Chapter 19

ENVIRONMENTAL MEDICINE: HEAT, COLD, AND ALTITUDE

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INTRODUCTION

Since the beginning of recorded history, there are clear descriptions of the effect of the environment on military campaigns. The armies of Alexander in Central Asia, Hannibal in the Alps, and Napoleon in Russia all suffered the consequences of harsh climate. American military personnel, too, have had ample experience with cold and heat from Valley Forge to the Persian Gulf. And there does not seem to be a reduction in the requirement for military forces to deploy and operate in these places. Just in the 1990s, military conflict has appeared in the altitude of the Himalayan and Andean mountains, in the heat of African and Asian deserts, and in the cold of Central Europe and Central Asia.

This chapter is concerned with three terrestrial environments: hot, cold, and high altitude. Each is characterized by a dominant physical stressor: heat, cold, or hypobaric hypoxia. These three are military occupational stressors that produce measurable physiological or psychological strain, which is a necessary first step on the path to illness (Exhibit 19-1). Although the incidence of illness and injury is a stochastic process and predictable only in population terms, strain is universally present in deployed populations. These stressors are not novel or exotic but everyday, so we assume that their management is within everyone's experience and competence. That is not necessarily the case, to the detriment of service members deployed or training in these environments around the world.

EXHIBIT 19-1

MILITARY OCCUPATIONAL STRESSORS

- Environmental Toxic Hazards
- Dehydration
- Weight Loss
- Physical Stress
- Physical Fatigue
- Emotional Fatigue
- Cognitive Fatigue
- Climatic Extremes
- Protective Uniforms

GENERAL PRINCIPLES

There are several important principles concerning the relationship of these environmental stressors and illness. The first of these is that all these stressors are interactive and synergistic. The strain each produces cumulates with the strains produced by all the others to cause a general reduction in physical and psychological performance and to increase the likelihood of illness and injury. Typically, military deployments expose service members to several of these stressors simultaneously. These exposures are an inescapable effect of the psychological, physiological, and social dislocations of military operations.

The clinical illnesses consequent on these exposures usually reflect the net effect of several stressors. For example, dehydration, exercise-related heat exposure, and sleep loss all independently contribute to heat illness. Moderating any one of these three factors will reduce the risk. So, in circumstances where sleep loss cannot be mitigated, efforts to guarantee adequate hydration and to control work rates in the heat become even more important preventive medicine tools.

It is important to remember, though, that a particular exposure does not result exclusively in any particular clinical illness. For example, trauma from a vehicular accident can be as much a consequence of altitude exposure as acute mountain sickness.

Frequently, one characteristic of an area of operations will be so extreme as to seem to require exclusive attention and intervention. However, the amount that this attention to a single stressor distracts attention from other stressors will prevent the implementation of a successful program of surveillance and prevention. Important and effective strategies to sustain health and performance may be overlooked if medical personnel become too focused on any one stressor.

A MODEL FOR ENVIRONMENTAL STRAIN AND DISEASE

The conceptual model used throughout this chapter expresses the idea that strain is a result of exposure to an environmental stressor moderated both by the capability of the individual to tolerate the stress and by protective technology. Disease only occurs in the presence of strain. It is important to remember that there are many potential clinical expressions of strain, including psychological stress reactions, accidents

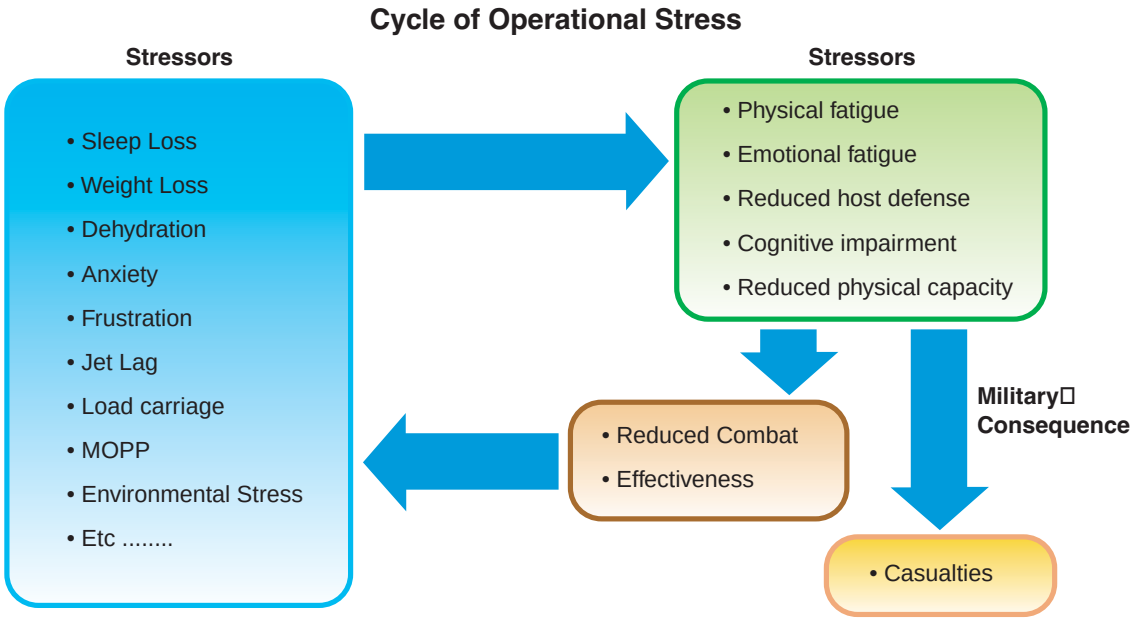

Fig. 19-1. Operational stressors reduce combat effectiveness and increase casualty rates through a variety of mechanisms. Loss of combat forces through stressor-related attrition increases the operational load on those not yet affected which contributes to an acceleration of the loss of combat effectiveness.

and trauma, and environmental illnesses. The individual feels the effect of all the stressors to which he or she is exposed. The cumulated strain will degrade physical and psychological performance, reducing military effectiveness and increasing casualty rates (Figure 19-1). The loss of personnel will take a toll in morale and increase the demands on those who remain, a process that will accelerate the accumulation of strain and further degrade unit effectiveness.

HOT ENVIRONMENTS

Heat and the other threats associated with hot environments are a constant of military training and operations. Acute and chronic heat illnesses have affected the outcomes of military campaigns since there have been military campaigns.[1,2] Heat illnesses continue to be a threat to US forces today.[3,4] Four of the major deployments in the 1990s were to hot environments: Panama, the Persian Gulf, Somalia, and Haiti. Heat stress affected operations in each of these deployments, both as a cause of casualties and as a threat complicating logistics and maneuver.

As the US experience in the Persian Gulf demonstrated, when military forces understand the threat of heat stress, provide appropriate logistic support, and incorporate prevention into the planning and execution of operations, heat illness is almost entirely preventable.[5] Heat illness rates have also fallen in the ordinary day-to-day activities of training and operations, but exertional heat illness, hyponatremia, and rhabdomyolysis continue to be common diagnoses among military populations and remain a cause of death and disability.

The Environment

Hot environments are characterized by a combination of temperature, humidity, and radiant heat; by affecting the rate at which heat energy can be dissipated from the body, these factors are associated with intense heat strain and dehydration during work. These conditions are usually thought of as confined to tropical or desert regions, but hot environments are encountered in every operational setting. In ground operations, the microenvironment of protective uniforms, even when outside temperatures are low, can be tropical and produce considerable heat stress. Military vehicles,[6] aircraft cockpits,[7,8] and mechanical spaces aboard ship[9] expose service members to high radiant and ambient heat loads. The outcomes of exposure to all hot environments are the same: reduced performance and illness.

Hot regions of the world, where heat is a climatic norm, present other threats beside heat stress and heat illness. They include limitations of water supply from either low rainfall or poor water quality,

skin disease from sunburn or miliaria, and bronchospasm from dry air and dust.

Physiological Adaptation to Heat Exposure

Humans have well-defined physiological mechanisms to counteract rises in body temperature from either internal heat production or environmental heat stress.[10] These mechanisms support our need to maintain a narrow range of body temperature for optimal function. In response to a rise in body temperature from an internal or external heat source, we increase both cutaneous blood flow and sweating.[11] Heat energy is then dissipated to the environment either directly from the warmed skin surface by conduction-convection and radiation[12] or by evaporation of sweat (Exhibit 19-2).

The rate of direct transfer of heat energy by con-

EXHIBIT 19-2

CORE TEMPERATURE AND THE HEAT BALANCE EQUATION

Core temperature is determined by the balance between heat loss and heat gain from the environment and metabolism. If, on average, the two are equal, core temperature will remain constant, permitting optimum function. If heat gain exceeds heat loss, core temperature rises; conversely, if heat loss exceeds heat gain, core temperature will fall.

Heat balance can be expressed algebraically in the heat balance equation

$$S = M + R + C - E$$

S = Net change in heat content (heat storage)

M = Metabolic heat production, always positive

R = Radiation

R = Radiation

C = Conduction/convection

E = Evaporation, always negative

Metabolic heat production (M) varies with activity. On average, an adult male at rest generates about 80 to 90 kcal per hour (roughly equivalent to a 100 W bulb). Maximum aerobic exercise increases metabolic heat production to about 10 times the resting rate. Individuals performing sustained hard physical work (eg, digging, marching under a load) who can control the rate of exertion will usually work at no more than four to five times their resting metabolic rate. Shivering can increase metabolic rate up to seven times resting level.

Radiation (R) is the loss or gain of heat in the form of electromagnetic energy. The direction of heat energy transfer is from warmer to cooler objects. The warming sensed standing in direct sun or near a hot surface is produced by radiative heat gain. We are warm objects in a cool environment and so radiate heat energy. Radiant heat gain and loss can be moderated by material barriers, which are considered either "shade" or "insulation" depending on the direction of the radiant heat energy flow.

Conduction and convection (C) are the mechanisms of heat energy transfer when there is physical contact between two materials of different temperatures. Conductive heat transfer occurs between two surfaces of different temperature. Conductive heat gain or loss is particularly significant when lying on hot or cold ground. Convection occurs between a surface and a fluid, such as air or water. The change in fluid density due to heat transfer causes the fluid to move and tends to maintain the thermal gradient. Wind and water currents add to the heat transfer process of natural convection and are significant components of heat gain and loss in extreme environments.

Evaporation (E) of water on the body surface causes heat loss. Each liter evaporated transfers 540 kcal to the environment. Sweat evaporation does not depend on the relative humidity but rather on the difference between the vapor pressure of sweat on the skin and the vapor pressure of water in the air adjacent to the skin. Even in cool-wet environments of high relative humidity, sweat on warm skin can evaporate into the air because the vapor pressure of the sweat exceeds that of the water vapor in the air. Sweating is an important form of heat loss in cold environments.

Adapted from: United States Army Research Institute of Environmental Medicine. *Medical Aspects of Cold Weather Operations: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1993: 4–5. Technical Note 93-4.

duction-convection and radiation depends on the difference in temperature between the body surface and ambient and radiant temperatures of the environment. The two routes of direct energy exchange (radiation and conduction-convection) between the body surface and the environment are two-way streets. If the body surface is warmer than the environment, the body will lose energy to the environment. If the converse is true, the body will gain heat energy from the environment. When the environment is sufficiently hot to cause heat gain by the direct transfer routes, evaporative cooling is the only thermoregulatory mechanism available to control body temperature.

Sweating is primarily controlled by the central nervous system.[13] Core temperature increases detected by thermosensitive neurons in the hypothalamus stimulate increases in skin blood flow and sweating. Sweat production rates can exceed 2 L/h for short periods and can reach 15 L/d. Each liter of sweat evaporated from the body surface removes approximately 540 kcal of heat energy. Under conditions that allow rapid evaporation (eg, the low humidity of deserts), the daily cooling capacity of the sweating mechanism is several thousand kilocalories, adequate to maintain body temperature even during vigorous work in the heat.

Physical work causes an increase in cardiac output and the redistribution of blood flow toward the working muscles and away from the viscera.[10] As exertion elevates core temperature, an additional portion of the cardiac output is directed to the skin for thermoregulation, and visceral flow is further reduced. High sweat rates will quickly compromise blood volume. Therefore, work in the heat requires constant fluid replenishment. Since the maximum rate of water absorption in the gut is about 20 cc/min or 1.2 L/h,[14] compensation for high sweat rates requires rest periods with reduced sweat rates and time for rehydration.

Acclimatization

Acclimatization to heat exposure is a true physiological adaptation that is critical to optimum performance and health in hot environments.[15–17] Both the rate of acclimatization and the degree of acclimatization achieved depend on the thermal stress to which an individual is regularly exposed.[18] Achieving the maximum rate seems to require about 1 to 2 hours of continuous exercise per day. Substantial acclimatization develops in 5 days of daily heat exposure and, for all practical purposes, is complete in 10 to 14 days.[19]

The acclimatization response includes these important physiological adaptations: a lowering of the threshold for the onset of cutaneous vasodilation and sweating,[11] an increase in the rate of sweating for any given core temperature, and a reduction in the concentration of sodium chloride in sweat[13] (Figure 19-2). The combination of a lower threshold and higher sweat rates allows a more vigorous response to heat exposure and increases the opportunity for evaporative cooling. In environments where evaporation contributes to cooling, acclimatized individuals can maintain lower body temperatures for any amount of heat stress. High sweating rates reduce the opportunity for the sweat gland epithelium to conserve salt, so at higher sweat rates, the concentration of salt in sweat rises. Acclimatized sweat glands conserve salt more effectively and produce sweat with a reduced salt concentration for any given flow rate. This conservative phenomenon is an important protection from salt depletion in hot environments. Furthermore, reducing the salt content of sweat increases the proportion of intracellu-

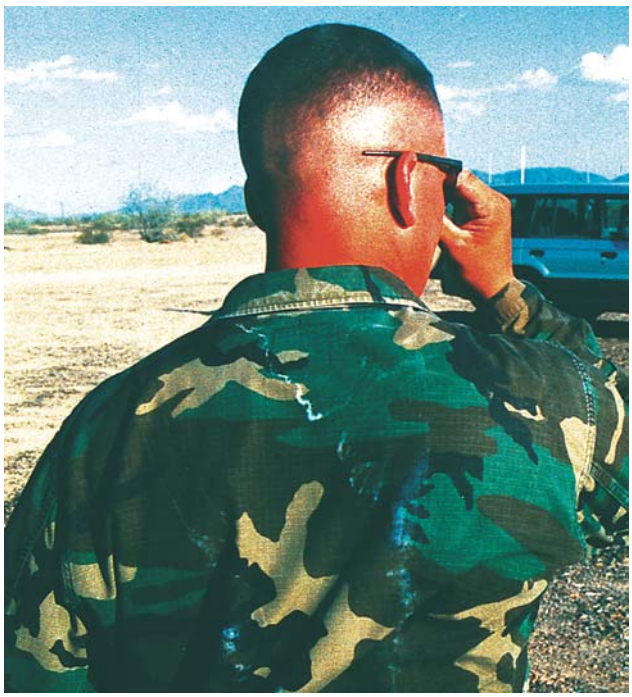

Fig. 19-2. This soldier has just finished his first day of work in a hot desert environment after air travel from a temperate climate. He is not yet acclimatized and has not yet developed salt conserving mechanisms appropriate to his high sweat rates. He has lost enough salt in his sweat to cause a saline crust on his uniform. Photograph: Courtesy of Dr. Robert E. Burr.

lar water contributing to sweat formation. Consequently, for any given amount of body water lost as sweat, less will be taken from the extracellular fluid, thus conserving plasma volume.

