of the left hand, and fingers 5, 4, 3, 2, and the tip of the thumb of the right. (**c**) The isotope study of the same hands shows obvious loss of deep circulation in the previously identified fingers. (The patient had worn a ring on finger 4 of his left hand; all metal rings are discouraged, and in fact are removed, during postfreezing care.)



Atlas Fig. 14. Rapid rewarming in a water bath. Freezing occurred in a young hunter, aged 15, at ambient temperatures of –15°F, while he was wearing very tight, borrowed boots. (**a**) A cold, rigid forefoot without sensation or digital motion. Tissue compression and sock marks are obvious. Treatment was whirlpool bath and thawing at 110°F for approximately 20 minutes. (**b**) By 1 hour after thawing, the toes had assumed an an ominous burgundy hue (indicating cyanosis). Cyanosis has since been demonstrated in other cases, more often at temperatures greater than 110°F. The cyanosis remained for approximately 6 hours, at which time small, discrete blebs began to appear. Gross sensation was present after thawing and remained so until bleb development. (**c**) Over the next 48 hours, large, clear blebs developed and ultimately extended to the digital tips. (Failure of distal bleb formation, in the presence of proximal blebs, is an ominous prognostic sign.) (**d**) At 30 days, gangrenous skin of the second toe can be seen. (**e**) By 6 weeks after the injury, the anatomy has been preserved but the changes of deep injury are obvious and include volar fat pad loss, subcutaneous fat loss, early interphalangeal joint contracture, nail changes, hypes-thesias, and hyperhydrosis. Epithelialization is complete. (**f**) By the 4th month, the extremity has adequate sensation; mild subcutaneous loss and interphalangeal joint contracture have occurred, with a few interphalangeal subarticular lesions present on roentgenographic examination. Increased sweating is present.



Atlas Fig. 15. Rapid rewarming with warm, wet towels. The victim's feet were frozen while barefooted in the snow; ambient temperature: 0°F (–18°C); exposure: 30 minutes. The police used warm, wet towels to rapidly rewarm the feet. (**a**) Within 24 hours, huge bullae formed on the plantar surfaces of the feet, giving the appearance of superficial freezing. (**b**) At the patient's request, the bleb on the left sole was ruptured because of severe formication. Fibrous strands formed in the serum 48 hours after freezing, before the bleb was ruptured. Serum content of blebs was similar to that of normal serum exudation, except for decreased protein. (**c**) Pain was much increased on the sole of the patient's left foot after the bleb rupture. No infection resulted with continued whirlpool therapy. At 5 weeks both feet were almost equal in pattern, with early return of sensation: the forefoot demonstrating hypesthesia; the arch and heel, hyperesthesia. (**d**) At 2.5 months, edema was still present on dependency or ambulation. The interphalangeal joints had less than 50% range of motion, with volar pad loss at the toes and incomplete desquamation.



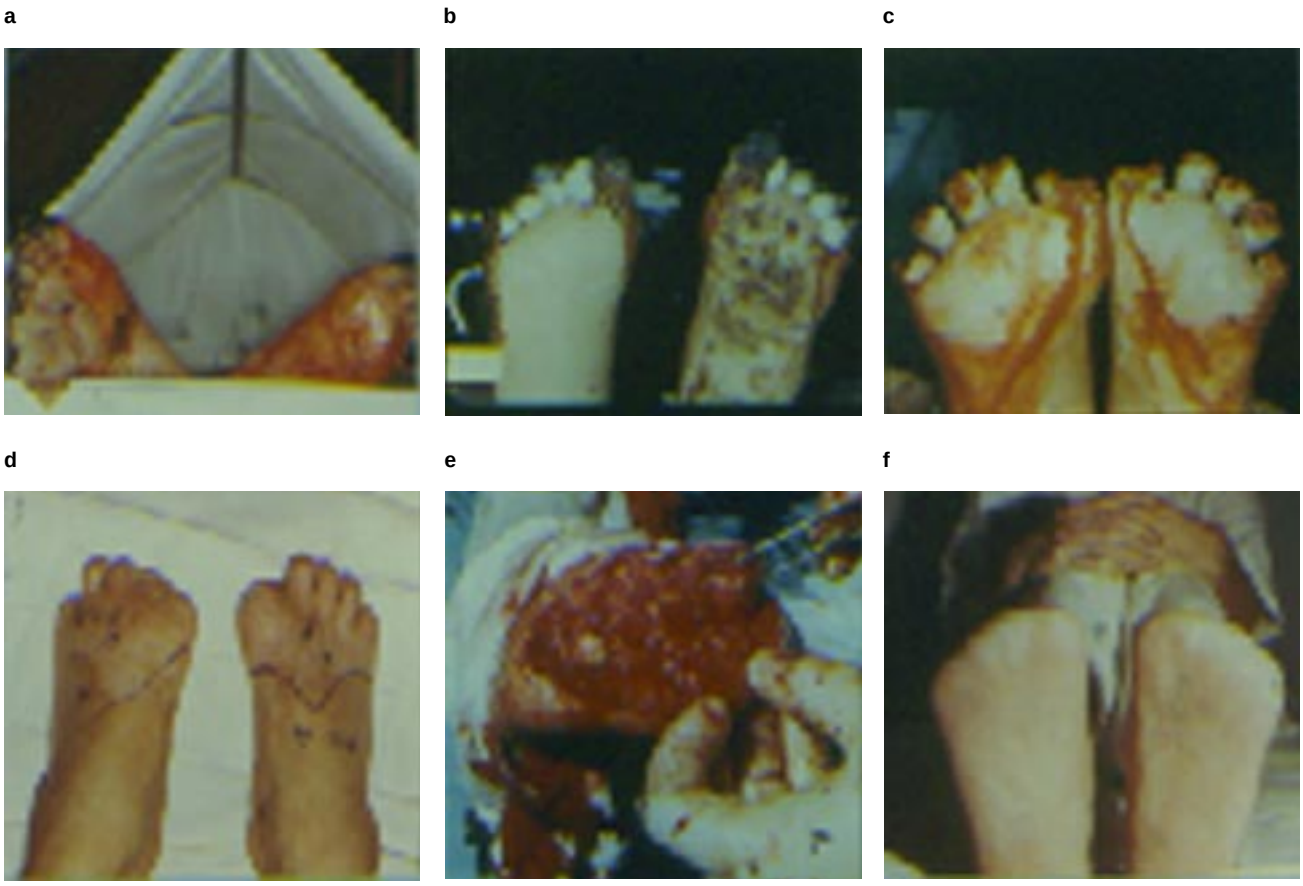
Atlas Fig. 16. Combined delayed warming (with ice and snow) and rapid warming (in water). A 23-year-old dog-team driver, who lost his dogs on the trail, froze both hands; ambient temperature: –14°C; wind: 10 mph; exposure time: 1–2 hours. Delayed warming with snow and ice water was followed later by rapid warming in warm water. (**a**) By the fourth day after the injury, the clear, pink, large, distal blebs on the digits are notable, except for the mid finger of the right hand, the site of a previous digital neurovascular injury: a knife wound. (**b**) Twice daily whirlpool baths with pHisoHex soap and digital exercises are performed. By the third week, the gangrenous tip of the mid finger, right hand, and site of the old injury, are noted. (**c**) By the sixth week the necrotic tip of the mid finger of the right hand is seen, with volar fat pad loss to the fingertips throughout. (**d** and **e**) By the 12th week, partial finger amputation of the mid digit, right hand can be seen, along with scarified skin over the dorsum of the right and left hands and contracture of the proximal interphalangeal joints of digits 5, 4, and 3 on the left hand and 5, 4, 3, and 2 on the right. (**f**) A followup roentgenogram 3 years later demonstrates periarticular, lytic destructive changes of cartilage and bone at the proximal interphalangeal joints, fingers 5 and 4 on the right, and similar changes on the left hand. The invasion of fibrous tissue through the joint surfaces results in marked limitation of joint motion.

ATLAS EXHIBIT 8

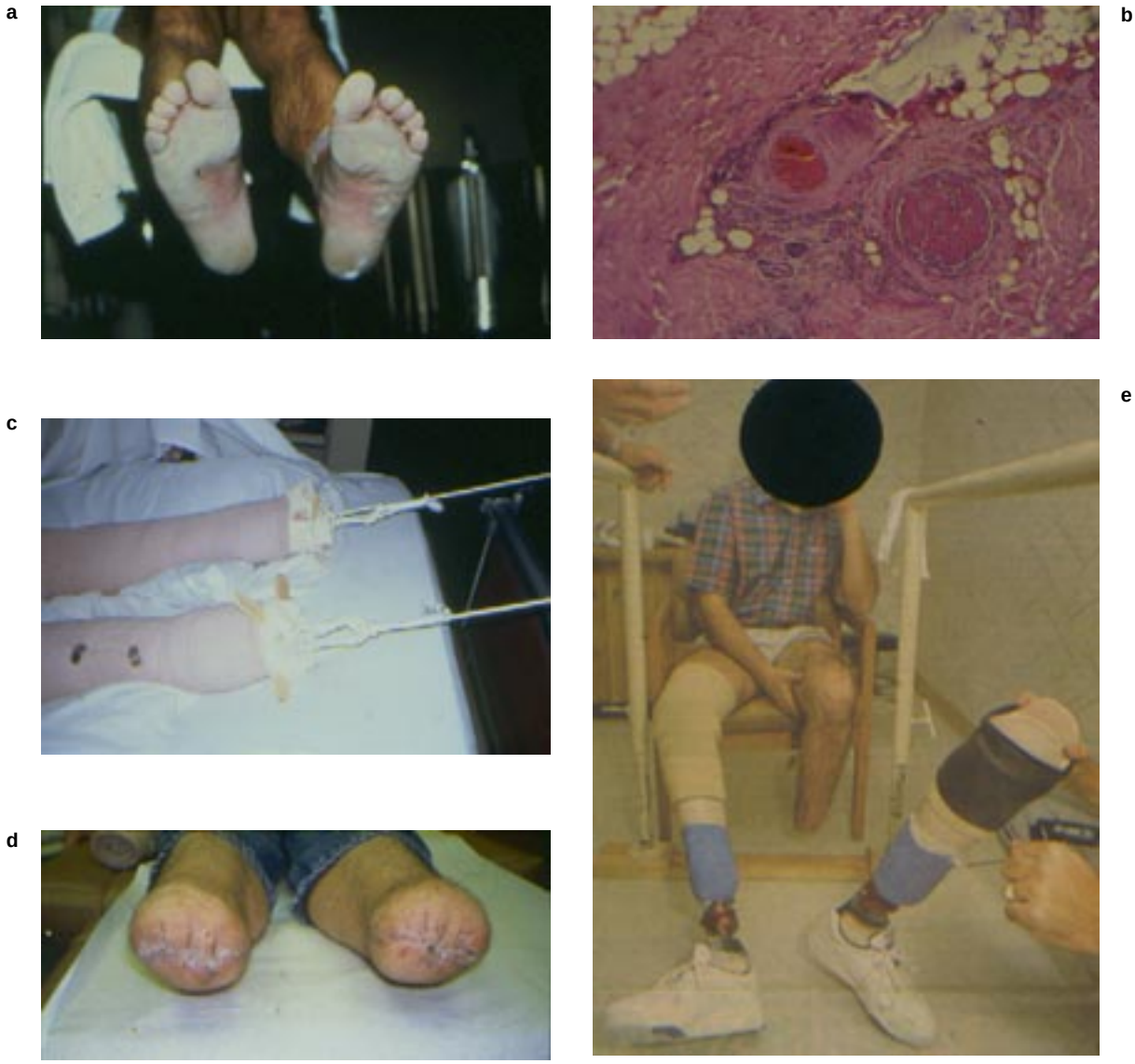
FROSTBITE PROGNOSIS, II

Prognosis is *uncertain* when

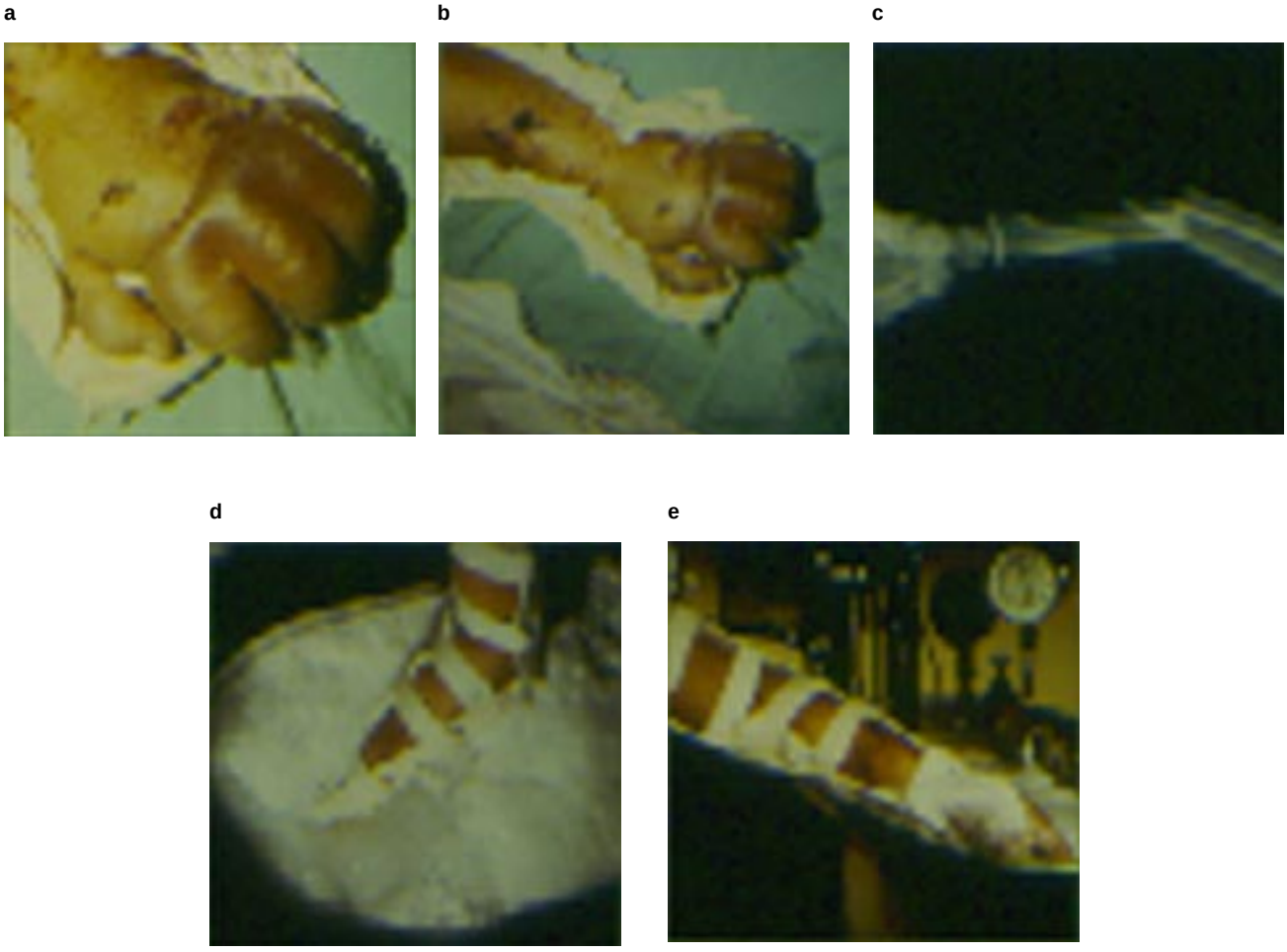
1. Thawing is spontaneous.
2. The frozen state is of long duration.
3. Freezing is superimposed on a fracture or dislocation or is associated with soft-tissue trauma.