Heat Stress and Heat Strain

Heat stress is the net effect of the metabolic heat load and the environment; it is the force acting to increase core temperature. The heat stress of an encapsulating uniform (eg, MOPP and HAZMAT gear) is much more related to the environment inside the uniform than to the outside environment. Heat stress that does not exceed the ability of the individual to maintain an acceptable range of core temperature is considered compensable. Heat stress that exceeds that level is considered noncompensable.[20] Noncompensable heat stress will produce heat illness if the exposure is long enough. Encapsulating, or occlusive, uniforms are notorious for creating noncompensable heat stress environments because they limit both evaporation and direct heat exchange and are physically demanding to wear.

Heat strain is the change in the individual exposed to heat stress. It includes the physiological and psychological consequences of the rise in core temperature, thermoregulatory load, and dehydration.

PREVENTION OF HEAT ILLNESS

The primary prevention of heat illness depends on a thorough analysis of the factors that affect the likelihood of heat illness and on plans to mitigate those factors that increase risk and maximize those that reduce risk. This section will review the assessment and control of the principal groups of factors in heat illness: heat stress, thermocompetence, and technology. The conceptual relationship between heat stress and heat strain is shown in this equation:

$$(1)\quad \text{Strain} = f\frac{\text{Time} \bullet \text{Heat Stress}}{\text{Thermocompetence} \bullet \text{Technology}}$$

Heat Stress

The heat stress to which service members will be exposed must be known if effective preventive measures are to be taken. Heat stress can be environmental (exogenous) or metabolic (endogenous).

Environmental Factors

There are a number of metrics that are used to measure environmental heat stress.[21] The simplest and most commonly used is ambient temperature, but it does not include allowances for humidity or radiant heat load and can dangerously understate the actual heat stress imposed by an environment. To address that limitation, other metrics have been developed. The Wet Bulb Globe Temperature (WBGT) Index is the most commonly used of these multifactorial indices.[22] It incorporates independent measurements of ambient temperature, radiant heat load, humidity, and wind speed. The measurements of three instruments are combined as a weighted average to calculate the WBGT Index. The wet bulb thermometer estimates humidity and air movement (weighted at 0.7). The black globe thermometer estimates solar load (weighted at 0.2), and the dry bulb thermometer measures ambient air temperature (weighted at 0.1). The high weighting of the wet bulb temperature acknowledges the critical importance of air moisture on evaporative cooling in hot environments (Figure 19-3).

Whatever environmental heat stress metric is used, it should be measured in circumstances as close as possible to those in which the service mem-

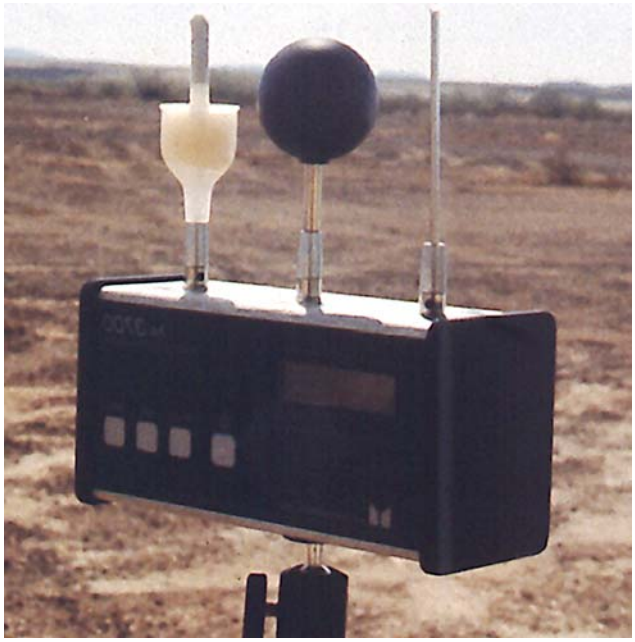

Fig. 19-3. This is an automatic instrument deployed to provide continuous real-time estimates of the Wet Bulb Globe Temperature Index. It should be set up at the spot where the activity is to be conducted.
Photograph: Courtesy of Dr. Robert E. Burr.

bers will be operating. These indices can vary tremendously over short periods of time and distances and in unpredictable ways. For example, on a sunny, calm day, an open field may have greater heat stress than an adjacent forest, but on a windy, cloudy day, the forest may have the greater heat stress. Heat stress indices calculated for a whole installation or region are only general guides. Particularly when conditions seem extreme, on-site measurements are essential. There is no substitute for knowledge of local conditions. Rapid changes in temperature or humidity present increased risk because those exposed are unacclimatized and may not accommodate to the change in weather.

Occlusive uniforms are an important source of heat stress.[23,24] By retarding the transfer of water vapor and heat energy to the environment, they create their own microenvironment. The air trapped in the uniform is warmed by the skin and saturated with water vapor from sweat, so that the service member's immediate environment becomes extremely hot and humid. The only opportunity to moderate the heat and humidity inside the uniform is to transfer water vapor and heat through the fabric—just the transfer the uniforms are designed to prevent. Protective uniforms that incorporate a mask can interfere with hydration, contributing to the development of heat illness (Figure 19-4). Heat exhaustion is the most common heat illness associated with protective uniforms but exertional heat stroke can also occur.[25]

Metabolic Factors

The metabolic component of heat stress can be estimated from knowledge of the work that is to be performed. Unlike civilian industrial hygiene practice,[26] military work is usually divided into four categories of intensity, and these categories are used instead of specific numeri-

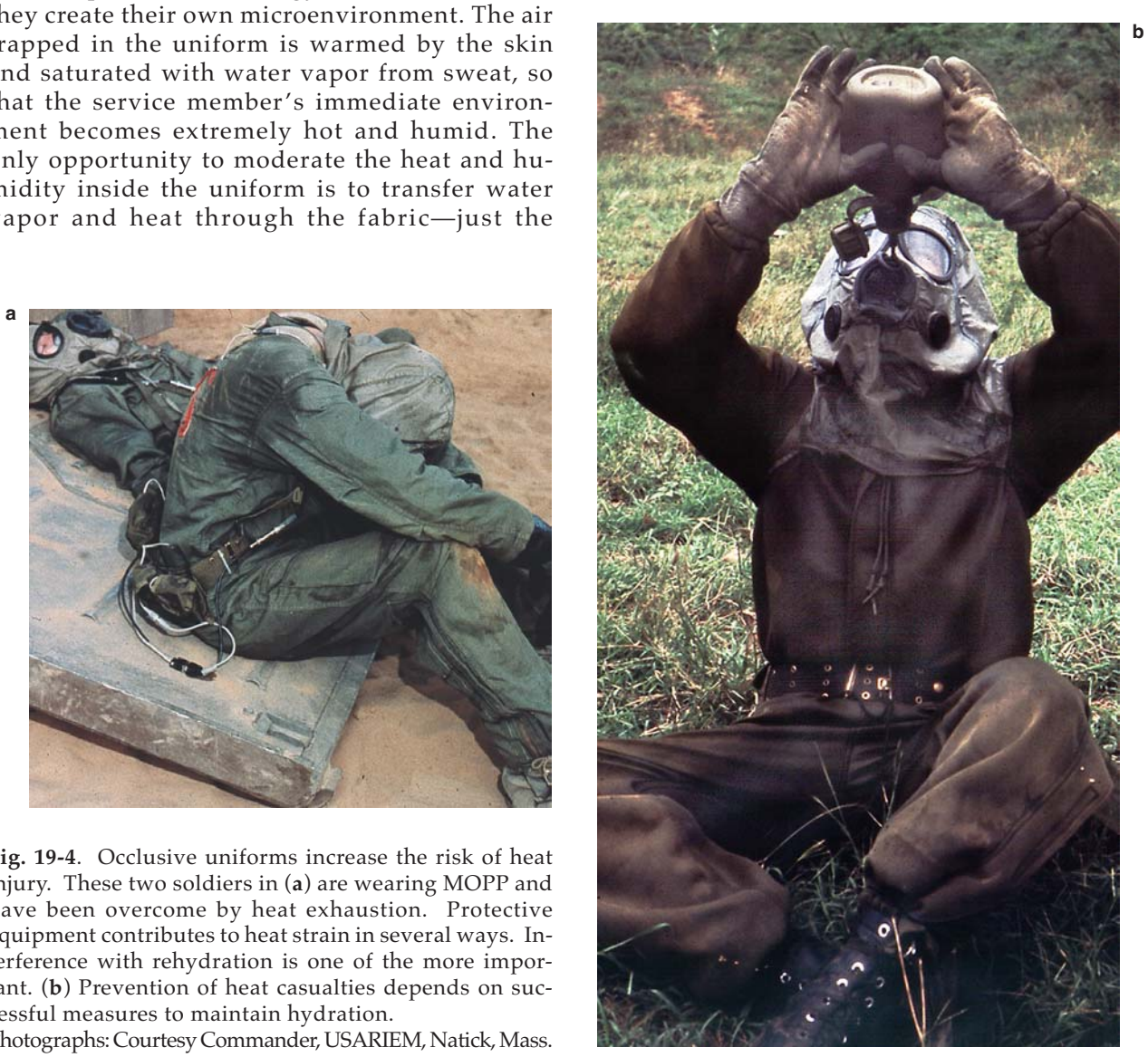

Fig. 19-4. Occlusive uniforms increase the risk of heat injury. These two soldiers in (**a**) are wearing MOPP and have been overcome by heat exhaustion. Protective equipment contributes to heat strain in several ways. Interference with rehydration is one of the more important. (**b**) Prevention of heat casualties depends on successful measures to maintain hydration.
Photographs: Courtesy Commander, USARIEM, Natick, Mass.

EXHIBIT 19-3

WORK INTENSITIES OF TYPICAL MILITARY TASKS

Work Intensity in MOPP* 0–1	Task	Work Intensity in MOPP* 2–4
VERY LIGHT	Sentry duty, Driving a truck Driving a truck	VERY LIGHT
LIGHT	Walking on hard surface (2.25 MPH, 1 m/s): no load Manual of Arms	LIGHT
MODERATE	Walking on hard surface (2.25 MPH, 1 m/s): 30 kg load Walking on loose sand (2.25 MPH, 1 m/s): no load Calisthenics Scouting patrol — Foxhole digging Crawling with full pack — Field assaults	MODERATE
HEAVY	Walking on hard surface (3.5 MPH, 1.5 m/s): 30 kg load Walking on hard surface (4.5 MPH, 2 m/s): no load Walking on loose sand (3.5 MPH, 1.5 m/s): no load	HEAVY

Adapted from: United States Army Research Institute of Environmental Medicine. *Heat Illness: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1991: 42. Technical Note 91-3.

cal estimates of metabolic rate (Exhibit 19-3). The heat stress associated with running is much higher than that associated with any other military task, typically two to three times the heat generation caused by moderate-to-heavy work. For this reason, work-rest cycles are not applicable to running.

Thermocompetence

Anything that impairs thermoregulatory capacity will reduce performance in the heat and increase the likelihood of heat illness. The most important factors include hydration, acclimatization, physical fitness, skin condition, and fever. Other factors that must be considered include prior heat illness, medications, nutriture, and state of rest. An individual is optimally capable to manage heat stress when he or she is fully hydrated, acclimatized, physically fit, healthy, well nourished, and well rested.

Hydration is essential to maintain blood volume for thermoregulatory blood flow and sweating. Both are reduced by dehydration, and the dehydrated service member has less ability to control body temperature in the heat. Water requirements are not reduced by any form of training or acclimatization. Exercises that attempt to teach personnel to work or fight with less water are fruitless and dangerous.

Service members who are required to deploy on short notice to hot environments will arrive unacclimatized. Adequate acclimatization will require several days to achieve. During the first few days of heat exposure, neither the ability to restrain the rise of core temperature during heat exposure nor the ability to conserve salt in sweat will be adequately developed. Aerobic fitness[17] provides the cardiovascular reserve to maintain the extra cardiac output required to sustain thermoregulation, muscular work, and vital organs in the face of heat stress. In addition, regular, strenuous aerobic physical training will provide a small degree of heat acclimatization.

Fever, whether due to immunization or illness, reduces thermoregulatory capacity by resetting the hypothalamus toward heat conservation rather than heat dissipation. This phenomenon eliminates the beneficial effect of acclimatization. Service members recovering from fever will have increased susceptibility to heat illness even after all clinical evidence of illness has disappeared. Until clearly able to manage normal work rates in the heat, they will require increased command supervision and moderated work schedules. Sunburn[27] and many other skin diseases[28–30]

reduce the ability of the skin to thermoregulate. Some medications will effect thermoregulatory adaptations and can increase the risk of heat illness. Prior heat illness also needs to be considered, as it is evidence of reduced heat tolerance in some individuals.

The requirements of military operations frequently mean lack of sleep and missed meals. Both these factors reduce thermoregulatory capacity and increase the risk of heat injury.[31]

Technology

Technology, whether sunshades, fans, air conditioning, or microclimate cooling systems, can be used to reduce heat exposure and to accelerate the removal of endogenous heat generated during exertion. This will allow service members to work longer in environments of high heat stress.

Implementation of a Heat Illness Prevention Program

The approach to the primary prevention of heat illness should include the consideration of ways to mitigate heat stress, to maximize the thermocompetence of the exposed, and to make the best use of available heat stress–control technology[32] (Exhibit 19-4). Primary prevention of heat illness is instituted by using the steps in the exhibit.

EXHIBIT 19-4

HEAT ILLNESS: IMPORTANT PREVENTION POINTS

- A heat casualty in a unit suggests others are at risk—all members of the unit should be evaluated immediately. Service members who are underperforming in the heat (eg, stragglers on a road march) may be incipient heat casualties. It is not safe to assume their underperformance is undermotivation.
- In military operations and training, risk factors for heat illness, all of which reduce thermoregulatory capacity, include:
  - -short-notice deployment (service members arrive unacclimatized)
  - -fever (from immunization or illness)
  - -dehydration
  - -fatigue
  - -undernutrition
- Acclimatization requires 7 to 10 days, regardless of physical condition. During the acclimatization period, service members who must work vigorously should be provided copious quantities of water and follow carefully supervised work-rest cycles.
- If rations are short or sweating is very heavy, salt supplementation may be needed. Acclimatization should eventually eliminate the need for salt supplementation.
- Service members in hot environments universally demonstrate dehydration of 1% to 2% of body weight. Command-directed drinking is effective in moderating dehydration and must be enforced. Unit leaders must reinforce hydration by planning for elimination as well as consumption.
- Fever reduces thermoregulatory capacity. Service members will have increased susceptibility to heat illness even after all clinical evidence of fever has disappeared. Until clearly able to manage normal work rates in the heat, service members will require increased command supervision and moderated work schedules.
- The requirements of military operations frequently produce lack of sleep, missed meals, and limited availability of water. All these reduce thermoregulatory capacity and increase the risk of heat illness.
- Reducing heat load reduces water requirements, so shade and night hours should be used as much as possible. Planning that ensures there will be enough water when and where needed must not be ignored.

Adapted from: United States Army Research Institute of Environmental Medicine. Medical Aspects of Cold Weather Operations: A Handbook for Medical Officers. Natick, Mass: USARIEM; 1991: 7, 39. Technical Note 91-3.

The service members who will be exposed must be assessed. Their acclimatization, physical fitness, and state of rest, nutrition, and hydration should be considered. Individuals or units at particular risk, such as recruits recovering from a febrile illness or units just beginning training, need to be identified.

The environmental conditions must be measured. Conditions can vary substantially even across short distances and in unpredictable ways. A shaded forest may seem to have less heat stress because of the lower solar load, but may, in fact, have a higher heat stress because of high humidity and lack of wind. Have the environmental conditions become more stressful recently? Sudden increases in environmental heat stress are particularly risky. Service members who have acclimatized to a moderate degree of heat stress will not be tolerant of sudden, more severe heat stress.

The workload must be assessed, especially the plans for work rate and duration. What uniform will be worn during training or while working? Will there be an opportunity to remove or loosen portions of the uniform? Unblousing trousers or removing jackets or helmets can reduce heat stress considerably but may not be possible.

Technological aids must also be considered. Will there be protection from the solar heat load? Although loosening clothing can permit better evaporative and conductive and convective cooling, the skin and head should be protected from direct sun by shade or light clothing.

Heat Stress Control

Heat stress exposure can be mitigated by reducing either the time or the intensity of exposure. The standard technique for regulating the time of exposure is the work-rest cycle. Other administrative controls that are used include the Threshold Limit Values (developed by the American Council of Governmental Industrial Hygienists),[26] Physiological Heat Exposure Limits (US Navy), and "Flag Conditions" used to regulate military training environments.

Tables of work-rest cycles provide explicit recommendations for the length of alternating periods of work and rest to allow work for an entire shift (Table 19-1). The work-rest tables for the civilian

TABLE 19-1

AN EXAMPLE OF A WORK-REST TABLE THAT GIVES MAXIMUM WORK TIMES IN MINUTES FOR DAYLIGHT OPERATIONS THAT CAN BE SUSTAINED WITHOUT EXCEEDING A GREATER THAN 5% RISK OF HEAT CASUALTIES

		MOPP0				MOPP4 + Underwear				MOPP4 + BDU			
WBGT	T	VL	L	M	H	VL	L	M	H	VL	L	M	H
78	82	NL	NL	NL	65	NL	177	50	33	NL	155	49	32
80	84	NL	NL	157	61	NL	142	49	32	NL	131	48	32
82	87	NL	NL	114	56	NL	115	47	31	NL	110	46	30
84	89	NL	NL	99	53	NL	104	45	30	NL	100	45	30
86	91	NL	NL	87	50	NL	95	44	29	NL	93	44	29
88	94	NL	NL	74	45	NL	85	42	28	NL	83	42	27
90	96	NL	NL	67	43	NL	79	41	27	NL	78	41	27
92	98	NL	NL	60	40	NL	75	40	26	NL	74	40	26
94	100	NL	193	55	37	NL	70	39	25	NL	70	39	25
96	103	NL	101	48	33	203	65	37	23	194	65	37	23
98	105	NL	82	44	31	141	62	36	22	140	62	36	22
100	107	261	70	41	28	118	59	35	21	118	59	35	21

MOPP: mission oriented protective posture
WBGT: wet bulb globe temperature (°F)
T: ambient temperature (°F)
VL: very light work intensity
L: light work intensity
M: moderate work intensity
H: heavy work intensity
BDU: battle dress uniform
NL: No Limitation

Source: United States Army Research Institute of Environmental Medicine. *Heat Illness: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1991: App E. Technical Note 91-3.

workplace are designed to strictly limit the rise in core temperature. They are more conservative than those available for military use, which are designed to limit heat casualties and are less concerned with specific physiological limits. Techniques that monitor physiological parameters of heat strain, such as temperature and pulse, can be used when work-rest cycles are not available or the exposures are particularly critical or demanding.

The intensity of heat exposure can be mitigated by controlling the rate of work. By slowing metabolic heat generation, the endogenous heat stress in reduced. Factors susceptible to this kind of control are march pace or cargo handling rates. Mechanical assistance (eg, moving by vehicle instead of on foot, using a forklift to move cargo) will also help control this factor.

Environmental heat load can be mitigated by changing the time of day when work is performed. Avoiding times of maximal solar load is one of the oldest techniques for controlling heat stress. Requirements to wear occlusive clothing and equipment should include consideration of the increased risk of heat illness.[33] Use of these items will require adjustment of work-rest guidance and will reduce the amount of work an individual can perform.

Maximizing Fitness for Exposure

In many military situations, the environment and the control technologies available will be dictated by circumstances, so the individual is the principal focus for measures to control heat stress and heat illness.

Some individuals should not be exposed to heat stress. These include those with significant histories of prior heat illness (discussed in more detail later), those with skin diseases that effect thermoregulation (eg, psoriasis, anhidrosis), those requiring medications that impair thermoregulation (eg, anticholinergics, diuretics), and those with illnesses that limit cardiovascular reserve. Sickle cell trait increases the risk of sudden death and exertional rhabdomyolysis during exercise heat stress.[34–37]

Some conditions will transiently limit thermoregulatory capacity. Any febrile response, whether to illness or vaccination, will substantially impair the ability to work in the heat. The duration of this effect is not known but almost certainly depends on a variety of parameters of the inflammatory response that caused the fever. Miliaria and sunburn both reduce the thermoregulatory capacity of the skin and so increase the risk of heat illness.

Hydration is the single most important factor in controlling heat stress and heat illness.[14,38] Training and operations produce many obstacles to maintaining adequate hydration. In hot environments, water losses can reach 15 L/d per individual, and service members do not drink enough water voluntarily to maintain hydration. This phenomenon has been called voluntary dehydration, although there is nothing willful about it. Thirst is not stimulated until plasma osmolarity rises 1% to 2% above the level customarily found in temperate climates. Consequently, if thirst is used as the guide for drinking, service members will maintain themselves at a level that is 1% to 2% dehydrated relative to their usual state. The only solutions to this problem are command-directed drinking and water discipline.

Even in the face of a clear understanding of the importance of water and hydration, other factors will interfere with maintaining hydration. Service members may decide that water drinking creates problems that outweigh its importance. For example, service members may not drink before going to sleep to avoid having to wake up and dress to urinate, or they may not drink before traveling in convoys if no rest stops are planned.

Adequate acclimatization is essential for optimal performance in the heat.[39] During the initial acclimatization period, service members must be provided copious quantities of water and carefully supervised to prevent excessive heat exposure. If possible, work tasks should be regulated using work-rest cycles tailored with the close involvement of unit medical personnel to the service member's physical capacity. Recent immunization, jet lag, and sleep loss, all of which reduce thermoregulation, will increase the risk of heat illness during this initial phase of exposure.

Sunburn must be prevented by adequate clothing, shade, and sunscreens. Skin diseases are best prevented by adequate hygiene. Commanders and logisticians must understand the importance of a functioning skin and provide adequate water for washing.

Salt depletion is a risk if service members are exposed during this time to sufficient heat or work stress to induce high sweating rates (more than several liters per day) if ration consumption is reduced. Salt depletion will contribute to heat exhaustion and heat cramps.[40] Salt supplementation may be necessary.[41]

Technology and Engineering Controls

Overhead shade and heat shields for outdoor workspaces will reduce radiant heat load, sunburn, contact burns, and heat illness. Air circulation by

any means facilitates convective cooling and may facilitate evaporative cooling by providing dry air. Air conditioning generates cool, dry air; when possible, it should be provided for indoor spaces to facilitate work and sleep quality. Ice vests and mechanical microclimate cooling systems[42,43] pump cool air or fluid inside occlusive uniforms to extend work times for individuals in high heat stress microenvironments.

Special Considerations: Minimizing Heat Casualties in Recruit Training

Recruits are particularly susceptible to heat illness during basic training in hot weather.[44] A number of reasons for their susceptibility are related to their rapid transition from civilian life to a demanding schedule of physical and military training. Most are neither acclimatized to heat on entry nor as physically fit as fully trained service members. They need to become fit in a short time and so quickly begin strenuous exercise. They also commonly suffer sleep loss and dehydration, and contagious febrile illnesses are common. Compounding their situation is their unfamiliarity with heat illness; they may not recognize early signs of heat illness or understand the importance of early treatment.

Heat illness can occur in any component of basic training. Certain activities, though, are associated with the highest risk: road marches, unit runs (including morning physical training), evening parades, and rifle range marksmanship training. Recruits on road marches and unit runs have very high sustained rates of endogenous heat production and muscular work. They usually develop temperature elevations and after 30 to 60 minutes, significant dehydration. Both temperature elevation and dehydration are aggravated if they begin their exercise dehydrated (eg, if they start just after waking without rehydrating), if they are wearing a heavy uniform that prevents loss of heat to the environment (eg, chemical protective equipment), or if environmental conditions retard heat loss. The combined elevated temperature, muscular work, and dehydration lead to a high risk of heat exhaustion and heat stroke. Heat casualties at evening parades usually result from dehydration developed during a day of vigorous physical training.

Most would not ordinarily associate a significant risk of heat casualties with rifle marksmanship training. The association exists, though, because rifle range training is often done during extreme heat that prohibits other outdoor training. Recruits are exposed for long periods to intense solar and ground-contact heat loads without consideration of the heat-induced water requirement. Under these conditions, recruits develop hyperthermia and dehydration.

Education and Training

The medical officer has an educational role as a unit prepares for operations in hot environments. Service members of every rank must know the steps they can take to minimize the risk of heat illness. They must understand the importance of hydration, nutrition, and skin hygiene. They must know that although thirst means dehydration, dehydration does not necessarily provoke thirst. They must be trained to recognize the signs of heat illness and the basics of buddy aid. Staff must understand the critical importance of water to the unit so they can incorporate adequate water logistics and management into their plans, which must not add impediments to water discipline. Planners must incorporate the degrading effect of heat into their operational schedules by adding rest and hydration stops. Leaders must understand the nature and the magnitude of the threat that heat stress presents to their units so they can emphasize the importance of required countermeasures. Small-unit leaders must know the techniques for managing work in the heat and understand the guidelines for water replacement and work-rest cycles.

Surveillance

Although heat stress modeling is well developed[45,46] and can predict with great accuracy the effects of exercise-heat exposure, the details of living and working in hot environments confound the ability of these models to describe the risks and responses of groups of people through time. Consequently, successful prevention depends on the detection of early evidence of accumulating heat strain. Some of this evidence is seen in day-to-day activities and appears in phenomena such as reduced appetite or physical vigor or deeply colored urine. Some manifests as minor medical complaints such as gastrointestinal disturbances and minor heat illnesses. These all presage both impaired performance and increased incidence of heat illness and dictate intervention.

Specific guidance on work-rest cycles or water requirements depends on assumptions about the population to which it will be applied. These assumptions usually include such factors as age, hydration state, physical fitness, and nutriture. If these

assumptions are violated, which is to be expected in many deployments, the guidance may underestimate heat exposure risks. Consequently, experience and formal surveillance of exposures and outcomes are required to develop guidance appropriate to the actual circumstances of the deployment.

Secondary Prevention

Heat strain and heat illness will worsen if not recognized and managed early. The key to secondary prevention, then, is early recognition followed by extrication, cooling, rehydration, and time for recuperation.

HEAT ILLNESSES

Heat illness can be separated into five categories. The first and largest is exertional heat illness, which is itself divided into heat exhaustion, exertional heat injury, and exertional heat stroke. Classic heat stroke, exertional rhabdomyolysis, exertional hyponatremia, and minor heat illnesses are the other categories. Exhibit 19-5 summarizes the salient clinical features of the more serious heat illnesses.

General Considerations in Management and Diagnosis of Heat Illness

There are three important clinical principles that apply to the diagnosis and management of acute illnesses occurring in the heat. First, there are no pathognomonic signs or symptoms of heat illness. Second—and as a consequence of the first—there

EXHIBIT 19-5

MILITARILY IMPORTANT HEAT ILLNESSES

Heat Exhaustion
- Occurs during exercise
- Headache, GI symptoms, exhaustion, collapse, syncope
- Rapid recovery with rest and hydration
- Peak CK < 1000
- No abnormal LFT
- No myoglobinuria

Exertional Heat Injury
- Occurs during exercise
- Headache, GI symptoms, exhaustion, collapse, syncope, muscle pain
- Rapid recovery with rest and rehydration except muscle pain
- Peak CK > 1000
- Creatinine: day 2 > day 1
- LFT up to 3 x ULN
- No encephalopathy or coagulopathy

Exertional Heat Stroke
- Occurs during exercise, often early
- May be critically ill from onset
- Encephalopathy
- coagulopathy common
- CK > 5000
- Peak creatinine > 2.0
- LFT > 3x ULN

Exertional Rhabdomyolysis
- Muscle pain after exertion
- CK > 10,000
- Myoglobinuria
- Peak creatinine > 2.0
- No encephalopathy or coagulopathy
- LFT < 3x ULN

Exercise-related Hyponatremia
- Gradual onset
- Symptoms late in the day
- Marked thirst
- Hyponatremia
- No significant change in LFT, renal function, CK, or hemostasis

Dehydration
- After heat exposure
- Headache, nausea, fatigue, constipation
- Heat intolerance, mild orthostasis common
- Mild hemoconcentration
- Concentrated urine

Abbreviations
CK: creatine phosphokinase
GI: gastrointestinal
LFT: liver function test
ULN: upper limit of normal

is always a differential diagnosis. Third, shade and cooling are always appropriate emergency responses to acute illness in the heat.[47]

Management

The initial management of acute illness in the heat should include putting the patient in the supine position; establishing shade and skin cooling; evaluating airway, breathing, and circulation; examining mental status; and measuring temperature. Since the illness may be life threatening (eg, heat stroke) but is hard to diagnose in the field, a rapid decision on disposition should be made.

Diagnosis

The differential diagnosis of acute illness in the heat includes infection (particularly meningococcemia and *P falciparum* malaria), pontine or hypothalamic hemorrhage, drug intoxication (eg, cocaine, amphetamines, phencyclidine, theophylline, tricyclic antidepressants), alcohol or sedative withdrawal, severe hypertonic dehydration, and thyroid storm. Specific questions should be asked about recent immunizations or illnesses and medications taken, including nonprescription medications.[48,49]

The early symptoms of exertional heat illness include fatigue, irritability, headache, and anorexia. Paresthesias and carpopedal spasm can occur in severe heat exposure. As the illness progresses, nausea, vomiting, and, occasionally, diarrhea can develop. Slumping posture and ataxia are signs of impending collapse. Sweating and hyperthermia are characteristic. If the illness progresses beyond its premonitory symptoms and signs, it presents as acute collapse, often with syncope and seizures. Persistence of seizures, delirium, disorientation, or combativeness is presumptive evidence of heat stroke.[47,50]

The most important question to answer in the approach to acute illness in the heat is "Is this heat stroke?" because of the seriousness of that condition. The mental status examination is the key to this determination. Any significant impairment in mental status at the scene of illness is evidence of heat stroke. The mental status examination should be performed quickly but carefully and should evaluate arousal, orientation, interaction, cognition, and memory. Any impairment beyond transient drowsiness and inattention is significant. Other clinical features that may be helpful in the initial field evaluation include exertional heat stroke's frequent presentation as a sudden, severe illness soon after beginning exercise-heat exposure. High temperature immediately after onset of illness is helpful in narrowing the differential diagnosis but is not diagnostic, as core temperatures in excess of 40°C (105°F) are routinely encountered during vigorous exercise.

While high temperature is not itself diagnostic, accurate and early measurement of body temperature is essential to the diagnosis of heat illness[51]: first, to determine if temperature is elevated and second, to monitor the response to cooling. Core temperature can be effectively measured in any deep body space; the rectum and esophagus are the usual sites.[52] Under field conditions, tympanic temperature is not an accurate reflection of core temperature[53,54] because, among other reasons, it is significantly influenced by the skin and tissue temperature of the neck.

Laboratory evaluation should be directed by the differential diagnosis appropriate for the clinical circumstances and those studies needed to monitor therapy and clinical state. Initial studies for all heat illness should include a complete blood count and measurement of electrolytes, blood urea nitrogen, and creatinine. Individuals suspected of having exertional heat injury or heat stroke should also have baseline studies that include liver function, clotting factors, creatine phosphokinase, myoglobin, calcium, and phosphorus.[55] Patients with heat stroke require serial monitoring of platelets and plasma clotting factors, renal and hepatic function, and electrolyte and acid-base status.