Atlas Fig. 17. Spontaneous thawing, in jail. After a drinking episode in rural Alaska, the victim suffered deep frostbite to both feet. (**a**) By 1 week after the injury, the prisoner, now with gangrenous toe tips, was transferred to a hospital. (**b**) The irrational prisoner escaped the hospital bed and ran barefoot in snow until captured. A diagnosis of postalcoholic encephalitis was made. (**c**) Necrotic tissues were removed; the operative incision, through which a penrose drain had been inserted, was approximated using one suture. Whirlpool therapy was continued. (**d**) The patient experienced pain, swelling, hyperhidrosis, hypesthesia, and anesthesia in the residual digits. (**e**) An avascular first metatarsal head was revealed at a revision operative procedure performed 1.5 years after the injury, after which the patient received (**f**) an adequate weight-bearing transmetatarsal amputation.

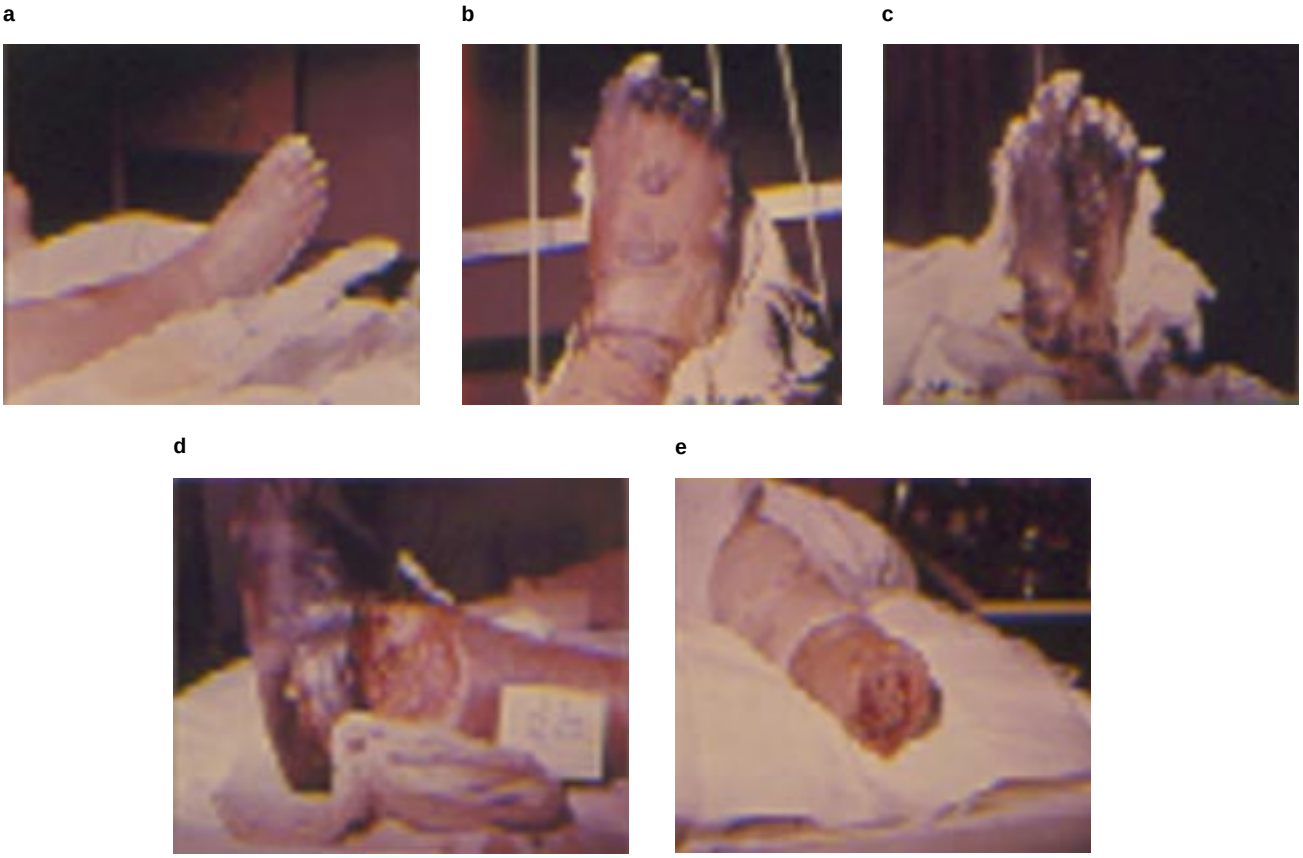


Atlas Fig. 18. Exposed for 5 days at 19,000 ft on Mount McKinley before helicopter rescue, this climber from Poland, secluded in a small snow cave without food or water for the last 3 days before rescue, sustained deep freezing injury of both feet to the ankle level. The injury was not one of freeze–thaw–refreeze but instead of severe, deep, long-duration freezing at temperatures far below 0°F, and with 25- to 50-knot winds for most of the first 4 days. Rescue was on the fifth day. (**a**) The climber is in the emergency department prior to rewarming. The impressive pallor of the climber's feet and ankles and the history are indicitive of freezing injury. Technetium scans demonstrated no capillary perfusion below the ankle level bilaterally. (**b**) The pathology slide, after transmalleolar amputation of the feet and ankles, demonstrates intraluminal clotting in a small artery (center) and vein (right of center); the clotting occurred throughout the foot. (**c**) After guillotine amputation, the climber's long tibial-fibular stumps are contained in skin traction with light weights to permit gradual closure and granulation. (**d**) The stumps, after revision amputation and closure. When severe gangrenous change is present, with always at least superficial infection, guillotine procedure followed by delayed closure or skin graft is chosen. (**e**) With long tibial stumps and a supracondylar patellar tendon bearing prosthesis, the patient has since recovered and has been climbing mountains again.



Atlas Fig. 19. Open fracture followed by freezing cold injury, thawed by rapid rewarming. A trapper was injured, sustained an open fracture of the distal radius and ulna on the left arm. He sustained freezing of the hand and wrist while seeking help. The warming at rescue was rapid rewarming in warm water; the forearm was splinted to protect the fracture. (**a**) Twenty-four hours after the warming, the blebs are large, light colored, and clear, and they extend to the fingertips, an excellent prognostic sign. (**b**) The open area above the fracture was debrided after thawing with thorough irrigation of the wound, fracture repositioning, and the antibiotic coverage (streptomycin and penicillin) that was in vogue at the time (the 1960s) was used. The arm was splinted only, and digital exercise immediately done. The wound was packed open and allowed to granulate closed. (**c**) The displaced (compound) fracture, with displacement and skin penetration of the fragments of radius and ulna demonstrated on roentgenogram. (**d**) The forearm and upper arm were contained in a plastic open splint, allowing for whirlpool therapy, motion of the joints, and observation unhindered by occlusive dressings. Whirlpool with soap (pHisoHex,[*] Hibiclens,[†] Betadine[‡]) was used twice daily for constant debridement. (**e**) Tissue healing was eventually sufficient to permit surgical fixation of the bone fragments. The patient sustained no anatomical loss. Fractures, followed by freezing, appear to do best if rapid rewarming is utilized as the thawing method. This can be supplemented if necessary by fasciotomy, stabilization of the fracture at least by splinting, and immediate reduction of fracture dislocations.

[*]hexachloraphene detergent cleanser; mfg: Sinofi Winthrop Pharmaceuticals, New York, NY
[†]chlorhexidine gluconate; mfg: Stuart Pharmaceuticals, Wilmington, Del
[‡]povidone-iodine; mfg: Purdue Frederick, Norwalk, Conn



Atlas Fig. 20. Fracture or dislocation with superimposed freezing and spontaneous thawing. Two caribou hunters' aircraft struck a mountain ridge. One hunter sustained a fracture of the right mid tibia (and of the dorsal spine, not shown here). (**a**) The extremity is cyanotic, demonstrating vascular insufficiency. Pedal pulses are absent, as is sensation to the level of the malleoli. No blebs are present at this stage. This is the typical picture of extremity fracture or dislocation when thawed by other than rapid rewarming. (**b**) By the 14th day, the minimal blebs formed were dark and serosanguinous. The foot was without sensation and the toes obviously gangrenous. (**c**) By 3 full weeks after the thaw, dry gangrenous change had encompassed most of the foot, including the plantar pad. Tissue necrosis continued; the extremity was amputated at the level of the fracture.

The second hunter, who sustained (**d**) a fracture dislocation of the left ankle, crawled 2.5 miles down a mountain, dragging his companion (views a–c) with him. Both legs of the second hunter froze, then thawed spontaneously in a tent. (**e**) His left ankle was amputated. The frozen right leg, not shown and not fractured, recovered.

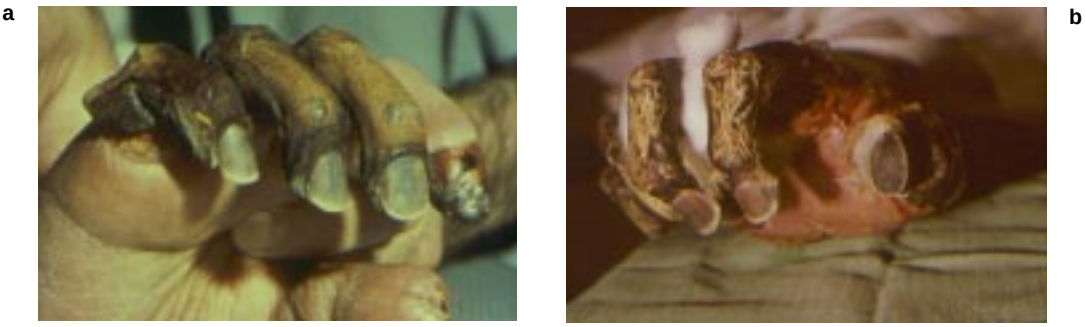
Because of swelling, edema, vascular compression, or compartment syndrome, good results are generally obtained only after rapid rewarming and with, most often, fasciotomy.