Recurrent heat illness is an indication for a formal evaluation for cystic fibrosis.[56,57] Heat stroke or rhabdomyolysis, particularly if recurrent, is an indication for muscle biopsy and evaluation for primary myopathy.[58–61]

Any patient in whom the diagnosis of heat stroke is possible will need at least 72 hours to complete an adequate period of observation, rest, and rehydration at a second- or third-echelon medical treatment facility. Patients who are clinically well but still being observed can be assigned supervised light duty at the treatment facility if shade and water are plentiful. Under no circumstances should they be reexposed to significant heat stress during this period.

Pathophysiology of Exertional Heat Illness

Exertional heat illness includes three disorders—heat exhaustion, heat injury, and heat stroke—which have in common that they occur acutely during exertion in the heat and are associated with substantial rises in core temperature. Although they are often thought of as degrees of heat illness along a single pathophysiological spectrum, it is not at all clear that they

share a common pathophysiology. The clinical effects caused by exertional heat illness range from functional impairment in heat exhaustion to life-threatening organ injury in exertional heat stroke.

The pathophysiological mechanisms of heat exhaustion include an inadequate capacity to maintain cardiac output sufficient to sustain the demands of thermoregulation (ie, skin blood flow), muscular activity, and the viscera.[62,63] Dehydration, high core and skin temperature, and vigorous activity all combine to determine the point at which heat exhaustion occurs. The limitation of blood flow to mesenteric viscera is probably responsible for the gastrointestinal symptoms of heat exhaustion. The limitation of muscular blood flow contributes to the physical collapse. When demands for blood flow are extremely high, the expansion of the vascular bed can lower blood pressure to the point of syncope. Although all the exertional heat illnesses occur in the setting of exercise-heat exposure, it is not clear that heat exhaustion leads to more serious exertional heat illness.

The pathogenesis of exertional heat injury and exertional heat stroke is unclear. In both conditions measurable tissue injury occurs. In exertional heat injury, the tissue primarily affected is muscle, although there is usually evidence of mild injury to liver and kidney tissue. Exertional heat stroke, in contrast, is characterized by serious injury to multiple organs systems, including the central nervous system, and to clotting mechanisms. A variety of hypotheses have been proposed for the mechanisms of the tissue and organ damage of these two more serious forms of exertional heat illness. These include gastrointestinal endotoxin release from mesenteric vasoconstriction,[64–66] cellular energy depletion,[67] potassium depletion,[68] direct effects of hyperthermia,[69] and primary myopathies.[58,59] Dehydration is not as important a component in these two conditions as it is in heat exhaustion. Other factors that contribute to the pathogenesis of exertional heat illness include skin disease, medications that influence sweating or skin blood flow,[49] and the effects of inflammation on central thermoregulation.[70]

Heat Exhaustion

Heat exhaustion is the most commonly encountered form of heat illness.[3] Heat exhaustion, by definition, is a "functional" illness and is not associated with evidence of organ damage. Classically, heat exhaustion has been divided into salt-depletion heat exhaustion and water-depletion heat exhaustion.

Salt depletion in hot environments develops from increased salt loss in sweat (particularly among the unacclimatized) and reduced salt intake due to anorexia. Salt depletion develops over several days, so the contraction of extracellular fluid is gradual and symptoms develop slowly. The reduced extracellular fluid volume produces symptoms of fatigue and orthostatic dizziness. Because salt depletion does not produce intracellular hypertonicity, thirst is not prominent until the extracellular fluid volume has contracted enough to cause volumetric stimulation of thirst. Nausea and vomiting are common but of unknown mechanism. Hemoconcentration occurs due to the contraction of extracellular fluid. Muscle cramps are a common accompaniment of salt depletion (see "Heat Cramps" below). Potassium depletion commonly accompanies salt depletion due to diminished intake and mineralocorticoid-driven kaliuresis. Frank hypokalemia is uncommon.

Water depletion in hot environments develops from sweat rates sufficiently in excess of water replacement rates to produce hypertonic dehydration. Even though the loss of water occurs from both intracellular and extracellular compartments, the rate of dehydration is usually quite rapid and symptoms evolve quickly. Thirst is prominent and is caused by the hypertonicity. Oliguria, clinical dehydration, tachycardia, and tachypnea with symptomatic hyperventilation are all prominent clinical features.

In practice, neither heat exhaustion is encountered in a "pure" form; rather, classic heat exhaustion always includes elements of both water and electrolyte depletion. Rest, cooling, and adequate rehydration with hypotonic saline solutions are common elements of the therapy of both forms of heat exhaustion.

The management of heat exhaustion is directed to correcting the two pathogenic components of the illness: excessive cardiovascular demand and water and electrolyte depletion.[47,71] The load on the heart is reduced by rest and cooling. Water and electrolyte depletion is corrected by administering oral or parenteral fluids. Heat-exhausted patients do not require active cooling measures; removal of heavy clothing and rest in a shaded and ventilated space provides an adequate opportunity for spontaneous cooling. If available, cool water can be used to cool the skin. The consequent cutaneous vasoconstriction will rapidly reduce circulatory demand and improve venous return. Intravenous fluids replenish the extracellular volume quickly. Oral fluids suffice for those patients who can take fluids without risk of vomiting. However, clinical observation suggests parenteral fluids produce more rapid recovery than oral fluids, probably because oral fluids are absorbed more slowly.

Patients with heat exhaustion experience rapid clinical recovery[72] but need at least 24 hours of rest and rehydration under first-echelon or unit-level medical supervision to reverse their water and electrolyte depletion. A single episode of heat exhaustion does not imply any future predisposition to heat injury. An attempt should be made to determine the reason for the heat exhaustion (eg, insufficient work-rest or water discipline, coincident illness or medication). Repeated episodes of heat exhaustion require thorough evaluation.

Exertional Heat Injury

Exertional heat injury is an exertional heat illness that causes significant tissue damage from exercise heat exposure but does not develop into encephalopathy or organ failure. Exertional heat injury can present acutely as collapse during exercise, but some cases present many hours after exercise with prominent muscle pain and marked elevations in creatine phosphokinase and mild evidence of liver and kidney injury. Lack of conditioning and acclimatization appear to be risk factors for this type of exertional heat illness but do not account for all the cases.

Diagnosis is based on the evidence of tissue injury after exercise heat exposure in the absence of progression to heat stroke. Clinical recovery requires rest and restriction from further exercise in the heat. Recovery, defined as the resolution of pain and laboratory abnormalities, takes 7 to 10 days. The risk of recurrence is not known.

Exertional Heat Stroke

Exertional heat stroke is distinguished from exertional heat injury by the degree of organ injury and the appearance of encephalopathy and coagulopathy.[47,73–75] The degree of injury appears to relate to both the degree of temperature elevation and duration of exposure. Five organ systems (ie, the central nervous system, the hemostatic system, the liver, the kidneys, and muscle) are the principal foci of injury in exertional heat stroke. Encephalopathy is the sine qua non of heat stroke. Its presentation ranges from syncope and confusion to seizures or coma with decerebrate rigidity. Disseminated intravascular coagulation is common.[76] The principal causes of disseminated intravascular coagulation seem to be thermal damage to endothelium,[77] rhabdomyolysis, and direct thermal platelet activation causing intravascular microthrombi. Fibrinolysis is secondarily activated. Hepatic dysfunction and thermal injury to megakaryocytes slows the repletion of clotting factors. Hepatic injury is common and may progress to frank hepatic failure. Renal failure following heat stroke can be caused by several factors, including myoglobinuria from rhabdomyolysis, acute tubular necrosis due to hypoperfusion, glomerulopathy due to disseminated intravascular coagulation, direct thermal injury, and hyperuricemia. Rhabdomyolysis is a frequent complication of exertional heat stroke. Acute muscular necrosis releases large quantities of potassium, myoglobin, phosphate, and uric acid and sequesters calcium in the exposed contractile proteins. Adult respiratory distress syndrome complicates heat stroke occasionally and is associated with a high rate of mortality.[78]

The clinical outcome of patients with heat stroke is primarily a function of the magnitude and duration of temperature elevation. Mortality is rare in settings prepared to treat heat casualties with immediate cooling. Therefore, the most important therapeutic measure is rapid reduction of body temperature.[79] Any effective means of cooling is acceptable. While many techniques have been used, none has been unequivocally demonstrated to be superior. Immersion in cool or iced water with skin massage is a classic technique for cooling heat stroke patients. Ice water produces the most rapid cooling,[80] but cool water is less demanding logistically and less uncomfortable for the medical attendants. In hot, dry environments, field-expedient immersion baths that will keep water cool can be constructed by digging pits in the shade and lining them with plastic or by rigging shallow canvas tubs in well-ventilated, elevated frames (Figure 19-5).

Although not as effective at cooling as immersion, wetting the body surface and accelerating evaporation by fanning can also work.[81] The water can be applied by spraying or by application of thin conforming cloth wraps (eg, sheets, cotton underwear). Cooling blankets will also lower body temperature but are unlikely to be available in the field. Although cooling blankets have the advantage of maintaining a dry working environment, their limited contact surface provides slower cooling than

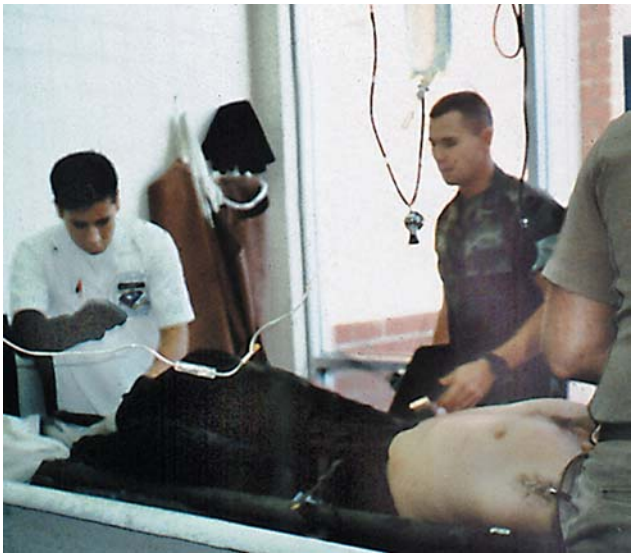

Fig. 19-5. The area for treatment of heat casualties at the USMC Training Base at Parris Island, SC. The tub is filled with ice. The casualty is placed on a litter above the ice and towels cooled in the ice water are placed on the body surface. Cooling comparable to immersion is achieved while preserving access to the torso and extremities for monitoring and emergency medical measures.
Photograph: Courtesy of Colonel John Gardner, Medical Corps, US Army, Uniformed Services University of the Health Sciences, Bethesda, Md.

immersion or surface-wetting techniques. Invasive cooling techniques have been tried, including ice water lavage or enemas and peritoneal lavage with cool fluids. These techniques do not provide faster cooling and have the additional disadvantages of potential complications and inappropriate fluid loads.

After cooling and hemodynamic stabilization, continuing care is supportive and is directed at the complications of heat stroke as they appear. Patients with heat stroke frequently have impaired temperature regulation for several days, with alternate periods of hyperthermia and hypothermia. Prognosis is worse in patients with more severe degrees of encephalopathy. Permanent neurological sequelae can develop after heat stroke, including cerebellar ataxia, paresis, seizure disorder, and cognitive dysfunction.[82,83]

Patients with heat stroke will require prolonged convalescence.[84] Heat stroke has been considered evidence for constitutional heat intolerance, but a recent study[85] demonstrates measurable heat intolerance in only 1 of 10 individuals after recovery from heat stroke. That same study also demonstrates that full heat tolerance was not achieved for up to a year even in those with eventual full recovery of thermoregulation.

Classic Heat Stroke

Classic heat stroke occurs in individuals, frequently those with impaired thermoregulation due to illness or medication, exposed passively to heat and dehydration.[86–88] It is principally an episodic affliction of young children in confined spaces, such as automobiles in the sun,[89] or an epidemic affliction in the elderly during urban heat waves.[90] Classic heat stroke differs in several ways from exertional heat stroke. Classic heat stroke evolves slowly, usually over a few days of continuous heat exposure. (Children in cars are exposed to much higher temperatures and are injured much more quickly.) Dehydration is a prominent feature. There is no exertional component, so rhabdomyolysis is less common, which reduces the likelihood of renal failure.[91] Mortality rates tend to be high because the patients often are alone and unable to summon help as the illness develops.

Heat Cramps

The specific pathophysiological mechanism of heat cramps is not known.[92–94] Heat cramps typically occur in salt-depleted individuals during a period of recovery after working in the heat[95] but are also a common component of salt-depletion heat exhaustion. Salt depletion is thought to be associated with muscle contraction of heat cramps.[40] Supporting that hypothesis is the efficacy of sodium chloride in treating heat cramps and the reduction of heat cramp incidence after salt supplementation in industrial populations.[96]

Patients with heat cramps present with extremely painful tonic contractions of skeletal muscle.[47,50,95] The cramp in an individual muscle is usually preceded by palpable or visible fasciculation that lasts 2 to 3 minutes. Cramps are recurrent and may be precipitated by manipulation of muscle. The cramps involve the voluntary muscles of the trunk and extremities. Smooth muscle, cardiac muscle, the diaphragm, and bulbar muscles are not involved. In individuals with only heat cramps, there are no systemic manifestations except those attributable to pain. The cramps can begin during work or many hours after work.

The diagnosis of heat cramps is usually straightforward.[95] The differential diagnosis includes tetany due to alkalosis (eg, hyperventilation, severe gastroenteritis, cholera) or hypocalcemia, strychnine poisoning, black widow spider envenomation, or abdominal colic. These entities should be distinguishable on clinical examination. Replenishment of salt orally or parenterally resolves heat cramps rapidly. The response to therapy is sufficiently dramatic to be valuable in the differential diagnosis. The route of administration should be determined by the urgency of symptom relief.

Patients with heat cramps usually have substantial salt deficits (15–30 g, 2–3 days of usual dietary intake). These individuals should be allowed 2 to 3 days to replenish salt and water deficits before resuming work in the heat. No significant complications have been reported from heat cramps except muscle soreness. An episode of heat cramps does not imply any predisposition to heat injury. As with any heat illness, an attempt should be made to determine the reason for the episode so that appropriate advice can be given to the service member and the chain of command to avoid future episodes.

Prevention of heat cramps depends on the recognition of populations at risk and intervention to assure adequate water and salt intake. Sudden changes in weather or sudden exposure to unaccustomed work in the heat combined with inadequate intake of salt in the diet will produce in 2 to 3 days the salt depletion required for heat cramps. Military rations contain sufficient salt to maintain adequate body stores if they are fully consumed. In situations where rations are unavailable or not being consumed completely, salt supplementation from snack foods or salt solutions is appropriate. Environmental controls to reduce heat stress will also reduce salt depletion and reduce the incidence of heat cramps.

Exertional Rhabdomyolysis

Rhabdomyolysis that occurs as a result of exercise-heat exposure but without any other characteristics of exertional heat illness (eg, encephalopathy, hepatic injury) is considered exertional rhabdomyolysis.[47,97] It usually develops in a setting of heavy work with significant

muscular loads.[98,99] Its specific pathophysiological mechanism is not known, but high muscle temperature and relative ischemia probably contribute. Some individuals with exertional rhabdomyolysis may have an underlying metabolic myopathy, which only manifests under extreme circumstances.[61,100]

Exertional rhabdomyolysis presents as collapse with marked muscle pain and tenderness during exercise heat exposure.[101,102] Systemic symptoms and signs, except the usual accompaniments of work in the heat, are not prominent initially.[98] As myonecrosis proceeds, however, acute renal failure, metabolic acidosis, and hypocalcemia develop. The diagnosis is established by myoglobinuria, marked elevation of creatine phosphokinase concentrations in blood, and renal injury in the absence of evidence of significant injury to other tissues or organs. Treatment is directed to minimizing renal injury and managing the acute metabolic consequences of the myonecrosis.