ATLAS EXHIBIT 9

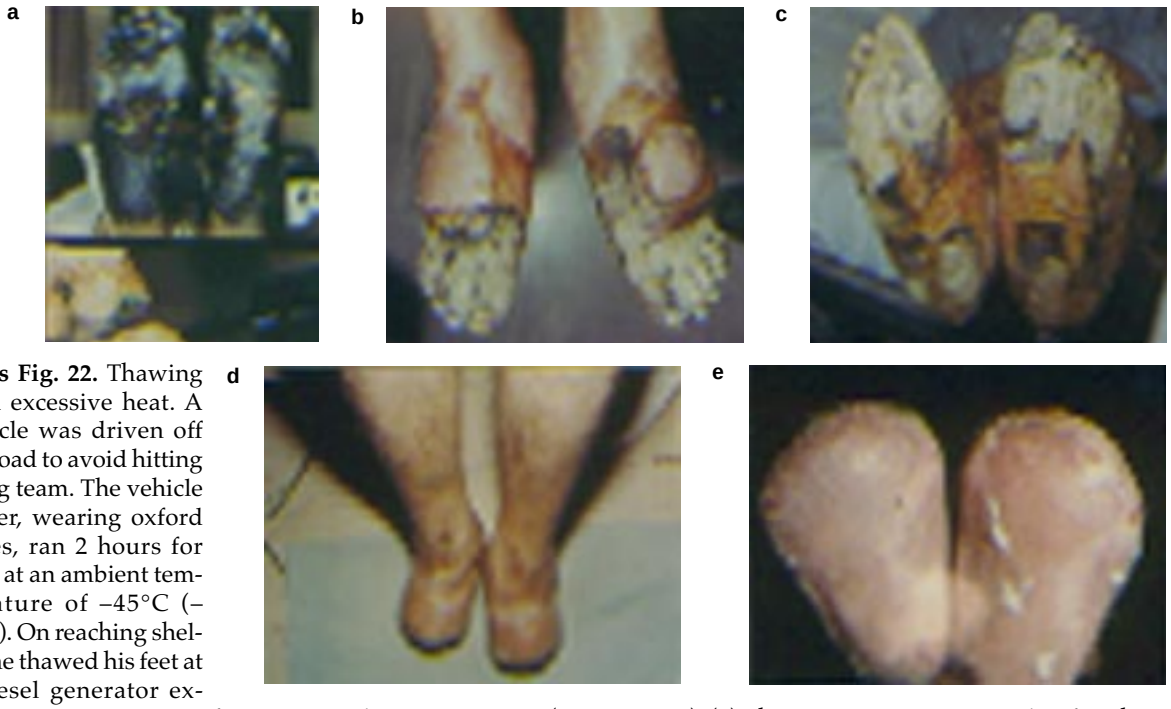
FROSTBITE PROGNOSIS, III

Prognosis is *poor* when

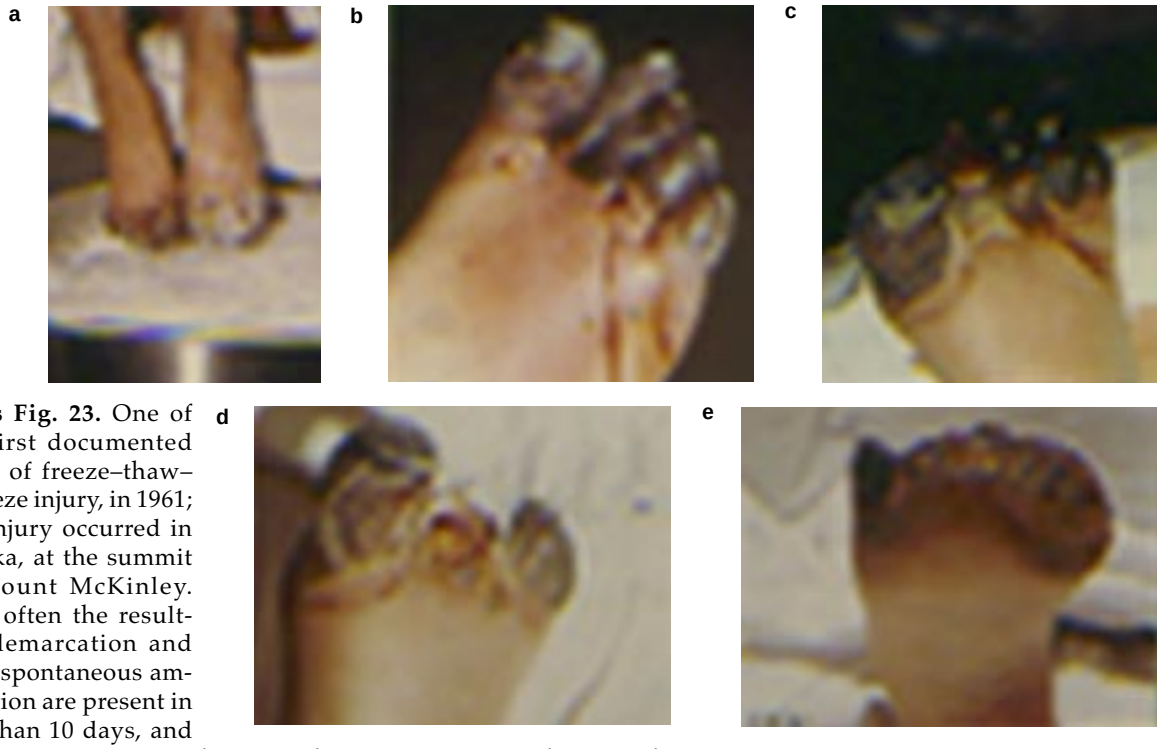
1. Thawing is delayed (eg, the part is packed in ice and snow).
2. Thawing is by excessive heat.
3. Thawing (by any method) is followed by refreezing.
4. Freezing is superimposed on long-standing immersion (wet–cold) injury.
5. Freezing is associated with fracture or dislocation with unrelieved, increasing tissue compartment syndrome and vascular occlusion.



Atlas Fig. 21. Freezing injury followed by thawing at excessive heat. (**a**) These frozen fingers were thawed in a hot oven, which results in rapid mummification and dry, gangrenous tissues with early demarcation, often by the fifth day. (**b**) To emphasize the danger of thawing with excessive heat (> 116°F), this hand, thawed in water simmering in a teakettle, demonstrates at 3 weeks absolute mummification, with digits hard and total tissue death. Because of whirlpool treatment with pHisoHex or Hibiclens, however, the level of infection is superficial at the area of tissue demarcation, preceding eventual spontaneous amputation.

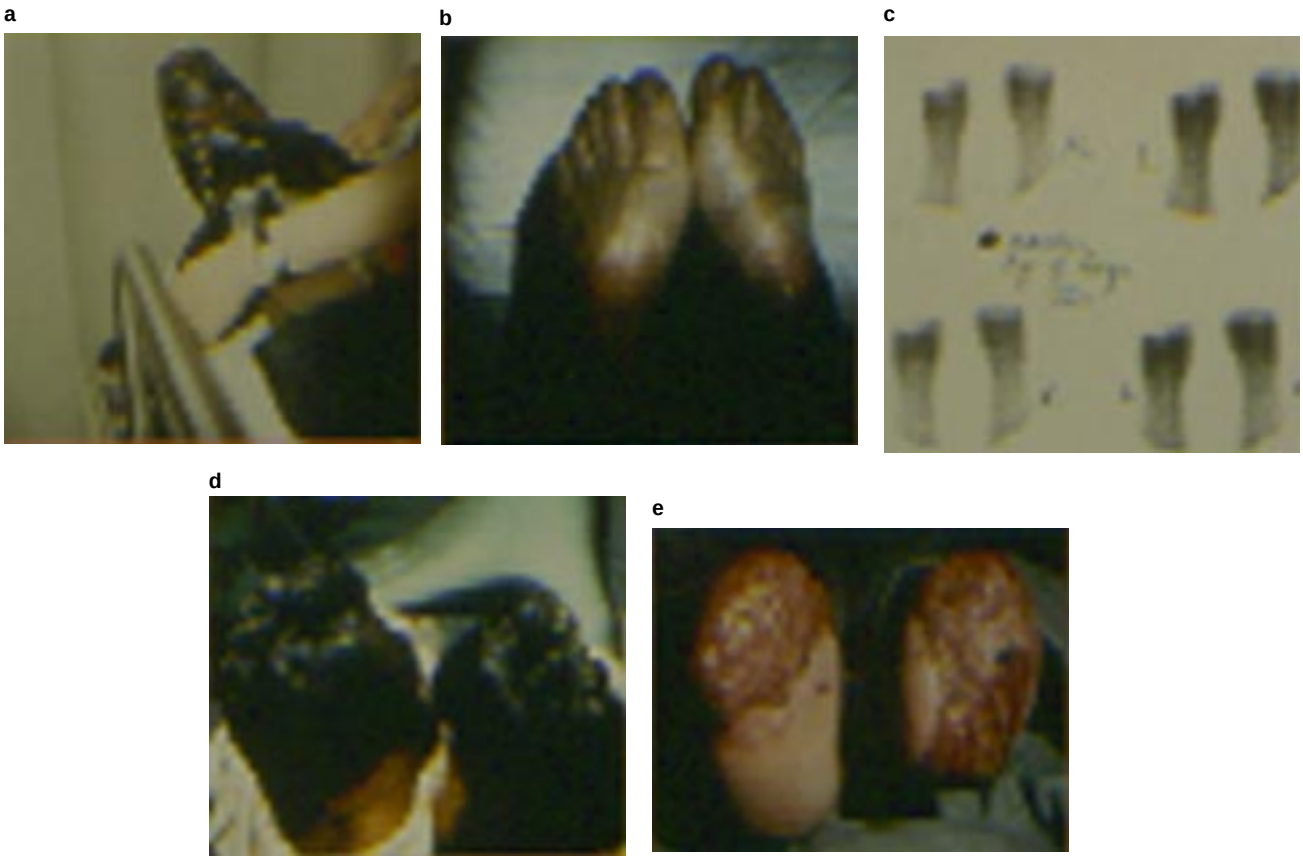


Atlas Fig. 22. Thawing with excessive heat. A vehicle was driven off the road to avoid hitting a dog team. The vehicle driver, wearing oxford shoes, ran 2 hours for help at an ambient temperature of –45°C (–50°F). On reaching shelter, he thawed his feet at a diesel generator exhaust at a temperature of approximately 79.4°C to 85°C (175°F–180°F). (**a**) The victim was seen 3 weeks after the injury, at which time mummification of toes and epidermal gangrenous plaques were present on soles, and multiple dorsal escharotomies were performed on the lower right foot. (**b**) The appearance of the feet 3 months after the injury (and about 2.5 months after the dorsal escharotomies). (**c**) On the same day, epithelialization could be seen under the black eschar of sole and heel. (**d** and **e**) The patient was permitted to return to work and normal activity 10 months after the injury.



Atlas Fig. 23. One of the first documented cases of freeze–thaw–refreeze injury, in 1961; the injury occurred in Alaska, at the summit of Mount McKinley. Very often the resultant demarcation and even spontaneous amputation are present in less than 10 days, and the part is often lost at the site of the second freeze. In this case, the soft tissue separated after 7 to 10 days, assisted by the surgeon. Frames **a** through **d** were made from 25 May through 30 May. Frame **e**, showing the same foot but viewed from the sole, was made 7 days later, on 6 June. From this injury came the dictum, "Do not rewarm in the rescue area if there is danger of refreeze."

Atlas Fig. 24. Bilateral freeze–thaw–refreeze injury of the hands and feet with severe dehydration and caloric depletion. Altitude: 19,000 ft (5,800 m); exposure: 4 or 5 days; ambient temperature: –35°C; winds: severe.



(**a**) After helicopter evacuation from Mount McKinley at 4,267 m, the patient's left foot is seen still in the climbing boot with its zippered neoprene liner; ice was found throughout the boot. (**b**) The feet, totally frozen to the malleolar level, demonstrate the zipper indentation of the neoprene sock. This phenomenon is not uncommon on high-mountain freezing injury. Unlike mountains near the equator, the barometric pressure at 4,572 to 4,877 m approaches 0.5 atm. As a consequence, cellular material in the boot or sock at this altitude expands; if the outer boot (made of leather or plastic) is rigid, then the pressure is directed inward to cause compression of the foot and occlusion of the underlying vessels. This contributes to loss of circulation, compartment swelling, pressure increase, and freezing injury. (**c**) Isotope studies done almost 2 weeks after the injury demonstrate loss of capillary perfusion to the level of heel pads and the mid tarsi. (**d**) At 2 months after the injury, infection is minimal because of twice-daily whirlpool therapy. The tissue is dry and mummified, with complete demarcation. (**e**) At 3 months, the guillotine amputation at the tarsal level, followed by split-thickness skin cover, has readied the feet for revision amputation and pedicle flap cover as necessary.

Atlas Fig. 24. *continued*

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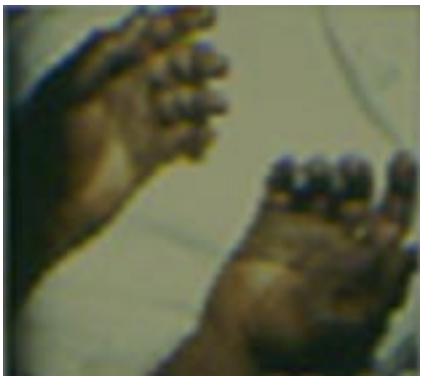

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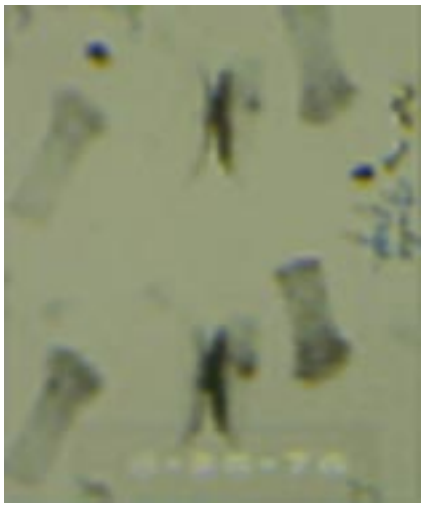

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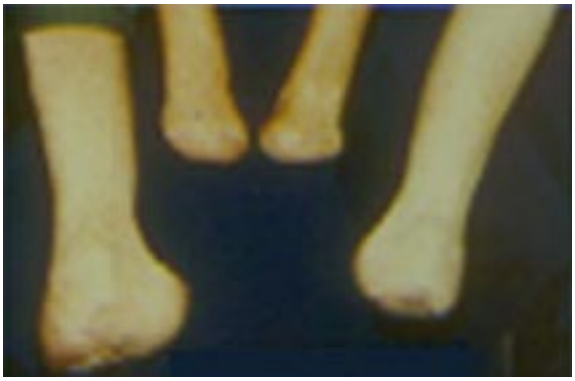
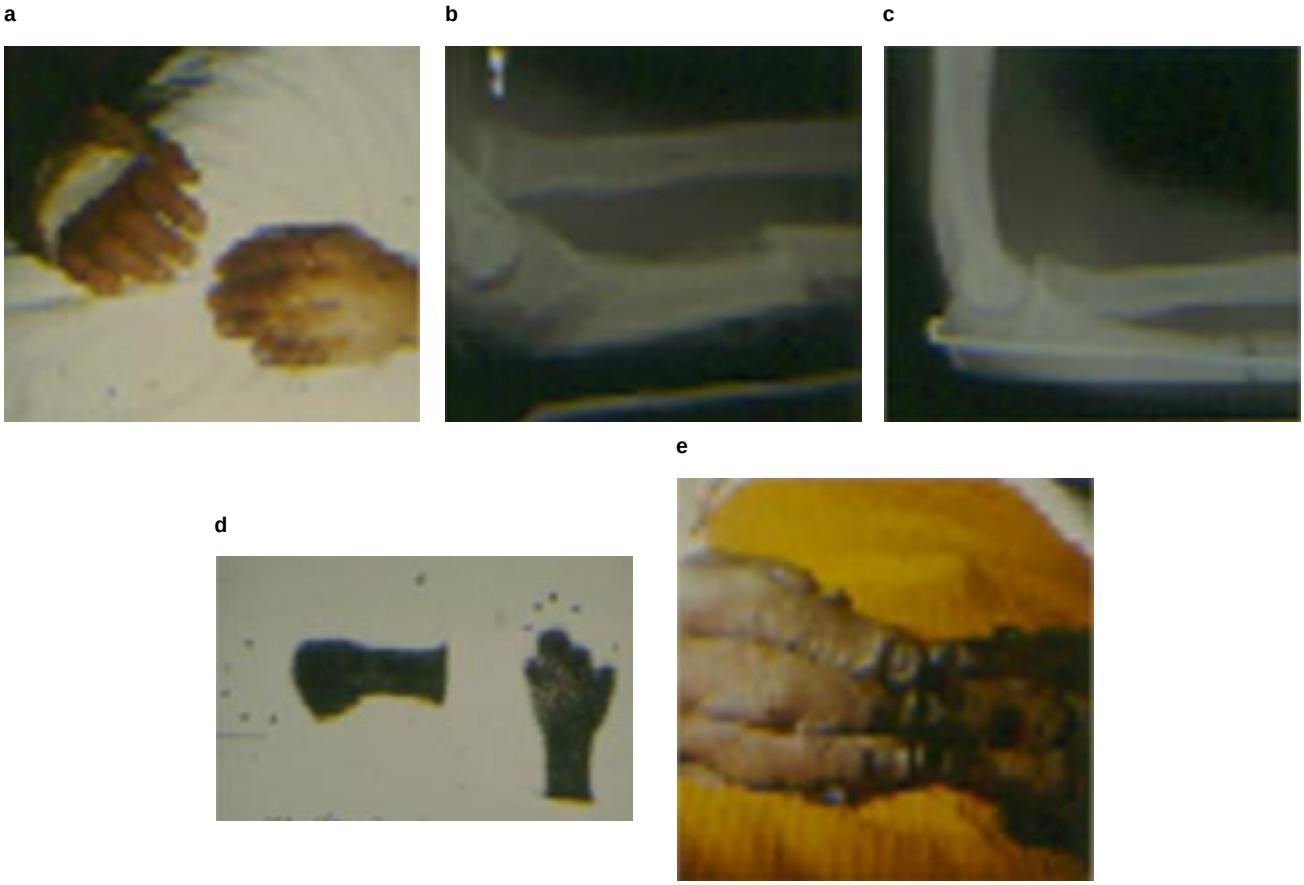
(**f**) Both hands, still frozen on the fifth day of exposure (a freeze–thaw–refreeze injury). An adequate history was limited because of severe disorientation of this patient and his companion. (**g**) Several hours after the thaw, the hand demonstrates no distal blebs, early mummification, flattening of the fingertips, and digital cyanosis—poor prognostic signs. (**h**) By 24 hours after the thaw, gangrene is developing in the hand and mummification of the digits is almost complete. This severe, early change is pathognomonic for a freeze–thaw–refreeze injury or thawing with excessive heat, especially the former. Fasciotomy revealed no viable tissues in digits or distal palm. (**i**) The isotope scan demonstrates total loss of capillary perfusion, just distal to the metacarpophalangeal junctions. (**j**) Guillotine amputation assisting the spontaneous demarcation was performed to the level of viability.

k



(**k**) The final result is a true disaster but one permitting function with prosthetic use. On the right foot, amputation left only a portion of the cuboid and cuneiform bones. On the left foot, only the calcaneus, talus, and navicular bones remain. On the left hand, amputation is at the mid-metacarpal level for the lateral four digits and at the metacarpophalangeal joint of the thumb. On the right hand, amputation is at the distal metacarpals for four lateral digits and at the metacarpophalangeal joint of the thumb.

Atlas Fig. 25. Multiple trauma with spontaneous thawing and refreezing. In the high arctic Bering Sea Island, the victim's helicopter crashed into a ridge. The helicopter had been buffeted by 50-mph winds; the ambient temperature was –8°F (–22°C). The patient was the lone survivor, his companions dying of injury and exposure. He was without gloves at crash time but had adequate boots. His right arm was fractured. On regaining consciousness, he found that his hands were frozen. He was able to crawl but discovered that he had multiple injuries that restricted motion. Numerous times over the next 36 hours he crawled to a high ridge to signal for help with a locator transponder. During this time he sustained multiple freeze injuries to his left hand and attempted to protect his right hand from further freezing. He was rescued by snowmachine, then flown to Anchorage, Alaska. Spontaneous thawing occurred in transit. On arrival at the hospital, his examination revealed him to have a closed head injury, compression fractures of two thoracic vertebrae, and soft-tissue injuries of the neck and possible fracture compression of several cervical bodies, in addition to bilateral freezing injury to the hands and Monteggia fracture dislocation of the right elbow.



(**a**) The initial admission photograph demonstrates, despite thawing of the hand, the presence of very small blebs primarily proximal in position. The tips of the fingers were very cyanotic. Both hands, although insensitive, seemed to have adequate warmth. (**b**) On the day of admission, the roengtenographic examination of the right elbow revealed a dislocation of the radial head and fracture of the proximal ulna (a Monteggia fracture). (**c**) The day after admission, the fracture of the ulna was openly reduced and gently fixed with a Rush intramedullary nail. The radial head spontaneously reduced itself when the ulna was fixed. The procedure was done to facilitate motion, care, and physiotherapy, and to reduce swelling that might limit vascular supply. (**d**) Before surgery, a technetium 99m scan of both hands demonstrated no perfusion of the fingers beyond the metacarpophalangeal junction of the right hand; the left hand has capillary perfusion of the proximal and mid phalanx of the 5th finger and proximal phalanx of the 4th and 3rd fingers, and only a small portion of proximal phalanx of the thumb and index finger. (**e**) A week after the accident the left hand demonstrates advanced mummification of the distal digits, which are dry with edema, and early liquefaction necrosis of the digits of the right hand. The pattern of the left hand is that of a typical freeze–thaw–refreeze injury. The pattern of the right may represent the effect of unreduced fracture dislocation over 36 hours and the uncertain result of spontaneous thawing, particularly with fractures or dislocations.

Atlas Fig. 25. *continued*

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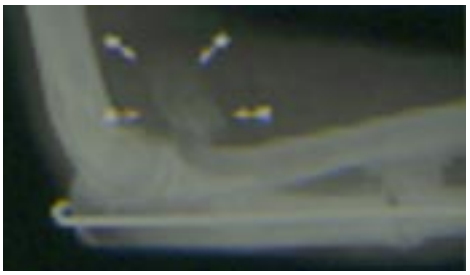

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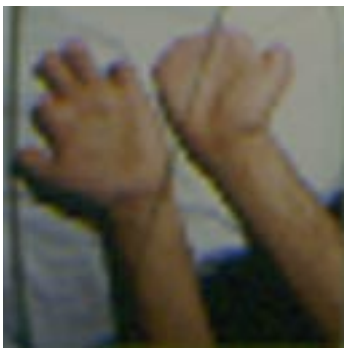
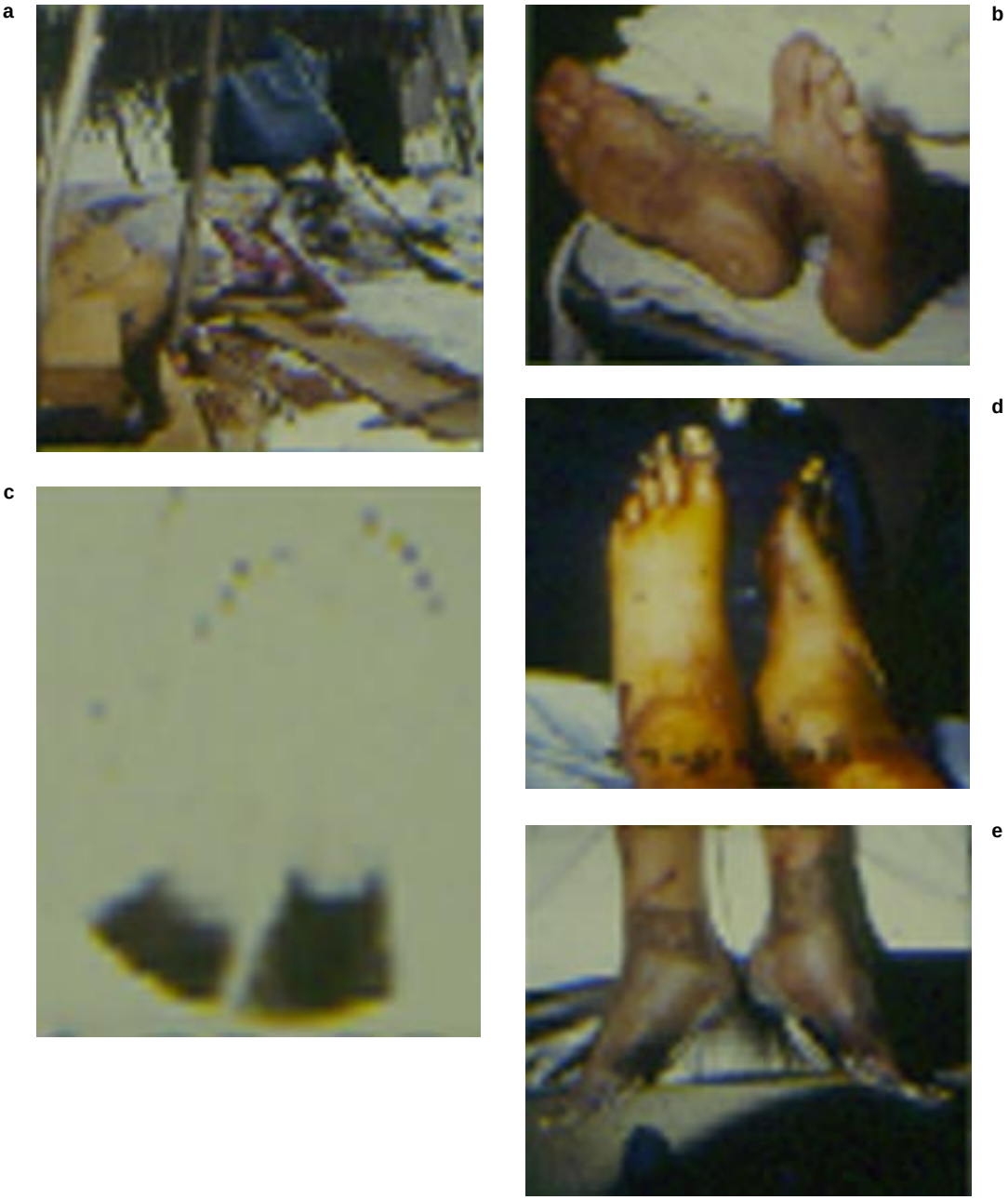


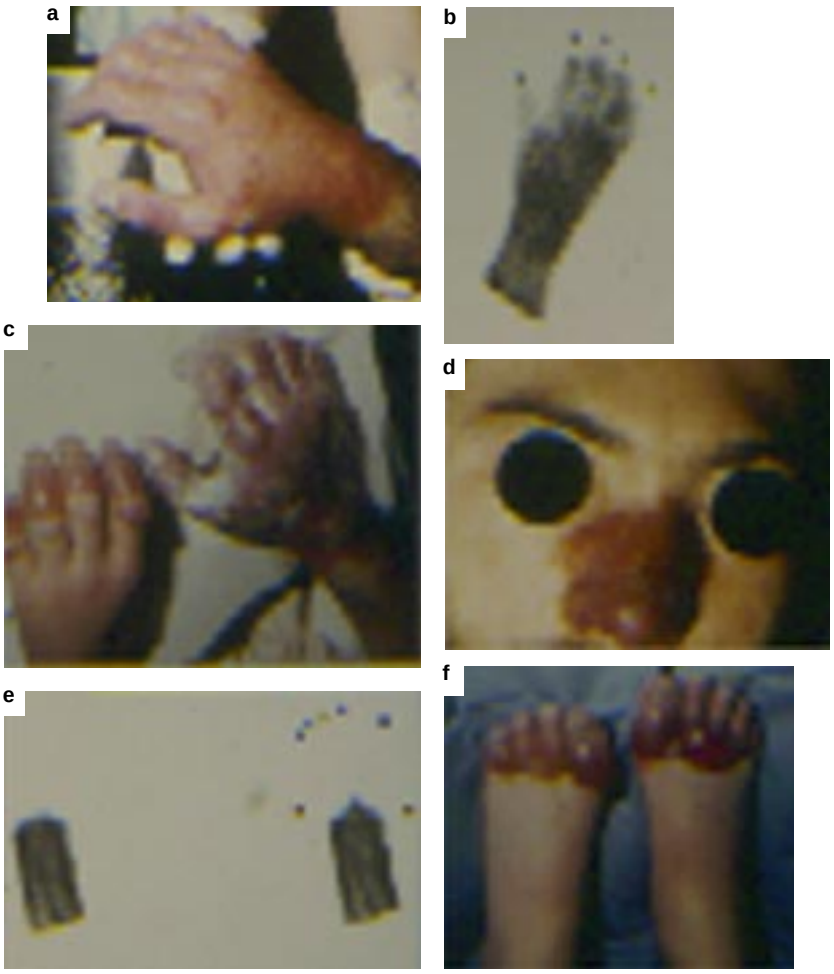
(**f**) After the surgical reduction of the Monteggia fracture, the postoperative technetium 99m examination demonstrated a partial return of capillary perfusion to both hands. However, after 2 weeks, severe necrosis, mummification, infection, and the edematous red-orange discoloration of tissue necrosis and vascular failure are present on the right hand. (**g**) At 30 days the dry, mummified tissues demarcating anatomically are present, and the left hand is ready for surgical debridement. (**h**) At the end of 4 weeks, guillotine-modified amputation of the metacarpophalangeal level on the right and at varied phalangeal levels on the left was performed. At 6 weeks, further debridement and split-thickness skin cover of both hands was performed. (**i**) At 2 months after the injury, a roentgenogram of the elbow revealed a soft-tissue calcification extending anteriorly from the radial head region. This was mushroom-shaped, measuring approximately 3 x 3.5 cm, somewhat ovoid. It appeared to represent either a myositis ossificans or calcified hematoma. A radial-ulnar synostosis was present at the ulnar fracture site and site of operation. (**j**) At 6 months after the injury, the right elbow was explored anteriorly. A large, pedunculated, bony mass was removed, which had lifted the radial nerve upward for 2 cm and displaced the radial artery and soft tissue. The mass originated from the radius, just below the radial head in the area of the annular ligament. The radial–ulnar synostosis was removed, permitting supination of 80°, a motion previously lost. (**k**) At 10 months after the injury, the patient has a "paddle hand" on the right, with a segment of thumb and phalanx remaining. Small remnants of the proximal phalanges remain as well. On the left, sufficient phalangeal residua are present for the patient to continue his work as an electronics engineer. A large toe transfer for a right thumb and web space releases are contemplated further on the right hand.

The findings in this case represent the disastrous results of severe associated fracture followed by thawing other than rapid rewarming, in this case spontaneous thawing, and represent the need for immediate care and early reduction of the fractures or dislocations. Thrombolytic therapy was inappropriate in this case because of the combined injuries to the head and neck and the thoracic spine, as the use of thrombolytic enzymes is considered likely to cause intracranial or intraspinal bleeding.