Although the circumstances under which exertional rhabdomyolysis develops are well known, it is an uncommon, sporadic illness. Although data are lacking about whether those who have had an episode of exertional rhabdomyolysis are susceptible to it again or to other heat illnesses, prior episodes should preclude future exposure. Prevention will depend on recognizing the circumstances in which it occurs, avoiding extreme muscular loading, and ensuring opportunities for rest and recovery during very strenuous work.

Exertional Hyponatremia

Exertional hyponatremia is a recently recognized heat illness.[103–107] It is a form of water intoxication, which produces dilutional hyponatremia.[107,108] It was originally recognized in elite athletes specializing in long-distance events but is found commonly among military training populations in hot climates. Since the illness requires sufficient water intake to cause hyponatremia, it is possible its recent appearance is related to increased water consumption during exercise-heat exposure in an effort to prevent dehydration and heat illness. Most cases of symptomatic exertional hyponatremia are sporadic, but at least one epidemic has occurred in a military unit required to drink too much water during training.[109]

The manifestations of exertional hyponatremia are thirst, fatigue, and anorexia. The illness develops over a number of hours. The symptoms usually limit work capacity, so hyperthermia is not a common clinical sign. If the water consumption continues, hyponatremia can progress beyond mild symptoms to frank seizures and rhabdomyolysis.[110–112] Hyponatremia responds to the usual measures for water intoxication, including water restriction and seizure control. Exertional hyponatremia resolves quickly, usually with full recovery.[107] The risk of recurrence is not known.

The prevention of exertional hyponatremia depends on the recognition that excessive water consumption can be as dangerous as inadequate water consumption. Guidance about water consumption should provide both a minimum and a maximum amount.[104,113–115]

Minor Heat Illnesses

Miliaria Rubra, Miliaria Profunda, and Anhidrotic Heat Exhaustion

Miliaria rubra is a subacute pruritic, inflamed, papulovesicular skin eruption that appears in actively sweating skin exposed to high humidity.[116] In dry climates, miliaria is confined to skin sufficiently occluded by clothing to produce local high humidity[117] (Figure 19-6). Each miliarial papulovesicle represents a sweat gland whose duct is occluded at the level of the epidermal stratum granulosum by inspissated organic debris.[118,119] Sweat accumulates in the glandular portion of the gland and infiltrates into the surrounding dermis. Pruritus increases with increased sweating. Miliarial skin cannot fully participate in thermoregulation, and therefore the risk of heat illness is increased in proportion to the amount of skin surface involved.[28,29,120,121] Sleeplessness due to pruritus and secondary infection of occluded glands have systemic effects that further degrade optimal thermoregulation.[122]

Miliaria is treated by cooling and drying affected skin, avoiding conditions that induce sweating, controlling infection, and relieving pruritus. Eccrine gland function recovers when the affected epidermis desquamates, which takes 1 to 3 weeks.

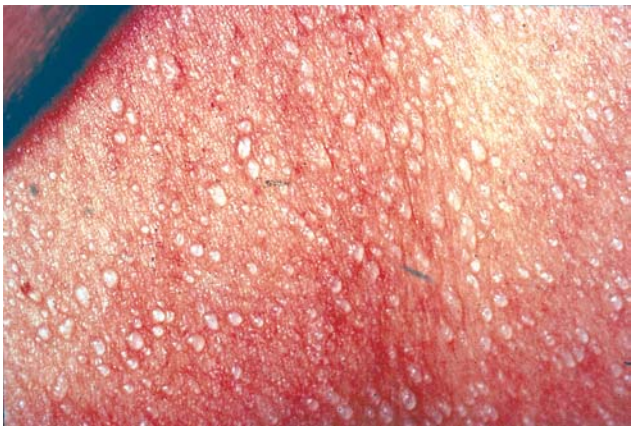

Fig. 19-6. In cases of miliaria crystaluna, sweat is trapped in the ducts of the sweat glands by inspissated material. The trapped sweat produces the appearance of small vesicles on the skin, such as in this photograph.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

Miliaria that becomes generalized and prolonged (miliaria profunda) can cause an uncommon but disabling disorder: anhidrotic heat exhaustion, which is also known as tropical anhidrotic asthenia. The lesions of miliaria profunda are presumed to develop from pre-existing miliaria rubra lesions by a superimposed inflammatory obstruction of the eccrine duct. The lesions are truncal, noninflamed, and papular, with less evidence of vesiculation than the lesions of miliaria rubra. They may only be evident during active sweat production. Sweat does not appear on the surface of affected skin. The lesions are asymptomatic, which may explain why the patient does not seek medical evaluation early in the course.

Miliaria profunda causes a marked inhibition of thermoregulatory sweating and heat intolerance similar to that of ectodermal dysplasia. Symptoms of heat exhaustion and high risk of heat stroke occur under conditions well tolerated by other individuals. Management of miliaria profunda requires evacuation to a cooler environment for several weeks to allow restoration of normal function of the eccrine glands.

Heat Syncope

Syncope occurring while standing in a hot environment has been called heat syncope, but it is probably not a discrete clinical entity. Rather, thermal stress increases the risk of classic neurally mediated (vasovagal) syncope by aggravating peripheral pooling of blood in dilated cutaneous vessels.[47,123] No special heat-related significance should be assigned to syncope occurring in these circumstances. Clinical evaluation and management should be directed toward the syncopal episode, not potential heat illness. However, syncope occurring during or after work in the heat or after more than 5 days of heat exposure should be considered evidence of heat exhaustion.

Heat Edema

Mild dependent edema ("deck legs") is occasionally seen during the early stages of heat exposure while plasma volume is expanding to compensate for the increased need for thermoregulatory blood flow. In the absence of other disease, the condition is of no clinical significance and will resolve spontaneously. Diuretic therapy is not appropriate and may increase the risk of heat illness.

Heat Tetany

Heat tetany is a rare condition, which occurs in individuals acutely exposed to overwhelming heat stress.[124] Extremely severe heat stress induces hyperventilation, which appears to be the principal pathophysiological process. The manifestations of heat tetany are characteristic of hyperventilation. They include respiratory alkalosis, carpopedal spasm, and syncope. Management requires removal from the heat and control of hyperventilation. Dehydration and salt depletion are not prominent features.

Chronic Dehydration

Chronic dehydration,[125] also known as voluntary dehydration, is associated with several disabling conditions, including nephrolithiasis,[126] hemorrhoids, fecal impaction, and urinary tract infection. Prevention requires the establishment of water consumption targets and command enforcement of those targets.

COLD ENVIRONMENTS

Cold and Military Campaigns

Casualties from cold exposure occur in all types of operations. Cold can be an effective offensive weapon. Forces including the US Army, the Russians, and the Finns have used cold in this way by displacing their opponent from shelter and allowing the environment itself to force surrender. Rapidly paced operations, though, can outrun supply trains and expose leading elements to unexpected cold weather bivouacs and risk of cold injury. It should not be surprising that the US military has suffered cold weather casualties in almost all of its conflicts, from the American Revolution to the Korean War.[127,128] Cold injuries remain a problem in military operations and training exercises today.[129–136]

Physiological Effects of Cold Exposure

Humans have evolved two physiologic mechanisms to maintain core temperature during cold exposure: (1) reducing skin temperature, which reduces the differential between the skin temperature and the environment and slows heat loss and (2) increasing heat production by shivering.[137]

When the body is exposed to cold, blood is diverted away from the skin and extremities to the trunk by vasoconstriction. Consequently, a layer of relatively

hypoperfused tissue is formed between the environment and the viscera. Deprived of the heat from the metabolically active core, this "shell" of tissue cools, thereby reducing the gradients for heat loss from the skin surface by radiation, conduction, and evaporation. This tissue insulation has been estimated to be about the same as that provided by wearing a wool business suit. In contrast, heavy arctic clothing provides six to eight times as much insulation.

Cold sensory receptors in the skin respond to both absolute temperature and the rate of temperature change. Consequently, sudden exposure to cold, such as walking from a warm building into a cold wind, will trigger an acute response with vasoconstriction and even transient shivering. As cold exposure continues, the skin equilibrates at the new colder temperature. As skin temperature stops changing, the response to the cold stimulus moderates and the acute shivering passes. The skin gradually (over a period of 2 to 3 hours) accommodates to the cold, and the sensation of cold becomes less uncomfortable. Conversely, if cold skin is warmed, the reflex response will reduce the insulating vasoconstrictive response, increase heat loss from skin and extremities, and inhibit shivering. This may be the explanation for Baron Larrey's observation during Napoleon's retreat from Moscow during the winter of 1812 that people near campfires were more likely to die during sleep than those sleeping further away.[138] As a consequence of reducing blood flow and volume in skin and extremities, peripheral vasoconstriction causes an expansion in central blood volume that can induce diuresis and dehydration.

If the insulating effect of vasoconstriction is insufficient to protect the core temperature, the continuing fall in temperature triggers the onset of heat production by muscle. Initially, muscle tone increases, which increases metabolic rate 2-fold. However, if core temperature falls further, the muscular activity changes to cycles of contraction and relaxation, producing visible shivering.[139] Maximal shivering increases heat production up to seven times the resting level. It is essential to remember that a fall in core temperature has already occurred when sustained shivering appears.

Although vasoconstriction is beneficial and protects core temperature because it reduces the flow of blood from the core to the periphery, it places the metabolically inactive acral regions of the body at risk of severe cooling and injury. Cold-induced vasodilation (CIVD) is a physiological mechanism that appears to reduce the risk of injury when the hands or feet are exposed to water below 10°C (50°F) and air below 0°C (32°F). As the hands or feet cool, vasoconstriction initially reduces blood flow and volume. After some minutes of low digital temperature, arteriovenous anastomoses in the distal phalanx open and allow a rapid increase in digital blood flow by bypassing the constricted precapillary arterioles.[140] While the anastomoses remain open, the digits remain warm. The phenomenon is usually cyclic, producing alternating periods of vasoconstriction and vasodilation 10 to 20 minutes long. Individuals vary in the magnitude of their CIVD response.[141] The magnitude and duration of the CIVD depend on core temperature. When core temperature is low, the phenomenon is substantially less.

Physiological adaptation to cold is not a phenomenon of the same significance as adaptation to heat or high terrestrial altitude.[142] Indeed, the successful use of clothing and shelter in cold environments prevents much of the cold stress that might induce adaptation or habituation. Consequently, there appear to be no practical means of significantly enhancing physiological cold tolerance through training or predeployment exercises. The principal mechanisms through which cold tolerance develops are familiarization and habituation (Exhibit 19-6).

EXHIBIT 19-6

IMPORTANT PHYSIOLOGICAL POINTS OF COLD EXPOSURE

- Humans cannot sense core temperature.
- Skin is sensitive to cold and will be painful until it cools to 10°C (50°F).
- Skin accommodates to cold; exposure reduces the sensation of both cold and pain.
- Skin is numb below 10°C. The disappearance of pain in an extremity during cold exposure may indicate serious cold injury. Immediate visual inspection for frostbite is mandatory.
- Cold exposure causes diuresis, which aggravates the routine dehydration in field settings.
- Extremities below 10°C are paralyzed.
- The most important adaptation to cold is proper training and equipment.

Adapted from: United States Army Research Institute of Environmental Medicine. *Medical Aspects of Cold Weather Operations: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1993: 45. Technical Note 93-4.

PREVENTION OF ILLNESS AND INJURY IN THE COLD

It is useful to analyze the risk of environmental cold injury as an interaction of three components: the environmental stress, the thermocompetence of the service member, and the protective technology available. This is an equation that shows this interaction:

$$(2)\ \text{Strain} = f\frac{\text{Time} \bullet \text{Cold Stress}}{\text{Thermocompetence} \bullet \text{Technology}}$$

Environmental Cold Stress

Cold land environments are generally classified as either wet-cold or dry-cold. Wet-cold environments have ambient temperatures from above freezing to about 18°C (65°F), with wetness ranging from fog to heavy rain. They are associated with nonfreezing peripheral cold injuries, such as trench foot. Usually, many hours to days of exposure are required to cause injury. Dry-cold environments have ambient temperatures below freezing (0°C, 32°F). Precipitation, if present, is in the form of snow. Dry-cold environments are associated with freezing peripheral injuries, which can develop in a few minutes to hours.

Certain exposures carry a high risk of rapid, severe freezing injury. Aircrew exposed to the airstream around a flying aircraft through an open hatch or port in the fuselage can incur serious freezing injuries in seconds. Exposure to fluids at subfreezing temperatures, such as gasoline or propane and butane propellants, will also cause immediate, severe freezing injury.[143]

A quantitative index of risk to exposed skin, the Wind-Chill Index, was developed in the 1940s and has been revised many times.[144,145] If used carefully, it is a useful tool for judging the risk of cold exposure. It is important to remember that the Wind-Chill Index does not provide an index either of hypothermic risk or of risk to covered skin. Effective cover will protect skin even in conditions of very "cold" wind chill. Predictive models for risk assessment and management of other types of cold exposure are being rapidly developed.[143,146]

Changing weather conditions are associated with an increased risk of cold injury. Exhaustion hypothermia classically occurs when individuals are caught in unexpected rain or snow. They may have to bivouac without adequate shelter or become lost or delayed in cross-country movement, any of which leads to prolonged cold exposure. Freezing injuries commonly occur at the conclusion of a period of bitter cold when the slightly warmer, but still cold, temperatures do not produce their usual sensation of cold. Thawing causes wet-cold conditions and increases the risk of nonfreezing cold injury.

Combat conditions often reduce the options for mitigating cold exposure and are the military circumstances associated with the highest risk of cold injury. It is in these circumstances that cold injury control relies on maintaining the highest level of cold tolerance and protection.

Thermocompetence

Characteristics of service members that are generally accepted to be risk factors for cold injury include prior cold injury, predisposing conditions, fatigue, dehydration, weight loss, lack of cold weather training and experience, lower rank, origin in a warm climate, black skin, and tobacco use.[147] The medical officer must be completely familiar with the unit and monitor it carefully to judge when particular risk factors are of sufficient magnitude to require intervention.

Previous cold injury is an important risk factor and should be considered in the predeployment assessment of each unit member.[148] Cold injuries, even of mild degree, are good predictors of the likelihood of another cold injury. The risk of reinjury is extremely high if the cold injury occurred in the same cold season but even years later remains significantly above the risk of others who have not had a cold injury. Military experience is consistent in showing the inability of service members with clinically healed cold injury to return successfully to their units in a cold environment. The rates of reinjury are so high that military medical officers have always eventually realized that these casualties must be accommodated by modified duty assignments.[149]

Predisposing conditions include neuropathic and vascular diseases, such as Raynaud's disease[150] (Figure 19-7) or diabetes mellitus. Increased age and sickle cell trait may increase the risk of peripheral cold injury.[37,151] Many cold injury risk factors are inevitable accompaniments of military operations and become more prevalent and severe as time passes. Among these are

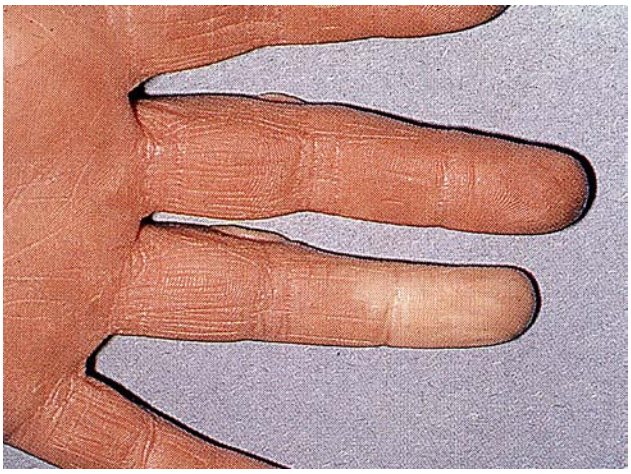

Fig. 19-7. Raynaud's phenomenon in the distal segment of the ring finger. Raynaud's disease is a predisposing factor for cold injury.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

dehydration, weight loss,[152,153] and fatigue[154] (Figure 19-8). An increasing incidence of cold injuries is one way these factors will be expressed. Successful primary prevention will depend on continuous monitoring for these conditions and on actions to mitigate them.