Atlas Fig. 26. Hypothermia and freeze–thaw–refreeze injury. (**a**) The home of a street person in an alder thicket. He was determined to be mentally incompetent. (**b**) Severe frozen feet after 10 days of freeze–thaw–refreeze injury on at least three occasions. The patient was found to be hypothermic (27.6°C, 81.7°F), with a blood glucose level of 900 mg/dL. He was warmed by peritoneal dialysis and, simultaneously, his frozen extremities thawed in a Hubbard tub. (**c**) Isotope technetium 99m studies of the feet demonstrated total block and failure of perfusion at the malleolar level. (**d**) At 72 hours after hospitalization, the feet were pulseless and lifeless, and gangrenous changes were seen to be developing in the toes. (**e**) Nine days after admission the patient developed evidence of acute bacterial sepsis and became febrile; a culture demonstrated overwhelming infection with *Proteus vulgaris*. A guillotine low-level amputation was performed.

Atlas Fig. 27. Freezing injury, with substance abuse, hypothermia, and vascular occlusion. A 25-year-old victim with frostbite to the face, hands, and feet was found semiconscious in a snow gully near a broken-down snowmachine. He was taken to a hospital in Anchorage, Alaska, 8 hours after rescue. On arrival at the emergency department, his core temperature was 86°F (33°C). His exposure time was said to be 12 to 18 hours; ambient temperature in the accident area: –4°F (–40°C); wind: 15 mph and gusting; wind chill factor: –85°F to –90°F.

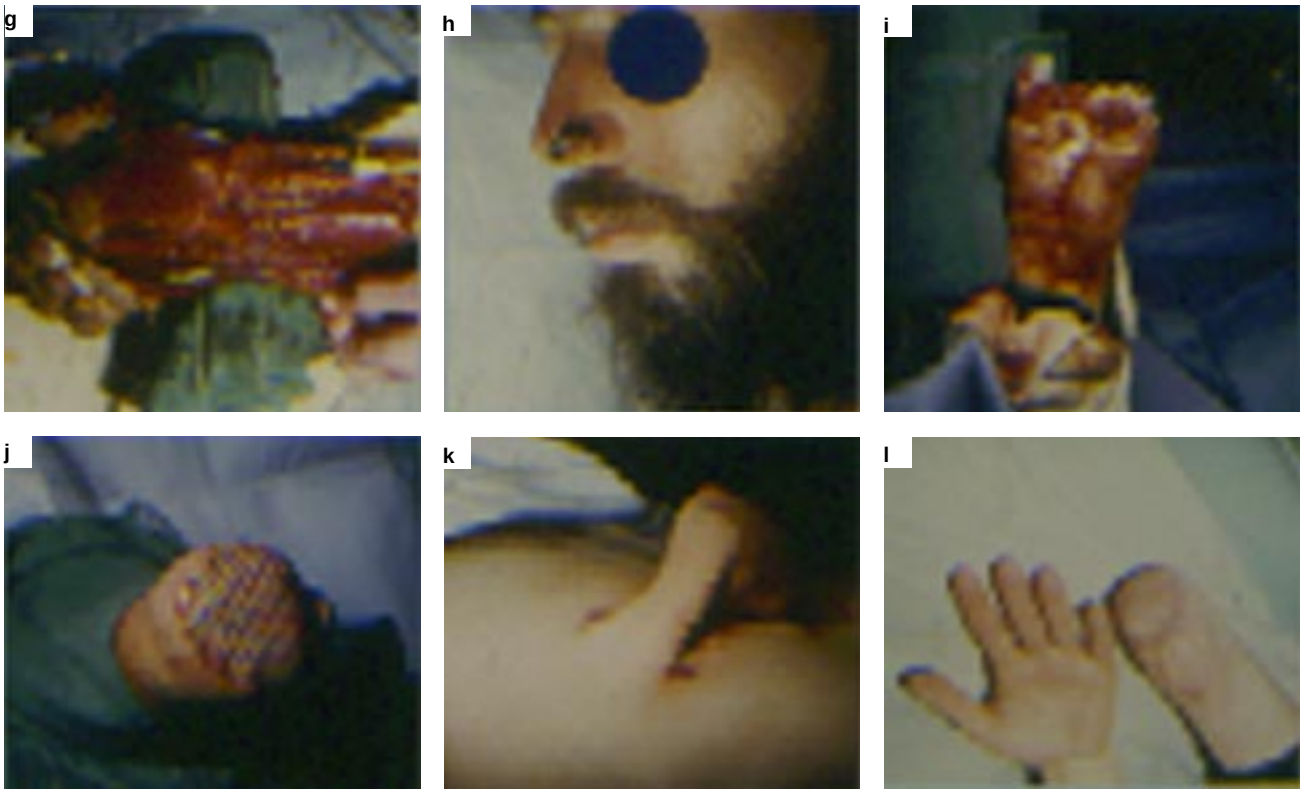


(**a**) The victim was found with the right sleeve of his coverall rolled tight over the mid forearm and frozen to the arm, giving a total tourniquet effect for the time he was lying in the snow. He was warmed in the field with warm blankets; and in the emergency department with a circulating warm-water blanket; warm, moist, inspired air; and brought to normothermia in a warm whirlpool Hubbard tank at 90°F (ie, he was rapidly rewarmed in warm water). A toxicology screen in the emergency department demonstrated a positive test for cocaine. (**b**) A technetium isotope (T99m) scan of the right hand and wrist made on admission demonstrated perfusion almost to the fingertips. Compartment pressures taken on the same day varied in the hand between 37 and 52 mm Hg. The hand was warm and, based on the T99m scan, appeared to have adequate perfusion. This evaluation is believed to have been marginal for a fasciotomy (**c**) The right hand is thickened, edematous, totally insensitive, and rapidly cooling.

(**d**) The nasal frostbite appears related to the victim's face-down position in the snow after the accident and aggravated by his snorting of cocaine the evening of the accident. The vasoconstriction of the nasal tissues—apparently secondary to (1) the severe contact with cold, snow, and ice, and (2) cocaine use—allowed increased tissue cooling. (**e**) A repeat T99m scan revealed no cellular perfusion distal to the wrist, a marked change in 3 days; the clinical change occurred in the last 6 hours of that time. (**f**) Adequate response to treatment was present in the feet, here demonstrated on the fourth day after the injury; T99m scan of the feet that day revealed adequate capillary perfusion.

(Atlas Fig. 27. *continues***)**

Atlas Fig. 27. *continued*



(**g**) Having demonstrated an increase in fascial compartment pressure of the hand, a fasciotomy of the mid palm was performed, demonstrating ulnar and radial artery occlusion. Clots were evacuated by small catheters and treatment instituted with a thrombolytic enzyme, slow-drip streptokinase. Massive bleeding resulted 3 hours postoperatively throughout the hand and operative site.

(**h**) The resolution of the nasal injury was present by the second week with the return of vascular supply. Treatment consisted primarily of the intermittent warm soaks as well as Dibenzyline* therapy (10 mg, orally, b.i.d.) utilized for treatment of all frostbitten areas.

(**i**) Because of sanguineous changes in the wrist (see [g]) and necrosis of all tissues, including blood vessels, open-pack amputation above the wrist was performed, with a split-thickness graft further added 2 weeks after the amputation. (**j**) The open, granulating stump was closed 3 weeks after the amputation. (**k**) A cross-abdominal, full-pedicle flap was applied to the forearm amputation 2.5 months later. (**l**) The final result was less than desirable. The severe loss of tissue and right hand amputation was considered the result of 12 to 18 hours of vascular occlusion to the hand, the depth and duration of freezing, and the failure to relieve the distal vascular tree artery and vein of severe clotting.

*phenoxybenzamine hydrochloride; mfg: SmithKline Beecham, Philadelphia, Pa

SURGICAL PROCEDURES

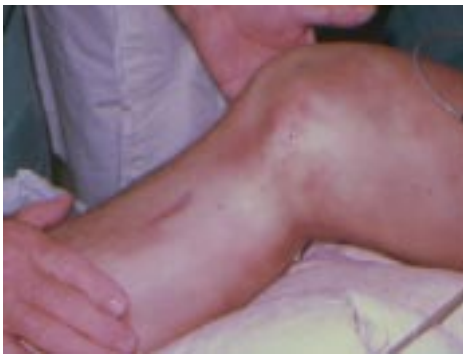
ATLAS EXHIBIT 10

FREEZING INJURY: SURGICAL PROCEDURES

1. Escharotomy, escharectomy
2. Bleb, bullae, wound debridement
3. Fasciotomy
4. Arteriotomy
5. Vascular wound repair
6. Dermal graft procedures:
   - Reverden (Davis) pinch graft
   - Split-thickness skin graft
   - Split-thickness skin graft (mesh)
   - Free, full-thickness skin graft
   - Cutaneous pedicle flap graft
   - Muscle, musculocutaneous vascular flap transfer
   - Very early digital debridement with vascular cutaneous flaps
7. Controlled subcutaneous balloon tissue expansion
8. Guillotine amputation, modified as necessary
9. Closed amputation, with closed suction irrigation
10. Closed or open reduction of fractures and dislocations
11. Joint contracture releases
    - Joint excision and replacement
    - Joint fusion
12. Soft-tissue web-space releases
13. Surgical regional sympathectomy
14. Periarterial, microdigital sympathectomy
15. Excision of sinus tract
    - If necessary, radical excision of squamous cell carcinoma within the sinus tract
16. Tissue compartment releases
    - Carpal, tarsal tunnel syndrome

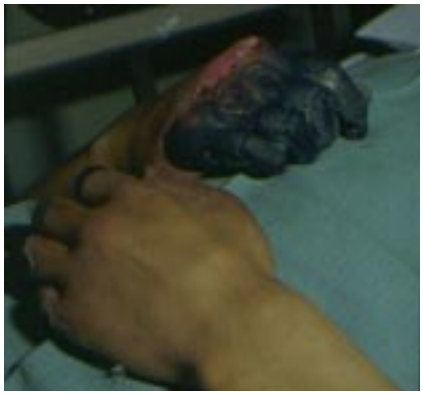
Atlas Fig. 28. Multiple surgical procedures are required in patients with freezing associated with multiple severe injuries. Late in May 1979 a climbing party of three fell approximately 2,000 to 3,000 ft (600–900 m), roped together, from a cornice of the west rib of Mount McKinley. Two died in the fall. The survivor was found hanging head down, a rope wrapped about his right knee and thigh. A University of Alaska research camp was sited at 14,000 ft (4,267 m). The fall was observed from the camp at approximately 10 PM. The medical research party reached the victims about 6 hours after the fall. The surviving climber was carried back to the 14,300-ft base camp and examined there by a physician climbing with another party. The patient was placed in a tent, where examination found him to be semicomatose and delirious.

a



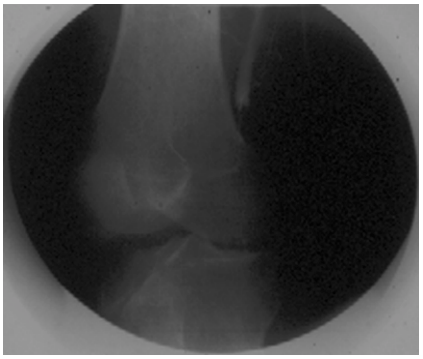
a. Frozen right leg to distal femur, thawed by rapid rewarming.

b



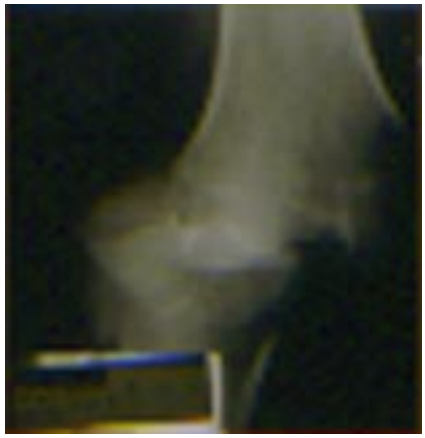
b. In the process of helicopter rescue (ambient temperature near 0°F) the patient lost his right hand cover. The wind generated by the chopper blades caused further cold injury to the already chilled climber.

c



c. The arteriogram demonstrates a vascular occlusion or arterial tear at the level of the distal femoral artery.

d



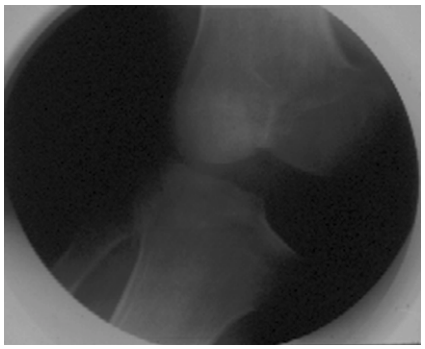
d. The roentgenogram demonstrates the lateral tibial plateau fracture.

(**a**) The patient had an obvious dislocation of the right knee with freezing of the hands and feet. (**b**) The hands began to thaw in the tent so that rapid rewarming was carried out. The right leg was packed in ice not only to prevent thawing but also because of the extent of the injury. Helicopter rescue was carried out approximately 20 hours after the fall.

On admission to the hospital in Anchorage, the patient was found to be semicomatose; both hands were thawed and were now edematous, cyanotic and still cool. X-ray examination demonstrated probable fracture of vertebra L2, and (**c**) an angiogram eventually revealed an obstruction of the popliteal artery 3 cm above the knee joint. (**d**) A lateral tibial plateau fracture was identified, with clinical evidence as well confirming a medial dislocation of the femur on the tibia. Rapid thawing of the right and left lower legs was performed in the emergency room with warm water and warm packs.

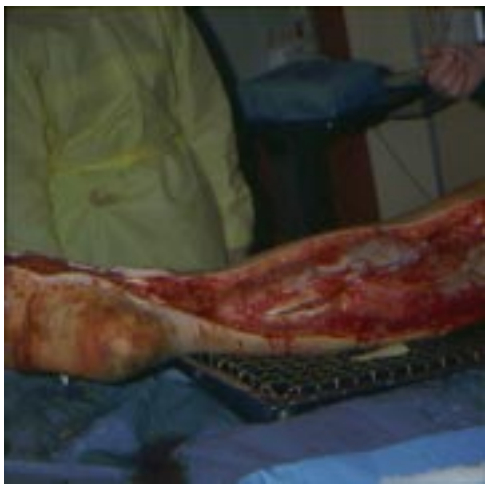
Atlas Fig. 28. *continued*

e



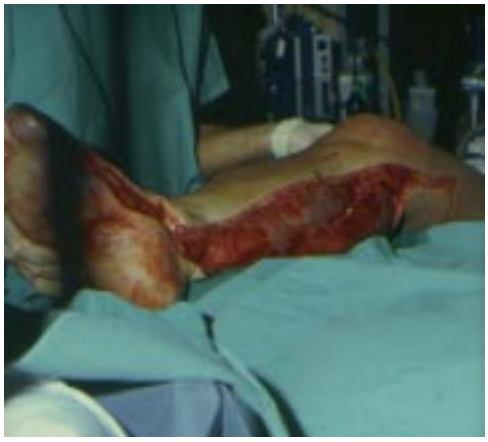
e. The radiograph of the frozen knee reveals the dislocation and loss of medial knee structures

g



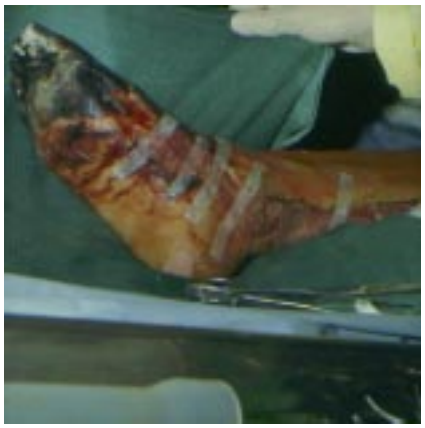
g. Demonstration of viable muscle in the distal lower leg.

f



f. Extent of postoperative fasciotomy and compartment pressure release with return of distal circulation after arteriovenous graft.

h

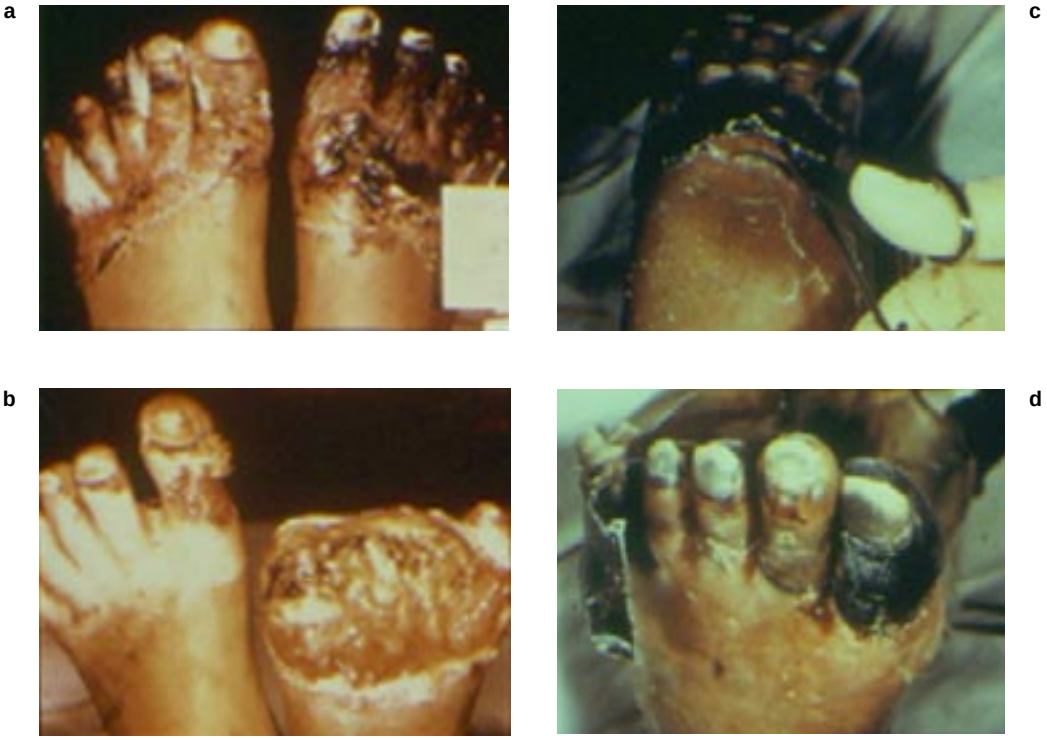


h. Final demarcation 3 weeks after repair of the knee structures and fasciotomy.

After thorough evaluation, the patient was brought to surgery and the completely thawed right leg then examined. The right lower leg was cold, edematous, and pulseless well about the knee. At the same time, a puncture wound of the right groin was found with multiple lacerations of the common femoral vein, caused by an accidental ice axe penetration during the fall. (**e**) At the time of surgery, the capsule of the right knee was found to be totally destroyed. The popliteal artery was contused and occluded, with subadventitial hemorrhage present. The injured, occluded artery was resected, and a 6-cm saphenous vein graft was taken from the left groin and sutured in place. (**f**) The fasciotomy, after arterial repair, was extensive and deep, revealing that tissues in the posterior compartment of the calf and distal femur were quite swollen and edematous. A necrotic segment of the gastrocnemius was resected. (**g**) Despite the 22 hours that elapsed from the time of injury (including the popliteal occlusion and laceration) to the time of rescue, the result was considered good, with tissue loss limited to toes at the metatarsophalangeal joint level.

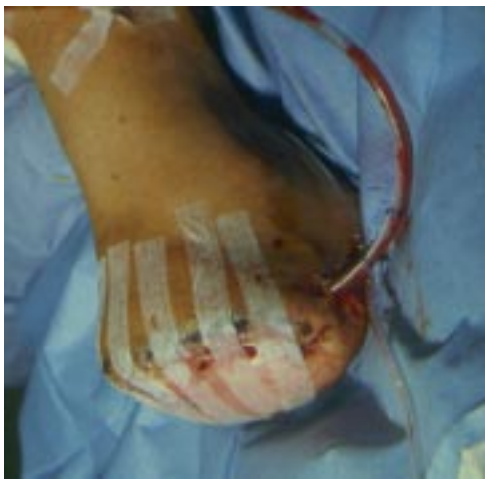
It is considered that by having the right leg frozen to the level of the tibial tuberosity and cool well above that, the metabolic needs of the extremity were minimized despite the laceration of the popliteal artery. The "metabolic ice-box" state may have allowed preservation of tissue and, along with the extensive fasciotomy, avoided compartment pressure injury and destruction of muscle, vessels, and nerves.

(**h**) At 3 weeks after the injury, the area of fascial release was covered with split-thickness skin graft, and the patient transferred to his native country for further care.



Atlas Fig. 29. Regardless of the thawing method, premature debridement through (**a**) wet, edematous, often infected tissues, including regional amputations, often results in (**b**) retraction tissues and a shorter stump.

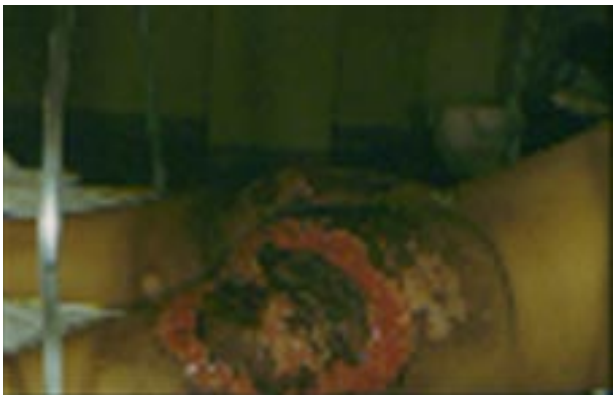
Amputation is not always necessary, however. An elderly woodcutter, who sustained severe frostbite injury to his feet, was referred for transmetatarsal amputation. (**c**) Careful dissection of the gangrenous epidermal cover revealed healthy epithelializing tissue under the eschar, and (**d**) his foot remained intact. Photographs c and d were both taken in the operating room on the same day.



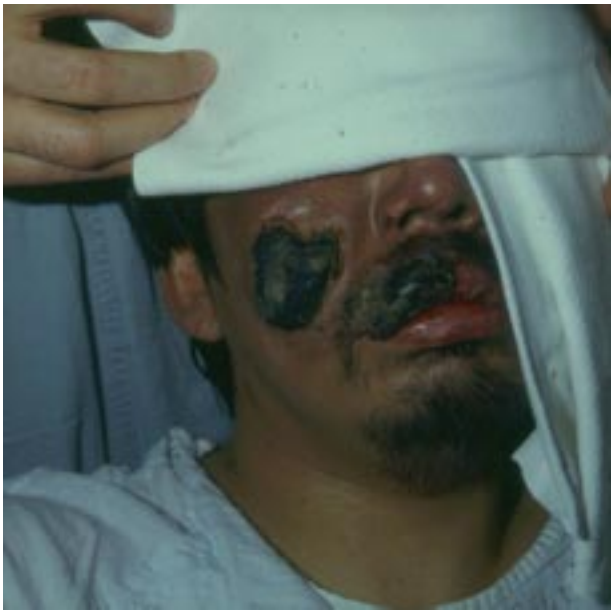
Atlas Fig. 30. Because of gangrenous and necrotic tissues, amputation (other than guillotine) at any level is followed by closed suction irrigation for 5 to 7 days.

UNUSUAL PRESENTATIONS

a



b



Atlas Fig. 31. Unusual presentations of freezing injuries. (**a**) A freezing injury, after contact with a bedspring and consequent loss of skin and superficial structures upon rising from the bed. Treatment: debridement and mesh graft care (not shown). (**b**) This Alaskan hunter was lost in a blizzard for 3 days with his face unprotected. Deep freezing of the right cheek and lips occurred. The freezing of the cheek has caused necrosis of a segment of the maxillary sinus, through the upper jaw and into the oral cavity.

SEQUELAE OF FREEZING COLD INJURY

ATLAS EXHIBIT 11

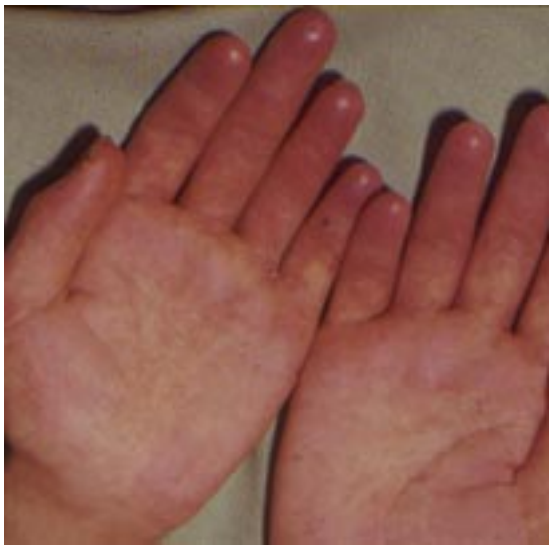
SEQUELAE OF FREEZING COLD INJURY

Transient, early signs:

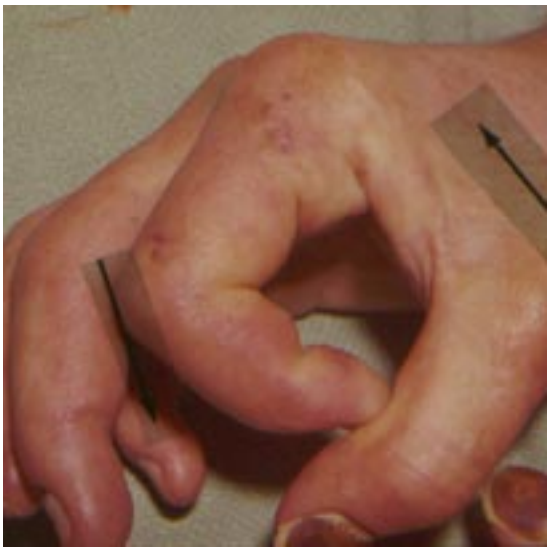
- Hyperhidrosis
- Hypesthesia or anesthesia of digits
- Limitation of motion in interphalangeal and metatarsophalangeal joints
- Swelling in interphalangeal and metatarsophalangeal joints
- Edema
- Thin, fragile epidermis in involved areas
- Nail loss
- Intrinsic muscle atrophy
- Fat pad atrophy of the distal tips

Long-standing, usually permanent signs:

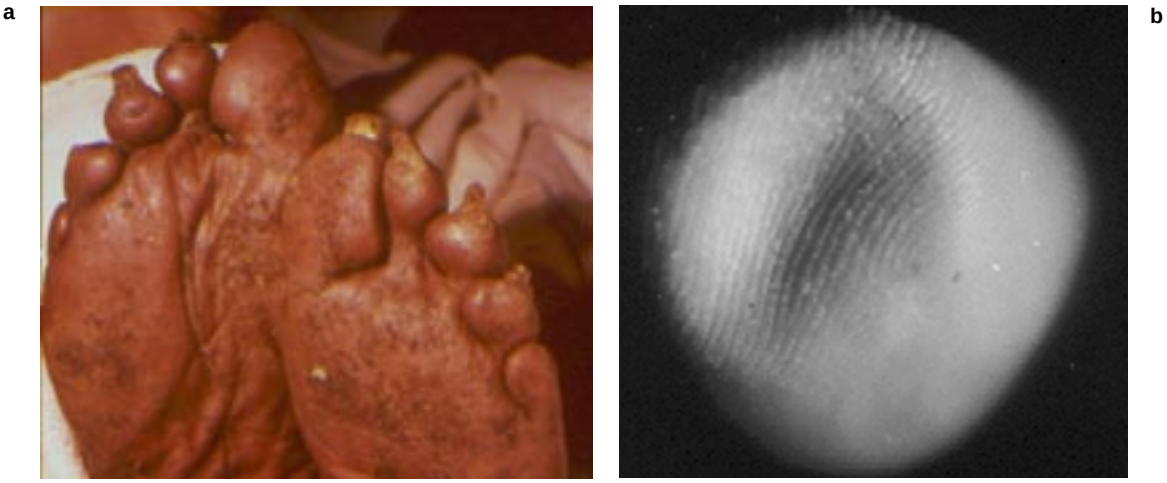
- Deep, fixed scars over the affected areas
- Atrophy or fibrosis of intrinsic musculature
- Contracture of digital joints (eg, hammer toe, claw toe)
- Loss of volar fat pad
- Hyperesthesia; digital tips with increased sensitivity to heat or cold
- Decreased proprioceptive sense of the digital tips
- Permanent nail deformity
- Roentgenographic evidence of periarticular and subarticular lytic destruction of bone and cartilage, especially of the phalanges
- Avascular necrosis of bone, especially of phalanges, metatarsi, and tarsi
- In children, epiphyseal necrosis or total destruction, with joint and phalangeal deformity, angulation, or shortening
- Chronic ulceration, infection, and osteomyelitis
- Decreased capillary perfusion
- Rarely, squamous cell carcinoma in a persistent sinus tract
- Interphalangeal joint immobility or fusion
- The ultimate of long-standing sequelae: amputation