Fig. 19-8. Operations in cold environments increase calorie requirements. Palatability is an important factor in maintaining adequate food intake to meet the increased needs. However, the circumstances of cold weather operations makes the provision of war food difficult. This meal is being consumed in ambient temperatures of –30°F and will freeze in a few minutes.
Photograph: Courtesy of Colonel Wayne Askew, MS, US Army (Ret), USARIEM, Natick, Mass.

Experience is consistent in showing an increased risk of cold injury among individuals from warm climates and individuals with black skin.[131] The reason of the increased risk is not known but the phenomenon must be addressed by the unit preventive medicine program.

Successful cold injury prevention ultimately rests on the skill and knowledge of the service members conducting operations in cold environments and, consequently, on the training they receive. The unit medics should be thoroughly trained in the signs, symptoms, prevention, and management of cold injuries and how to survey aggressively for illness and injury. They must understand that early recognition of cold injuries is essential to minimize their consequences. Unit leaders must understand the causes and manifestations of cold injury, both for their own benefit and for that of their unit. They should understand the importance of hydration, adequate rations, the "buddy" system, and clean, dry, properly used cold weather clothing and footgear on preserving unit function in the field. They should understand the importance of adequate predeployment preparation for cold by conducting cold weather training, inspecting cold weather clothing and equipment, and assuring appropriate health maintenance and medical screening measures.

In World War II, hospitalized cold injury casualties were twice as likely (about 2/3 to 1/3) to say they had never received training in cold injury prevention than casualties with similar cold exposures hospitalized for other injuries.[149] All unit members should know the signs and symptoms of cold injury in themselves and their buddy and know what to do if they suspect a cold injury. They should be alert to the consequences of dehydration, weight loss, fatigue, tobacco use, and alcohol consumption. They must understand and implement the established techniques for foot care in the cold (Exhibit 19-7).

Technology

The ability of a military force to train and operate in a cold environment is critically dependent on the technology they have to protect themselves from the climate (eg, shelters, heaters) and to transport themselves and food and water.[155,156] Technological failures, inappropriate or unskilled use of the available technology, or contingencies that disrupt the function or supply of this equipment will lead to outbreaks of cold injury.[149,157,158]

EXHIBIT 19-7

FOOT CARE IN COLD ENVIRONMENTS

- Assure the best possible fit of the boots with heavy socks.
- Keep the body as warm as possible, avoid chilling.
- Remove boots and socks at least twice a day; wash, dry, massage, and move the feet to restore circulation and feeling; allow enough time and provide appropriate shelter to complete this task; after massaging and warming feet, put on clean, dry socks, or, if dry socks are not available, remove as much water from the wet socks as possible before putting them back on.
- Do not sleep with wet footgear; remove wet boots and socks for sleep; protect the feet with as much dry cover as possible to keep them warm.
- Dry wet socks by keeping them in the sleeping bag during sleep or placing them inside the field jacket against the chest or across the shoulders.
- In fixed positions, stand on rocks, boards, or brush to keep the feet out of water and mud.
- Keep the feet and legs moving to stimulate warming circulation; instead of crouching all the time to keep low in a fixed position, try to sit or lie back periodically with the feet slightly elevated to reduce swelling of the feet and ankles.
- Watch carefully for numbness or tingling—these are early symptoms of injury; if these develop, immediately take measures to warm the feet.
- Keep the clothing and footgear loose enough to permit easy circulation.

Source: United States Army Research Institute of Environmental Medicine. *Medical Aspects of Cold Weather Operations: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1993: 12. Technical Note 93-4.

Implementing a Cold Injury Prevention Program

Cold injury prevention starts before exposure, with screening and identification of service members at increased risk (Exhibit 19-8). In some circumstances risk can be reduced, such as by stopping tobacco use or providing extra protective clothing (Exhibit 19-9). Occasionally individuals will not be able to be accommodated, and they should not be assigned to duties requiring cold exposure. In addition, appropriate equipment and training must be provided before exposure. The unit command group should establish standard cold weather operating procedures that incorporate guidelines for sustaining overall fitness and health (eg, hydration, nutrition, rest), limiting exposure times (eg, work-warming cycles, intervals for inspection and rewarming of face and extremities), and assuring the timely maintenance and replacement of personal equipment. Military medical personnel should develop their own policies and procedures for medical monitoring and rapid reporting of cold injuries.

The most important control measure for preventing cold injury is a command requirement to inspect and rewarm the face and extremities periodically. The interval between rewarming should be determined by the immediate circumstances of the unit and may be as frequent as every 20 or 30 minutes in very cold weather.

Medical surveillance should be performed in an organized fashion by all military medical personnel. Regular written reports are an essential part of the discipline that successful medical surveillance requires. Without organized data collection, surveillance becomes anecdotal and loses a significant amount of its sensitivity for early detection and successful intervention (see Exhibit 19-9).

ILLNESS AND INJURY DUE TO COLD

Freezing Injury (Frostbite)

Frostbite is currently the most common cold injury encountered in US forces and has always been a particular problem for the Army. Isolated episodes are usually associated with an episode of carelessness or sudden weather change, either warming or cooling. Clusters of frostbite injuries occur in exer-

EXHIBIT 19-8

COLD INJURY: IMPORTANT PREVENTION POINTS

- The best prevention against cold injury is a healthy, trained, equipped, well-fed, and hydrated service member with alert and conscientious leaders.
- In military operations and training, risk factors for freezing injuries include
  - dehydration,
  - weight loss,
  - unplanned or unduly prolonged exposures to cold,
  - undertrained or overtired service members,
  - previous cold injury, and
  - poor or insufficient equipment.
- When one freezing injury has occurred in an operation, remember that everyone in the unit was exposed to the same conditions. Inspect everyone immediately.
- Loss of sensation in the feet (they feel like "blocks of wood" or "like walking on cotton") is an ominous symptom and must be immediately evaluated by direct inspection of the feet.
- Cold injuries during operations usually occur in clusters.
- A service member who is shivering is already too cold.
- No one in whom hypothermia is suspected should be left alone.
- Exercise is dangerous if significant hypothermia is present.

Adapted from: United States Army Research Institute of Environmental Medicine. *Medical Aspects of Cold Weather Operations: A Handbook for Medical Officers*. Natick, Mass: USARIEM; 1993: 45–46. Technical Note 93-4.

cises and operations and are frequently the result of poor planning or inattention to control measures. Fortunately, most frostbite injuries occurring during training do not result in permanent tissue loss.[159] The long period of recovery, however, usually means the loss of the injured service member to field duties for the remainder of the cold season. In cold regions, this can mean months of limited duty. For that reason, a unit that suffers a cluster of freezing injuries may become ineffective.

Most freezing injuries will be recognized and initially managed by unit medics and other nonphysician medical providers. Because optimal treatment of the freezing injury depends on early detection and immediate, appropriate management, ultimately the clinical outcome will depend on the successful training and skills of the unit medics.

Pathogenesis

Frostbite injury results when tissue is cooled sufficiently to freeze.[160] Tissues with large surface-to-mass ratios (eg, ears) or with restricted circula-

EXHIBIT 19-9

COLD INJURY PREVENTION PROGRAM

- Screen, select, immunize, and train service members before exposure.
- Provide appropriate equipment and training in its use.
- Establish unit exposure-control SOPs.
- Establish medical SOPs for medical monitoring, first aid, and rapid reporting of cold injuries.
- Obtain supplies for casualty management and evacuation in cold environments.
- Predict and monitor exposure.
- Monitor unit members' thermocompetence.
- Maintain and replace equipment.
- Inspect periodically the entire unit for injury.
- Respond to cold injuries with modification in policy and procedure as needed.

SOP: standard operating procedure

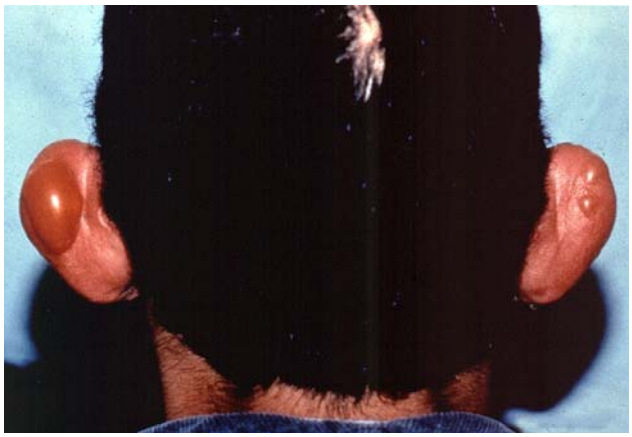

Fig. 19-9. This is a case of second-degree frostbite. The ears are commonly affected by frostbite.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

tion (eg, hands, feet) are particularly susceptible to freezing, but any tissue exposed to severe cold can freeze (Figure 19-9).

Tissue does not freeze at 0°C (32°F); the high concentration of electrolytes and other solutes prevents freezing until tissue is cooled below –2°C (28°F). At that point, ice crystals form, which segregate some tissue water and cause concentration of the remainder into a progressively more hypertonic and harmful solution. Once solidly frozen, tissue injury is probably arrested. Additional injury to frozen tissue occurs during and after thawing, probably in two phases. First, on restoration of blood flow, reperfusion tissue injury occurs.[161] Second, marked endothelial swelling develops in the thawed tissue, causing secondary loss of perfusion, ischemia, and infarction of tissue. Despite freezing and reperfusion injury, some frostbitten tissue is able to recover. Refreezing of injured tissue, however, causes irredeemable injury, a phenomenon used therapeutically in cryosurgery.

Clinical Manifestations and Classification

Initially, all frozen tissue has the same characteristics: it is cold, hard, and pale.[162] Except in minor and severe cases, the degree of injury will usually not become clear for 24 to 72 hours. Most significant injuries include areas with different degrees of frostbite, with the distal areas usually more severely affected. Digits, ears, and exposed facial skin are the most commonly injured areas.

Frostbite is classified by depth of injury, which determines both the prognosis and speed of recovery. Superficial injuries are categorized as first- and second-degree frostbite. Deep injuries are categorized as third- and fourth-degree frostbite. The depth of the injury depends on both the duration and the intensity of the cold exposure. Very intense cold for a few seconds will produce a superficial injury whereas prolonged exposure to moderate freezing cold can freeze an entire extremity.

First-degree frostbite is an epidermal injury. The affected area is usually limited in extent, involving skin that has had brief contact with very cold air or metal (eg, touching an outside door handle). The frozen skin is initially a white or yellow plaque. It thaws quickly, becoming wheal-like, red, and painful. Since deep tissues are not frozen (though they may be cold), mobility is normal. The affected area may become edematous but does not blister. Desquamation of the frostbitten skin with complete clinical healing follows in 7 to 10 days (Figure 19-10).

Second-degree frostbite involves the whole epidermis and may also affect superficial dermis. The initial frozen appearance is the same as a first-degree injury. Since the freezing involves deeper layers and usually occurs in tissue with prolonged cold exposure, some limitation of motion is present early. Thawing is rapid, with return of mobility and appearance of pain in affected areas. A blister, with clear fluid, forms in the injured area several hours after thawing. Usually, the upper layers of dermis are preserved, which permits rapid re-epithelialization. Second-degree injuries produce no permanent tissue loss. Healing is complete but takes at least 3 to 4

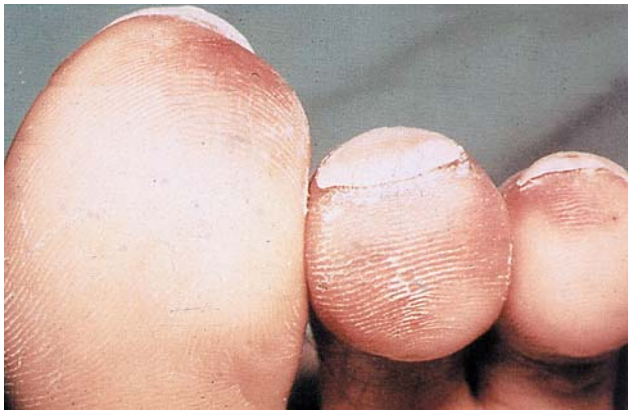

Fig. 19-10. First-degree frostbite ("frostnip") is the most common military cold injury. The affected area is susceptible to deeper injury until healed so cold exposure needs to be curtailed for even this apparently modest injury.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

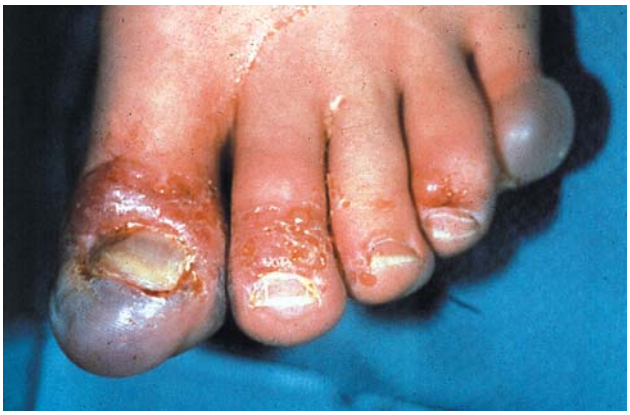

Fig. 19-11. Second-degree frostbite is a severely disabling injury and is likely to affect the service member's ability to remain on active duty. Urgent evacuation is required. Recovery will take many weeks to months.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

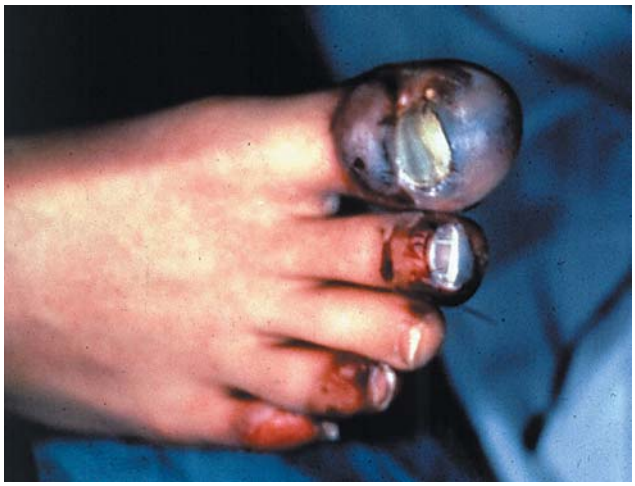

Fig. 19-12. Significant tissue loss and long-term cold sensitivity is to be expected after third-degree frostbite. This is likely a career-ending injury.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

weeks. First-degree injury is frequently present in the immediate vicinity of second-degree frostbite. Frostbite should be looked for on all other exposed areas of skin. Following second-degree frostbite, cold sensitivity may persist in the injured area (Figure 19-11).

Third-degree frostbite involves the dermis to at least the reticular layer. Initially, the frozen tissue is stiff and restricts mobility. After thawing, mobility is restored briefly, but the affected skin swells rapidly and hemorrhagic blisters develop due to damage to the dermal vascular plexus. Significant skin loss follows slowly through mummification and sloughing. Healing is also slow, progressing from adjacent and residual underlying dermis. There may be permanent tissue loss. Residual cold sensitivity is common (Figure 19-12).

Fourth-degree frostbite involves the full thickness of the skin and underlying tissues, even including bone. Initially the frozen tissue has no mobility. Thawing restores passive mobility, but intrinsic muscle function is lost. Skin reperfusion after thawing is poor. Blisters and edema do not develop. The affected area shows early necrotic change. The injury evolves slowly (weeks) to mummification, sloughing, and autoamputation. Whatever dermal healing occurs is from adjacent skin. Significant, permanent anatomic and functional loss is the rule.

Basic Principles of Management

Since many frostbite injuries result in formal investigations, careful records should be made from the outset, including at least a complete description of the circumstances of the injury, its initial extent and appearance, and the first steps of management.

The first essential step in cold injury management is detection. Frostbite injuries are insidious. Injured tissue, which is painful initially while it is getting cold, is anesthetic when frozen and is often covered by a glove or boot. Detection requires direct inspection of at-risk tissue, including the hands, feet, ears, nose, and face.

Active warming of frozen tissue should be deferred until there is absolutely no risk that the injured tissue can be reexposed to freezing cold. This recommendation does not mean that the injured part should be deliberately kept frozen by packing in snow or continuing the cold exposure. If refreezing can be prevented during evacuation, then frozen tissue can be immediately warmed by contact with warm skin. Once tissue has thawed, it is essential that it be protected from reexposure to cold. The tissue must not be exposed to temperatures in excess of 40°C (104°F–105°F), which will aggravate the injury. Exposure to exhaust manifolds, open flames, stovetops, incandescent bulbs, or hot water is particularly dangerous. Frostbitten tissue is vulnerable to trauma and should be carefully protected from physical injury during evacuation.

Digits or entire hands or feet can be warmed in a temperature-monitored water bath kept from 39°C to 41°C (102°F–105°F). Facial tissue or the ears can be thawed with warm, wet towels. Warming should be continued until no further improvement in the return of circulation and mobility is noted. The time required will depend on the initial temperature and

size of the injured part and can take more than an hour in severe cases. After warming, the frostbitten tissue should be carefully and atraumatically dried, completely covered in bulky, dry dressings, and kept slightly elevated to moderate swelling.

After the necessary emergency stabilization is accomplished and warming has begun, early management includes tetanus prophylaxis as appropriate and analgesics. During warming, pain appears and is often intense. Nonsteroidal antiinflammatory drugs and narcotics should be provided as needed.

Freezing injuries should always be considered serious. Military medical practice is to evacuate all cold-injured casualties to rear echelons for discharge or reassignment to modified duty without cold exposure. The continuing care of freezing injury is intended to minimize the loss of tissue by providing the optimum environment for healing, avoiding additional injury and infection, and permitting spontaneous evolution of tissue loss.

The principal late complications of frostbite include tissue loss, contractures, persistent pain, cold sensitivity, susceptibility to reinjury, and hyperhidrosis. After healing, cold exposure of any portion of the skin may precipitate symptoms in the area of a previous injury. Relocation to a warm climate may be required if cold intolerance is intractable. Tissues that have suffered a frostbite injury are probably more susceptible to cold injury and should receive extra protection and attention when exposed to cold. Hyperhidrosis of the feet can increase the incidence of dermatophyte infection and maceration.

Nonfreezing Cold Injury

Nonfreezing cold injury (NFCI) is the result of prolonged (many hours) exposure of the extremities to wet-cold of between 0°C and 18°C (32°F–65°F).[163] The feet are the most common area of injury, which is reflected in the common names of the two principal types of nonfreezing injury: trench foot and immersion foot. Trench foot occurs during ground operations and is caused by the combined effects of sustained cold exposure and restricted circulation. Immersion foot is caused by continuous immersion of the extremities in cold water and usually occurs in survivors of ship sinkings. Trench foot is rare outside of military operations, but immersion injury is a risk of any maritime venture.

Pathogenesis

Prolonged cooling produces some damage to all the soft tissues, but peripheral nerves and blood vessels suffer the greatest injury.[164–166] The vascular injury causes secondary ischemic injury, which aggravates the direct effect of cold on other tissues. Wet conditions increase the risk and accelerate the injury both because wet clothing insulates poorly and because water itself cools more effectively than air at the same temperature. Factors that reduce circulation to the extremities also contribute to the injury. In military operations, these factors include constrictive clothing and boots, prolonged immobility, hypothermia, and crouched posture. Maceration of the wet skin can complicate NFCI and predisposes the service member to infection.

Clinical Manifestations and Classification

When first seen, the injured tissue is pale, anesthetic, pulseless, and immobile but not frozen.[167] Trench foot or immersion foot (depending on the environmental medium causing the injury) is likely when these signs do not change immediately after warming. Like freezing injury, the degree of the injury is usually not apparent early.

The course of NFCI is classically divided into preinflammatory, inflammatory, and postinflammatory phases.[168] In the preinflammatory phase, despite rest and warmth, the injured part remains pale, anesthetic, and pulseless. After several hours (occasionally as long as 24-36 hours), the inflammatory phase begins with the appearance of a marked hyperemia associated with burning pain and the reappearance of sensation proximally but not distally. The hyperemia represents a passive venous vasodilation and blanches with elevation. Edema, often sanguineous, and bullae develop in the injured areas as perfusion increases. Skin that remains poorly perfused after hyperemia appears is likely to slough as the injury evolves. Persistence of pulselessness in an extremity after 48 hours suggests severe deep injury and high likelihood of substantial tissue loss. The hyperemia lasts a few days to many weeks, depending on the severity of the injury (Figure 19-13).

Recovery from NFCI is slow due to its neuropathic component. Except in minor injuries, deep aching develops that is associated with sharp, intermittent "lightning" pains in the second week after injury. Improved sensitivity to light touch and pain in the area of anesthesia within 4 to 5 weeks suggests reversible nerve injury and less likelihood of persistent symptoms. Persistence of anesthesia to touch beyond 6 weeks suggests neuronal degeneration. Injury of this degree takes much longer to resolve and has a greater likelihood of persistent disabling symptoms.

Hyperhidrosis is a common and prominent late feature of NFCI and seems to precede the recovery of sensation. A distinct advancing hyperhidrotic

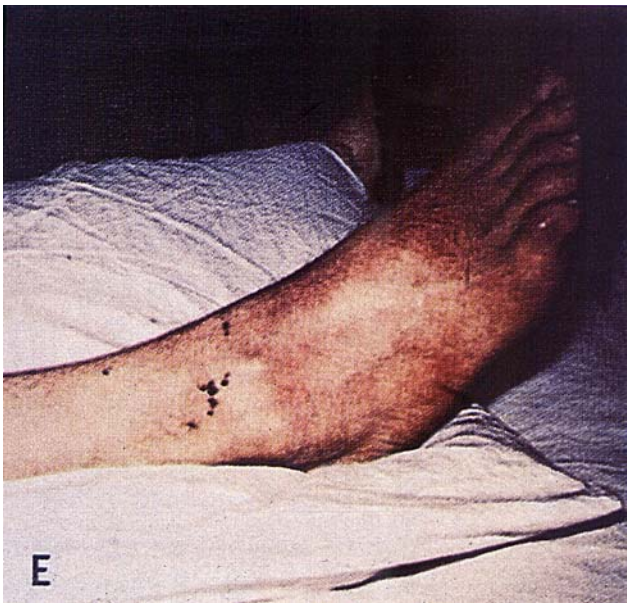

Fig. 19-13. This is a case of trench foot from Italy in 1943. This photograph was taken several months after the initial injury during convalescence in the United States. Source: Whayne TF, DeBakey ME, ed. *Cold Injury: Ground Type*. Washington, DC: Office of The Surgeon General, United States Army; 1958: 265.

zone can develop and is presumed to mark the point to which sudomotor nerves have regenerated.[169] The excessive sweating may be permanent. It predisposes the individual to blistering, maceration, and dermatophyte infection.

NFCI has been classified into four degrees of severity.[170] Two schemes of classification have been used based on clinical case series from World War II. The Webster classification[171] is based on the clinical appearance of the foot 2 to 3 days after injury. The Ungley classification[172] is based on the distribution of anesthesia 7 days after injury. These two systems correlate well and provide useful prognostic information.

Basic Principles of Management

NFCI, like frostbite, is an insidious injury because the affected tissue is cooled to the point of anesthesia while the injury is occurring. So, like frostbite, the first essential of management is detection. Foot inspection and care every 8 hours under cold-wet conditions will prevent most cases and allow detection of early injury. Boots and socks should not be replaced on the feet until the feet are warm and have normal feeling. Residual anesthesia after warming is evidence of NFCI. Service members who suspect a NFCI should warm their feet immediately and seek medical evaluation.

If NFCI is suspected, priority evacuation is appropriate. Because tissue injured by NFCI is as vulnerable to trauma and cold exposure as thawed freezing injuries, the injured extremity must be carefully protected during evacuation. If the lower extremity is involved, the casualty must be moved by litter, vehicle or aircraft; ambulation is not possible. If warming does occur during evacuation, severe pain may develop before arrival at a medical treatment facility. Consequently, if a prolonged evacuation is anticipated, the service members performing the evacuation should be equipped and trained to provide adequate analgesia. The possibility that pain may appear during evacuation is not a reason to keep an injured extremity cool.

Active warming is not necessary for NFCI. The extremities will warm spontaneously when the casualty is removed from cold-wet conditions. Massage of the injury "to restore circulation" may worsen the injury.

NFCI, even in its mildest expression, evolves slowly and requires time (more than 1 week) for evaluation and recovery. Therefore, when a casualty is considered to have this type of injury, he or she should be evacuated to a rear-echelon hospital. Nothing is to be gained from observation in forward echelons.

The principal requirements of initial hospital management are tetanus prophylaxis, management of concomitant hypothermia and dehydration, and pain relief.[167] The injured extremity should be kept at the temperature providing the greatest comfort. After hyperemia appears, cooling by a fan usually provides some relief from the burning pain. External warming should not be used. Dry, loose dressings can be used to cover the injury, but even the weight of bedclothes may aggravate the pain. Pain relief should be provided as needed.

As the injury evolves, pain and infection present the primary clinical challenges. To minimize pain and avoid mechanical injury, weight bearing should not be allowed until the circulation has been fully restored, edema has cleared, and any maceration or ulceration has healed. Patients with areas of indolent dry gangrene in the toes may walk if the other parts of the feet can be protected against further injury. Deep pain on weight bearing may limit walking for periods of a few days in minimal injury to months in severe injury. Macerated and ulcerated skin increases the likelihood of infection. The skin should be assiduously protected with dry dressings. Intact bullae should be left intact; ruptured bullae should be sharply debrided and dressed.

Anatomic defects and functional symptoms commonly cause persistent disability after NFCI. The more common of these defects and symptoms include loss of toes and other forefoot tissues, ham-

mer toe deformities, flexion contracture of the great toe, hyperhidrosis predisposing the individual to skin maceration and dermatophytosis, persistent pain (either spontaneous or when bearing weight), and cold intolerance.

Accidental Hypothermia

Hypothermia is the clinical syndrome that results from reduced core temperature.[173–177] By definition, hypothermia is considered present when the "core" temperature (clinically usually taken to be the same as rectal temperature) is below 35°C (95°F). Hypothermia is always the product of loss of heat to the environment in excess of the rate of heat production by the body. Hypothermia may be induced deliberately or may occur due to a failure of thermoregulation due to environmental exposure. This last category is called accidental hypothermia and is the type that occurs in military settings. It is the subject of this section.

Pathogenesis

The sequence of events during whole-body cooling and rewarming is well known. The initial response to a fall in core temperature is peripheral vasoconstriction, followed by an increase in muscle tone and metabolic rate.[178] With continued fall in core temperature, shivering, tachypnea, tachycardia, and hypertension develop. These become maximal when the core temperature is about 35°C. Below that temperature, the depressant effect of hypothermia begins to offset the metabolic activation. As core temperature falls from 35°C to 30°C (86°F), metabolic rate, shivering, respiratory rate, heart rate, and cognitive function all decline. The individual may initially become quiet and withdrawn or confused and combative but eventually becomes obtunded. Furthermore, since the metabolic depression of hypothermia stops the hypermetabolic response to cold, the individual loses a substantial defense against any additional fall in core temperature. Below 35°C core temperature, heart rate, blood pressure, and respiratory rate decline roughly in parallel. Metabolic rate, oxygen consumption, and cardiac output are about half of normal at 29°C (85°F) and about 20% of normal at 20°C (68°F). At these lower temperatures, ventilation and perfusion do not quite keep up with metabolic requirements and a mixed respiratory and metabolic acidosis develops.[179]

When the core temperature falls below 30°C (86°F), atrial tachyarrhythmias and repolarization abnormalities (Osborne waves[180]) appear. Below 28°C (82°F), the ventricular fibrillation threshold declines, presumably due to reduction of Purkinje fiber conduction velocity. Peripheral voluntary muscle activity and reflexes disappear at about 27°C (80°F). Brainstem reflexes disappear at about 23°C (73°F). Below 20°C (68°F), electrical activity disappears, first in the brain and then in the heart. Despite the disappearance of all objective evidence of life at these low core temperatures, resuscitation is possible.

Clinical Manifestations and Classification

Immersion hypothermia is usually the result of boating, ice skating, or automobile accidents. Airplane accidents and shipwrecks can produce mass hypothermic casualties. The fall in core temperature during cold water immersion is rapid and steady. Several factors influence the rate and magnitude of core temperature reduction, including water temperature, protective clothing, body posture and movement, body size and adiposity, and thermoregulatory aggressiveness. Individuals vary in their thermoregulatory response to cold-water immersion. Individuals with less vigorous vasoconstrictive and shivering responses to cold will cool more quickly than individuals with more vigorous responses.

Exhaustion hypothermia (sometimes called "exposure") results when individuals exposed to cold conditions on land are unable because of fatigue or injury to sustain a metabolic rate sufficient to balance the loss of heat to the environment.[181] Factors that influence the rate of temperature fall during exposure to cold land environments are ambient temperature, wind, clothing, precipitation, rate of physical activity, and shelter. Precipitation reduces the insulating value of clothing and adds an additional source of cooling. Physical activity during cold exposure on land, in contrast to activity during immersion, is an important mechanism of maintaining core temperature. The benefit of physical activity lasts only as long as activity is maintained. Dry shelter moderates the cooling effect of wind and precipitation and may allow an opportunity for rest without risk of excessive cooling.

The severity of hypothermia depends on the degree of temperature depression. Hypothermia is classified as mild, moderate, or severe, based on core temperature. Mild hypothermia is defined as core temperatures between 32°C and 35°C (90°F–95°F). Casualties with mild hypothermia usually retain the ability to rewarm spontaneously and do not develop cardiac arrhythmia. Between 32°C and 28°C (90°F–82°F), the range of moderate hypothermia, atrial arrhythmias become common, and meta-

bolic rate is sufficiently depressed to significantly slow the rate of spontaneous rewarming. Below 28°C, the range of severe hypothermia, spontaneous rewarming is markedly depressed, and the risk of ventricular fibrillation becomes substantial.

The clinical manifestations of mild-to-moderate hypothermia are frequently insidious and subtle. Mild-to-moderate hypothermia may not be recognized unless it is suspected and core temperature is measured. If oral temperature is not over 35°C, rectal temperature should be measured with a low-reading thermometer.