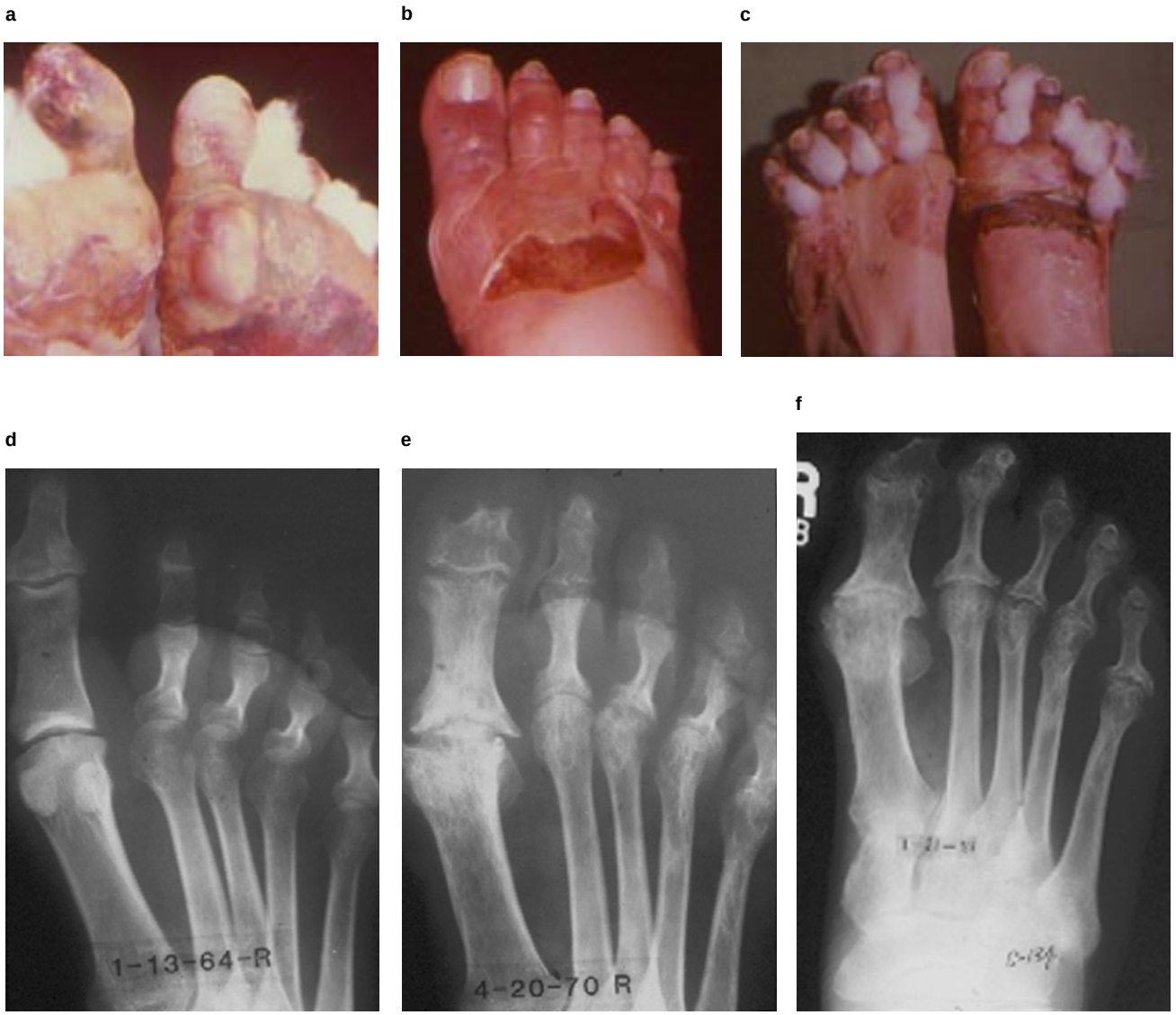
Atlas Fig. 32. Hyperhidrosis, a sympathetic nerve response, is sometimes a transitory sequela of freezing and thawing in superficial frostbite, but in deeper frostbite is often a permanent sequela.



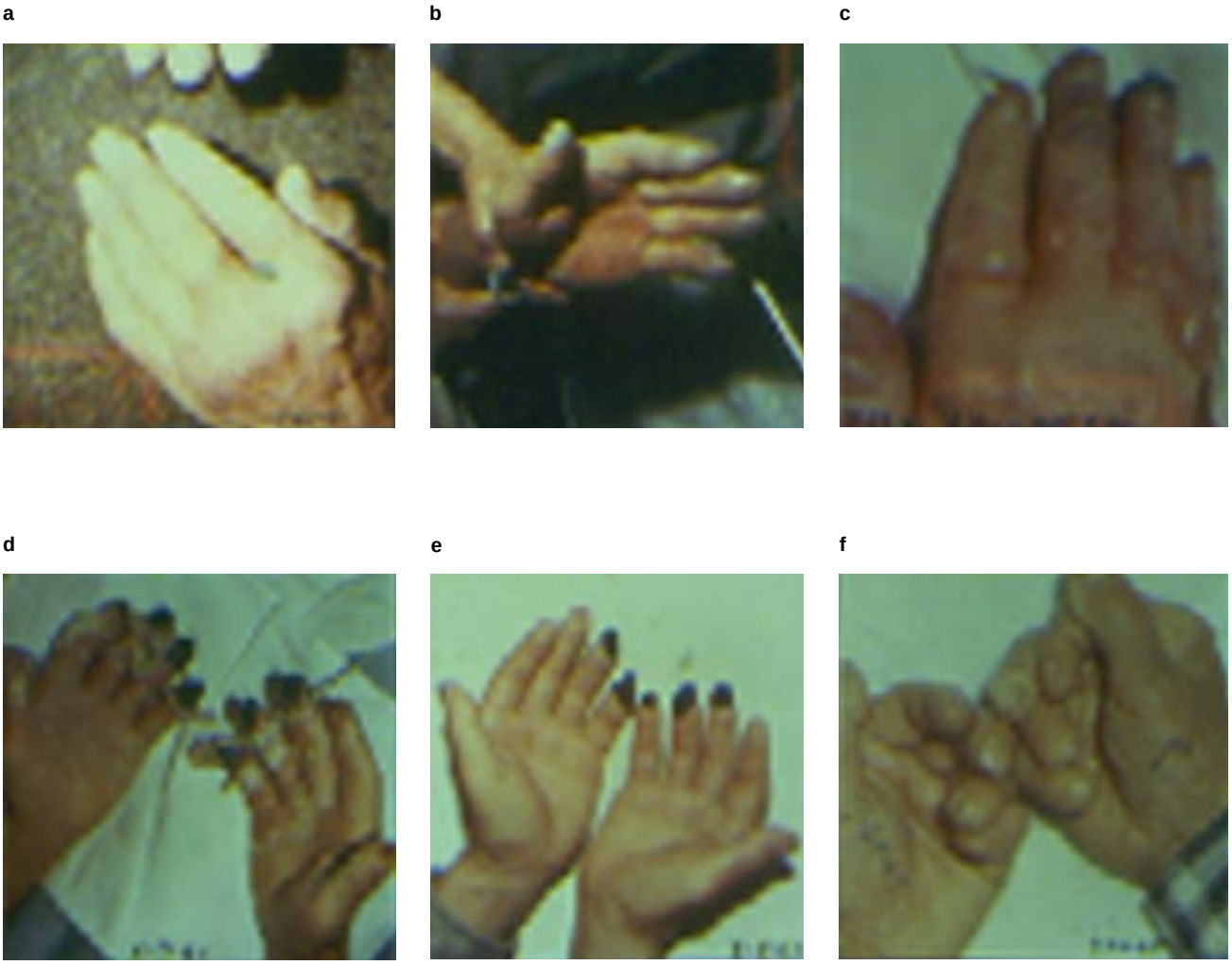
Atlas Fig. 33. Early and permanent loss of intrinsic muscle (first dorsal interosseus) and fat pad loss (volar fingertips), both demonstrated by arrows.



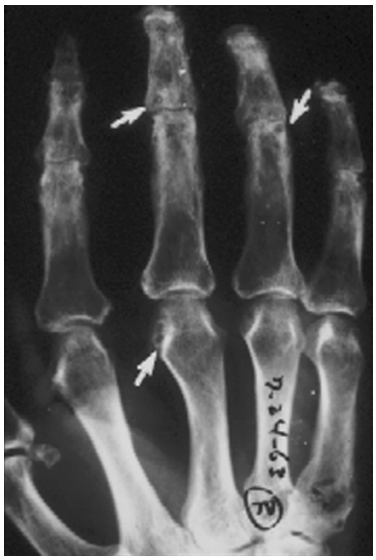
Atlas Fig. 34. (**a**) An Alaskan "sourdough," who came to the Yukon in 1901 and worked as a cook in gold camps, had frostbite on "lots of occasions." He treated each episode with cold or warm coal oil, a common Alaskan home remedy. He had evidence of multiple injuries: bulbous digital tips, fat-pad loss, and osseous atrophy. (**b**) Usually after any frostbite of the digital skin (even mild), loss of the fat pad of volar digits (here, a thumb) occurs. This loss also may occur after exposure over years to winter workers or sports people, especially those handling tent poles, lines, or other cold materials at low temperatures.

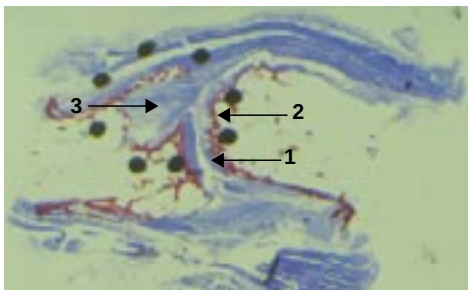


Atlas Fig. 35. Immersion and bilateral freezing, followed by rapid rewarming. After hip-high immersion in a creek, the victim walked back to his car (ambient temperature: –5°F [–20°C]; exposure: 3–4 h) and partially warmed his feet in his vehicle. Both feet were frozen, particularly the dorsum of the right foot. Rapid rewarming in warm water was carried out in the hospital. (**a**) After rapid rewarming following cold exposure of 3 to 4 hours, large, pink, distal blebs formed—a good prognostic sign. (**b**) The right foot, 10 days after the thaw. (**c**) At 3 weeks after the injury, viable tissues are present below the epidermal eschar; full-thickness loss is seen on the right dorsum. The eschar eventually sloughed and a mesh graft was applied to the remaining defect, which healed 10 days later. (**d**) A roentgenogram taken 7 weeks after the injury reveals no definite degenerative changes in the phalanges. (**e**) A roentgenogram taken 6 years after the injury reveals that lytic destructive changes are present in the distal joints and that osteocartilaginous destruction with marked joint limitation is present in the first metatarsophalangeal joint. (**f**) A roentgenogram taken more than 30 years later (36 y postinjury) reveals loss of joint surface in all metatarsal joints and narrowing of the interphalangeal joints. (**g**) A photograph taken the same day as the preceding roentgenogram shows the patient's typical loss of intrinsic muscle, swelling of the interphalangeal joints, mild hammer toe deformity, nail-bed deformity, early onychogryphosis, and onychomycosis.

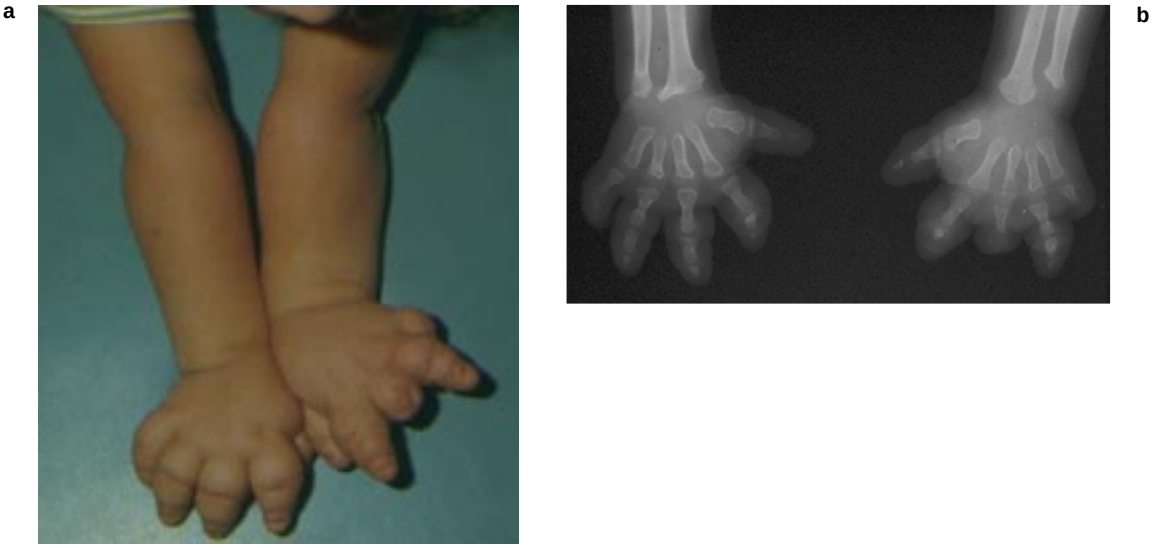


Atlas Fig. 36. Interrupted rapid rewarming in warm water. (**a**) Hard, cold, immobile, frozen hands (bilateral) were thawed in warm water (106°F) for less than 20 minutes. (**b**) Because of the second onset of a cardiac episode, warming was stopped before fingertips were flushed. (**c**) One hour postthaw, proximal blebs and cyanotic fingertips can be seen. (**d**) Two months postthaw, the fingertips are gangrenous. (**e**) Five months postthaw, mummification and separation of necrotic tissues can be seen. (**f**) Two and one-half years postthaw, the hands demonstrate flexion contractures of the interphalangeal joints, nail deformity, and intrinsic muscle atrophy. (**g**) The arrows on this roengtenogram made the same day (2.5 y postthaw) point to narrowing of joint spaces (cartilage necrosis) and lytic destructive changes of bone.

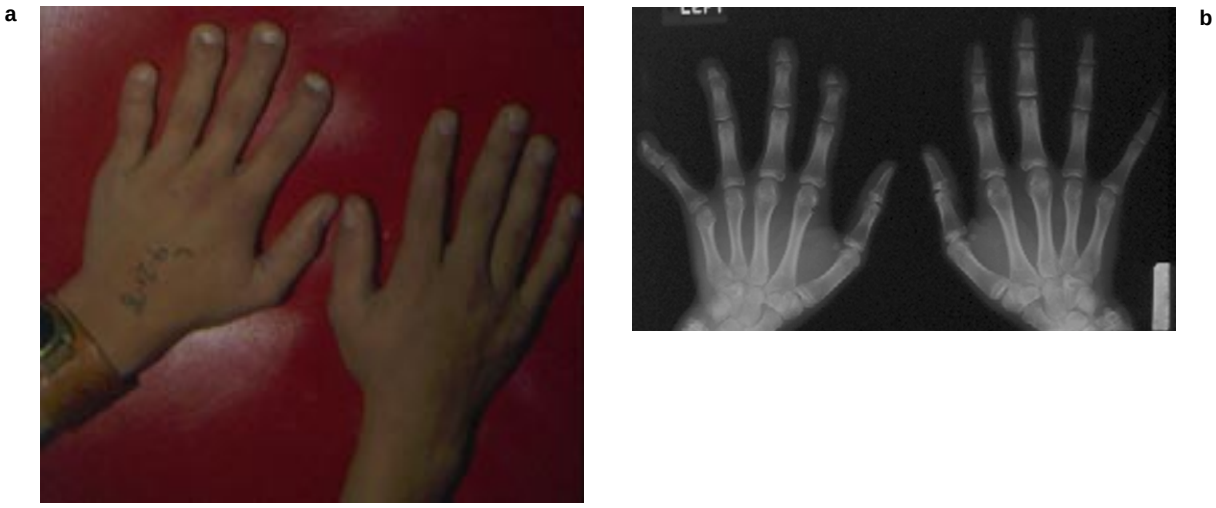




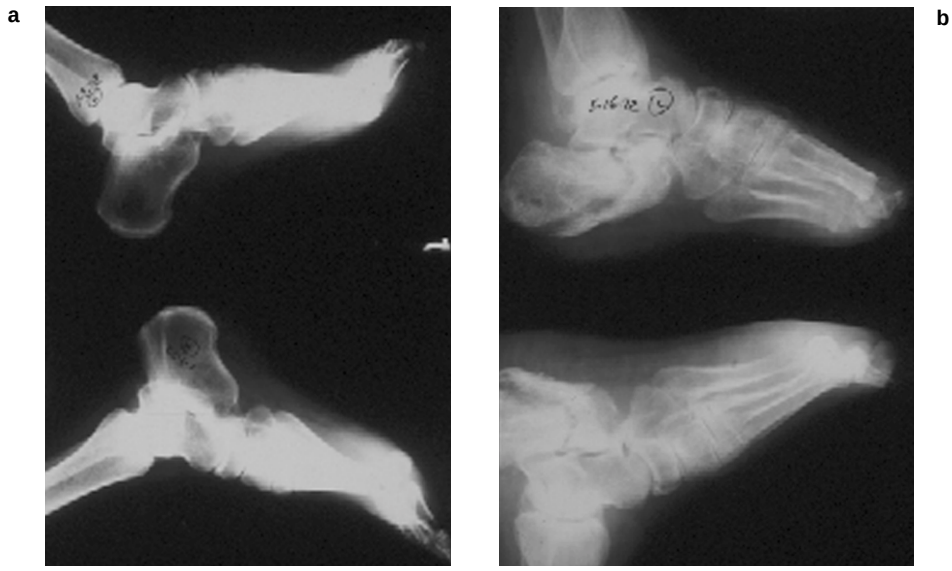
(**h**) The following findings were made at a postmortem biopsy of the proximal phalanx mid finger, right hand (trichrome stain) of the same patient, 15 years after the frostbite injury: (1) degenerative articular cartilage, (2) segmental destruction of subarticular bone proximal interphalangeal joint, and (3) periosticular destructive lytic lesion of bone with invasion of joint surface by dense collagenous tissue.



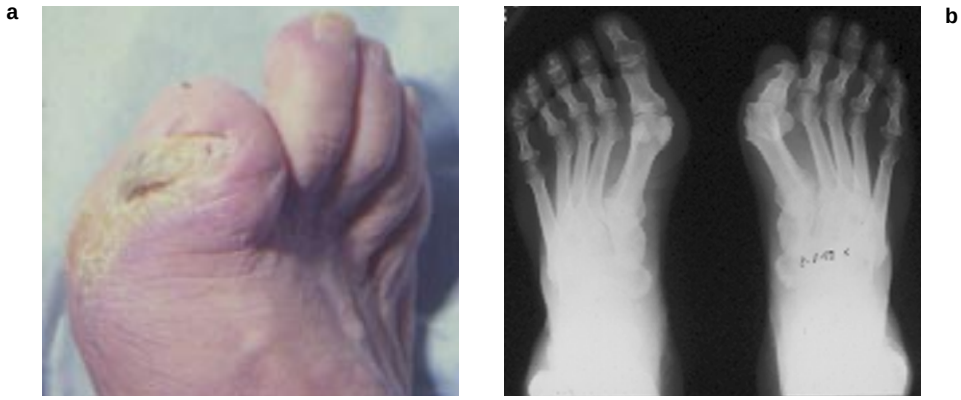
Atlas Fig. 37. A 4.5-month-old infant was found outdoors wrapped in a blanket; ambient temperature –30°F. The infant had severe hypothermia (core temperature < 70°F) and the hands and feet were frozen; thawing was accomplished in a warm water bath. The epiphyses of both hands and wrists sustained injury, and the infant suffered severe hypermobility of joints. (**a**) This photograph was made when the child was 5 years of age; only the long flexors and extensors of the hands are functional. All intrinsic musculature of the hands has been destroyed. (**b**) A roentgenogram, made when the child was 8 years of age, revealing the absence of the carpals and digital epiphyseal necrosis with phalangeal shortening. An arthrogram of the wrists demonstrated total absence of cartilage as well as bone. This roengtenogram demonstrates the total epiphyseal lytic destruction of all carpi bilaterally and all epiphyseal plates of the interphalangeal joints. More than 20 years after the injury, the patient's hands had not grown since they were frozen at age 4.5 months.



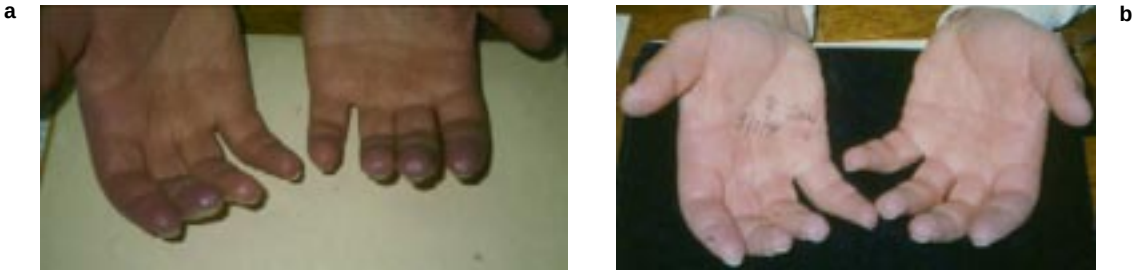
Atlas Fig 38. Resultant epiphyseal necrosis from freezing. While carried in an infant "backpack" carrier, an infant younger than 6 months of age lost the protective glove from his left hand. The incident occurred in the vicinity of Point Barrow, Alaska, where freezing injuries can occur in less than 1 minute at –50°F. (**a**) Epiphyseal necrosis 12 years later. Note on the left hand the mild length discrepancy, ulnar deviation, and distal digits at the interphalangeal joints. (**b**) A roentgenogram of the same patient on the same day demonstrates distal digital atrophy and distal interphalangeal epiphyseal necrosis.



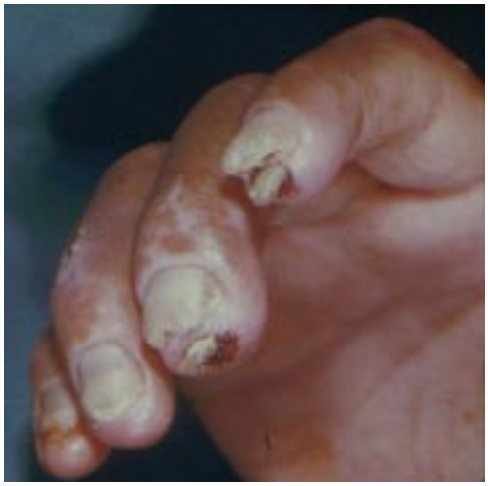
Atlas Fig. 39. An individual with alcoholic peripheral neuropathy was unaware of his freezing injury on a snowmobile trip until his toes mummified 3.5 weeks later (the thawing was probably spontaneous). (**a**) The roentgenogram taken 3.5 weeks after the injury demonstrates the appearance of osteoporotic calcanei and amputation of the distal digits. (**b**) Unexpectedly, avascular changes with collapse of calcaneal structures occurred. Owing to the peripheral neuropathy, the bony changes were asymptomatic. A roentgenogram taken 4.5 months later (5.5 mo postinjury) demonstrates gradual development of severe, avascular necrosis of the calcanei and tarsal joints (generally, degenerative changes are not seen so soon after the freezing injury). The patient eventually required triple arthrodesis of both feet.



Atlas Fig. 40. Changes in the large toe are evident 23 years after the feet were frozen in dogsled travel in interior Alaska, with multiple-day exposure. (**a**) General destruction of interphalangeal joint toe I, and subluxation of metatarsophalangeal joint after osteonecrosis. Severe onychogryphosis is present. (**b**) Roentgenographic evidence of subluxation, metatarsophalangeal joint, large toe, destruction of segment of intact phalanx and interphalangeal joint.

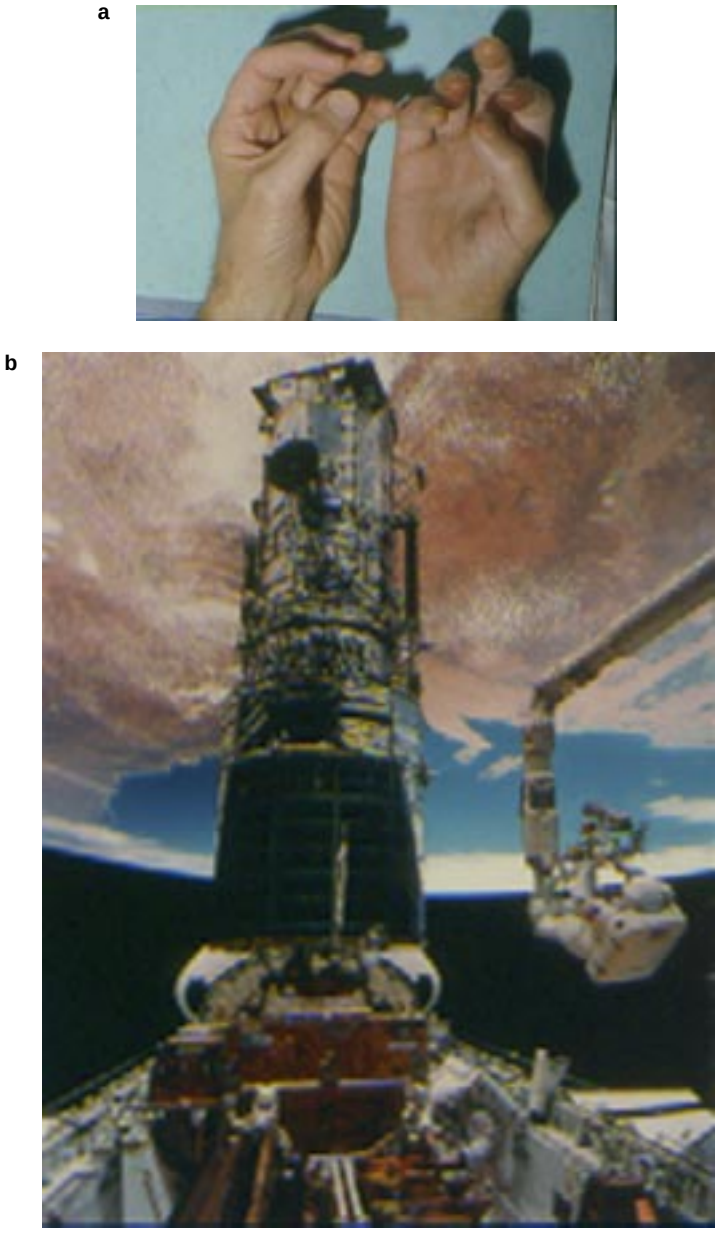


Atlas Fig. 41. (**a**) Six months after superficial frostbite at subzero temperatures, the patient still has chronic cyanosis of the fingertips. (**b**) The patient developed a labile vasomotor hand problem, occasionally "spitting" out calcific pieces from fingertips (see the ring fingertips of both hands). Now the patient is extremely sensitive to cold.



Atlas Fig. 42. Necrosis of fingertips and extrusion of the distal phalanx almost 2 years after an Aleutian fisherman's immersion injury followed by freezing injury. This patient carried on his usual activity as a fisherman after he recovered from his injury, even ignoring the drainage from the localized osteitis of bony tips. Eventually he was brought to the Alaska Native Hospital with appendicitis, during which time the extended bony segments were excised and the wounds closed over small, transverse drains.

SPACE-AGE THERMAL INJURIES



Atlas Fig. 43. Nonterrestrial high-altitude cold injuries are sometimes seen now that astronauts venture into space, where temperature extremes of hot and cold are far greater than those seen on Earth. This astronaut's hands, despite protective gloves, sustained superficial freezing injury at an extravehicular temperature near –143°F while training at the National Aeronautics and Space Administration facility at Houston, Texas. He was treated there with rapid rewarming. (**a**) Three weeks after the injury the astronaut was transported to Alaska for further treatment. His isotope studies demonstrated excellent capillary perfusion of all digits. In this view, he is demonstrating the recovered range of motion in his hands; the discoloration seen in the fingertips of the right hand is temporary. (**b**) Almost a year after the injury and wearing newly designed gloves, the astronaut (Storey Musgrave) was able to go into space and perform extravehicular work on the Hubble space program telescope without recurrent injury.