The principal manifestations of mild-to-moderate hypothermia are shivering and mental status change.[182] Persistent shivering is evidence of incipient hypothermia and should always be taken seriously. Shivering will diminish as hypothermia worsens. Since some individuals do not shiver, mental status change may be the only clinical evidence of significant hypothermia. Withdrawal and irritability are common. As hypothermia worsens, subtle mental status changes progress to frank confusion, lethargy, and obtundation. The degree of mental status change is not a reliable guide to the degree of hypothermia. For example, individuals have been reported to remain conscious at core temperatures of below 27°C (80°F).

The clinical manifestations of hypothermia become more dramatic and more obvious as core temperature falls. Cool, pale skin, obtundation, atrial arrhythmias, bradycardia, and hypopnea are all present at core temperatures between 27°C and 32°C (80°F–90°F). At core temperatures between 21°C and 27°C (70°F–80°F), reflexes and vital signs become imperceptible, the skin is cold and waxy, and muscular rigidity may be present. The brain and the heart become electrically silent at core temperatures between 16°C and 21°C (60°F–70°F), and the hypothermic casualty appears clinically dead.

Basic Principles of Management

Anyone suspected of hypothermia should be considered to be at risk of sudden death from ventricular fibrillation due to ventricular irritability, hypovolemia and orthostasis, and sudden intraventricular cooling.[183–185] Handling should be minimal and gentle. Copious insulation to prevent heat loss should be placed around the casualty at the same time wet clothing is removed.[186] The insulation under the casualty should be incompressible. Airway heat loss should be prevented by any means available, even if only a scarf or non-occlusive bandage.

Since dehydration and hypovolemia are common in hypothermic casualties, an intravenous (IV) line should be started with warmed fluid. If hypoglycemia, alcoholism, or opiate intoxication are possible causes of hypothermia, naloxone, thiamine, and glucose should be administered intravenously.

The goal of successful resuscitation from hypothermia is the restoration of normal core temperature without causing complications. Many techniques have been used to accomplish rewarming. Techniques that take advantage of the casualty's own inherent metabolic heat generation, which is present to some degree in every hypothermic patient, are called passive rewarming. Those that apply external sources of heat are called active rewarming.

Passive rewarming techniques provide sufficient insulation to both the body and the airway to prevent further heat loss. Passive rewarming is effective even in those with core temperatures as low as 27°C. Depending on the effectiveness of the insulation, core temperature increases from 0.45°C to 1.8°C per hour (0.25°F–1°F per hour). Passive rewarming is appropriate only as long as temperature continues to rise, although it may take 24 to 36 hours to restore normothermia. Passive rewarming consumes relatively few intensive care resources, allows for gradual re-equilibration during rewarming, and avoids the complications of the invasive techniques. The principal disadvantages of passive rewarming are the long time to normothermia and the need for continued surveillance to assure that core temperature is increasing.

Active rewarming techniques of several types have been used. Active "surface" rewarming techniques apply heat to the periphery (eg, warm baths to trunk or extremities, heating blankets, warm towels to groin and axilla). These techniques are not technically demanding and are probably helpful for mild and moderate hypothermia, but there are four caveats. First, they are not effective if cardiac arrest has occurred. Second, by increasing blood flow to skin and extremities before central rewarming has occurred, they may increase the delivery of cold peripheral blood and precipitate hypotension and cardiac cooling. Third, since hypothermic skin is vulnerable to burning, careful monitoring of the temperature of the heat source is needed. And fourth, they may inhibit vasoconstriction and reduce endogenous heat generation in mild-to-moderate hypothermia.

Active rewarming of the core is required for resuscitation of hypothermic cardiac arrest and for most severely hypothermic patients.[187] Core rewarming techniques are intraluminal lavage,[188]

heated air,[189,190] direct vascular warming,[191–193] and radiant energy.[194] The choice of technique for rewarming depends on the state of the circulation and the degree of hypothermia. The principal postwarming complications of accidental hypothermia are pneumonia (including aspiration after immersion), pancreatitis,[195] rhabdomyolysis,[196] myoglobinuria, and renal failure. Temporary left ventricular dysfunction has been seen after severe hypothermia. In addition to the late complications of cerebral anoxia and organ injury, hypothermia occasionally causes cold sensitivity and contractures in the hands or feet or both.

Other Medical Problems Associated With Cold Exposure

Chilblains

Chilblains (pernio) are small erythematous papules that appear most commonly on the extensor surface of the fingers but can appear on any skin chronically exposed to above-freezing cold.[197–199] Ears, face, and exposed shins are other common locations. Multiple lesions are the rule. The lesion is pruritic and painful, particularly after reexposure to cold. It is indolent and does not remit until cold exposure has ceased. Chilblains frequently recur upon the return of cold weather. Chilblains occasionally ulcerate (Figure 19-14). Management of chilblains is by protection from cold with suitable clothing. Nifedipine has been shown to be effective in treating refractory cases.[200] Symptoms will remit when cold exposure is eliminated.

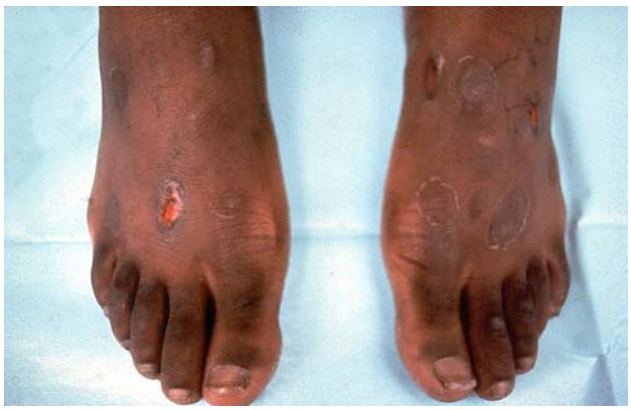

Fig. 19-14. Chilblains are pruritic and painful lesions that develop in the extremities in cold, damp environments. Their pathophysiology is not understood. They remit on termination of cold exposure but can recur.
Photograph: Courtesy of Commander, USARIEM, Natick, Mass.

Respiratory Tract Conditions

The nasal and bronchial mucosa respond to cold air by increasing mucus production. Consequently, chronic nasal discharge and cough are frequent accompaniments of cold weather operations. Rhinorrhea and bronchorrhea are probably protective but can cause two principal complications. Nasal discharge causes wetting and chapping of the philtrum and nasolabial sulcus, which increases the risk of cold injury and local infection. And the increased secretions may accumulate and interfere with drainage of the sinuses, leading to sinusitis. Decongestants may reduce nasal secretions but commensurately reduce their protective effect. Careful hygiene is a better measure than decongestants to prevent chapping. Military arctic mittens are designed with a pad to wipe nasal secretions from the face without taking off the mitten.

Crowding and poor ventilation in tents and other shelters increase dissemination of respiratory infections (Figure 19-15). Influenza vaccination is an essential preventive measure. Medical personnel should perform aggressive surveillance, particularly for streptococcal infection. Early intervention with appropriate infection control can curtail the spread of respiratory infection. Some individuals will experience bronchospasm on exposure to cold dry air or fumes from fuel-fired heaters. These individuals should be evacuated for evaluation.

Fig. 19-15. Close quarters are a consequence of cold weather operations. The combination of weight loss, cold-induced changes in skin and respiratory mucosa, and closed living environments all contribute to a high rate of infectious disease among deployed personnel.
Photograph: Courtesy of COL Wayne Askew, MS, USA (Ret).

Cold Urticaria

Cold urticaria, which is manifested as local or systemic urticaria on exposure to cold, can be familial, congenital, or acquired.[198] Its onset is usually abrupt and distinctive and reflects the activation by cold of immune modulators. It can be reliably induced by local application of ice or immersion of an extremity in an ice bath, which will reproduce local or systemic symptoms. Cold urticaria is potentially lethal from either systemic anaphylaxis or laryngeal swelling on drinking cold liquids.

Immune mediators activated by cold can cause other reactions besides urticaria, including cyanosis, livedo reticularis, pruritus, paresthesia, and Raynaud's phenomenon in the acral regions of the body. Immune complexes deposited in the microcirculation can cause distal ulceration and even frank gangrene. Cold hemolysin and agglutinins can precipitate episodic hemolysis and hemoglobinuria.

Intolerance of Cold Exposure

Some individuals without any history or evidence of past or present cold injury complain of recurrent pain and burning on cold exposure. This phenomenon has not been well studied, but clinical impressions are that it seems to be more prevalent among those who have repeated, prolonged lower extremity exposures to moderate cold. No objective findings have been identified. Occasionally, the symptoms seem to become manifest with progressively less cold exposure. There is, at present, no diagnostic term for this problem and no management plan beyond avoidance of cold exposure. Service members who develop this complaint may require medical and fitness-for-duty evaluations if symptoms are disabling.

Eczema

This condition, also known as winter itch or "eczema craquele," is extremely common on the hands in the cold and frequently will generalize to involve all the skin. It is manifested by persistent painful itching, thickening and painful cracking of the skin of the fingers and toes, and fine scaling of the skin on extremities and trunk. The cause appears to be loss of the neutral lipids from the stratum corneum, which allows drying and irritation of the lower layers of skin. Frequent washing appears to be the principal cause of the cutaneous delipidation. The cracking and fissuring of skin on the digits is painful and carries with it the risk of infection. The problem can be prevented and managed by moderating the frequency of washing (not usually much of a problem during cold weather training or operations), avoiding harsh soaps, and applying emollient creams to replace neutral skin lipids.

Operational Considerations: Medical Operations in the Cold

Medical units must develop medical support plans that allow the earliest possible care and stabilization of cold casualties with the least possible risk to rescuers and casualties. Provision for initial management in the field in portable shelter while waiting for vehicular evacuation is often preferable to an immediate attempt to move a casualty cross-country.

In the cold, the advantage of vehicular evacuation (eg, ground ambulance, tracked vehicle, helicopter) over manual litter evacuation is magnified. Movement of casualties by litter or sled is very slow, significantly delays their treatment, and exposes them to significant risk of hypothermia. An entire squad of 10 to 12 service members is needed to move a casualty in the mountains or in snow. The long nights in winter mean more movement must be done in the dark, increasing the risk of injury to the rescuers and to the casualty. The techniques of movement and the cover required to keep the casualty warm on a litter make observation and medical intervention difficult during evacuation. Whatever technique of evacuation is used, sufficient protection from cold must be available for the casualty during transportation. It is very important to remember to prevent heat loss from beneath the casualty. The down or synthetic material of sleeping bags will compress and lose its effectiveness under the casualty's weight. Additional incompressible insulation (eg, a foam mattress) is required regardless of the surface beneath since the casualty will be at rest and often hypometabolic; two arctic sleeping bags may be barely sufficient insulation. Airway heat loss should also be prevented.

Helicopter landings may raise huge opaque clouds of snow, blinding both pilots and ground staff. Landing areas should be cleared of snow and debris. If a clean landing site is impossible, helicopter operations should be performed at a distance from the medical treatment facility. This would help to prevent injury in case of a landing accident and avoid snow blowing into the hospital interior spaces. Patients awaiting evacuation

should be kept in shelter until the rotorwash and blowing snow have cleared.

Fluids and medications may freeze and become useless if carried in packs or bags. Certain medications are damaged by freezing and must not be used after thawing.[201] These include epinephrine, NPH insulin, sodium bicarbonate, magnesium sulfate, tetanus toxoid, and mannitol. A frozen bag of IV solution in the field is only excess weight. Carrying medication and bags of IV fluid inside cold weather clothing during evacuation will prevent their freezing. Even warm fluids can freeze while running through IV tubing in the cold. If necessary, an IV bag can be placed under the casualty in the sleeping bag and the fluid infused by the casualty's own weight.

The management of hypothermia in the field presents special problems. To avoid sudden death due to cardiac arrest, hypothermic casualties must be kept absolutely quiet. They must not participate in their own rescue. They must be kept supine or head down.

To prevent further heat loss, wet clothing must be removed, copious insulation provided, including insulation of the airway. The casualty must be ventilated with warm, humidified air. Endotracheal intubation is safe if needed. Oxygen supplementation is not required for hypothermia alone. Moderate volume resuscitation should be provided (1–2 L) with warm fluid. If evacuation requires reexposure or interruption of resuscitation, rewarming before evacuation should be considered.

Hypothermia is a significant risk during resuscitative care of burn and trauma casualties in forward areas, particularly if surgery is required.[202] These casualties often arrive already hypothermic due to the effect of shock and cooling during evacuation. Careful monitoring of temperature during and after resuscitation will detect significant hypothermia and permit treatment before and during transportation to rear echelons.

Medical staff members are as susceptible to cold injury as anyone else. Since medical areas are usually kept relatively warm, the basic work uniform is light. Consequently, significant frostbite can result from not taking the time to dress appropriately for outside exposure. Frostbite injury is also caused by hasty handling of litters or equipment brought in from outside. Outside air drafts during helicopter evacuation are particularly dangerous because of the rapidity with which they can cause freezing injury.

Other Hazards in Cold Environments

Carbon Monoxide Poisoning

During cold weather operations, the continuous running of vehicle engines to prevent freezing and the use of fuel-fired heaters in tents and other closed spaces poses a risk of carbon monoxide (CO) poisoning. CO is extraordinarily toxic. Concentrations of 100 parts per million in air at sea level will produce carboxyhemoglobin concentrations of up to 20% and frank toxicity. Headache, vomiting, and change in mental status are typical symptoms of CO poisoning. "Cherry red skin," although frequently mentioned as a specific physical finding, is unusual even in severe cases. Its absence should not be considered as excluding CO poisoning. Extremities below 10°C (50°F) reduce their oxygen utilization to low levels, so that the perfusing blood retains its arterial color. Cold, bright red extremities are, therefore, not evidence of CO poisoning. The bright red color of carboxyhemoglobin should be looked for in warm tissue (eg, oral mucosa).

Management of CO poisoning is immediate administration of 100% oxygen by a close fitting mask or endotracheal tube. Hyperbaric oxygen will accelerate the clearance of CO. Indications for hyperbaric oxygen are carboxyhemoglobin of greater than 25%, metabolic acidosis, angina pectoris or electrocardiogram change, and neurologic symptoms other than headache. If air evacuation is necessary, the lowest possible altitude should be used to maintain the highest possible PO_2 (partial pressure of oxygen).

Solar Keratitis (Snow Blindness) and Sunburn

The dry air and brilliant reflectivity of snow combine to generate a risk of ultraviolet burns to skin and eyes. This risk is tremendously enhanced at altitude. The injury is not apparent until after exposure, so prevention by appropriate protective equipment is essential. Management of both types of burn is symptomatic. Solar keratitis is managed with topical ophthalmic antibiotics, cycloplegics, and oral analgesics. If outdoor exposure is unavoidable, the eyes must be protected by patching. Solar keratitis is disabling for several days, and injured eyes are susceptible to reinjury.

Sunburn can be severe, with blistering of the skin and intense pain. Treatment is conservative,

using oral analgesics and protecting skin from further injury. Control measures include using clothing and sunscreen to protect skin, and sunglasses to protect the eyes. If sunglasses are not available, opaque eye covering (eg, tape-covered eyeglasses) with narrow horizontal slits provide adequate field-expedient eye protection.

Traumatic Injury and Falls

Control measures include careful preparation, maintenance, and marking of paths, roads, and load-handling areas; separation of pedestrian and vehicular traffic; establishment of one-way vehicular traffic; extra help for work details; and illumination of work areas.

Alcohol

Alcohol consumption increases the risk of all forms of illness and injury in the cold.[203] It increases the risk of hypothermia and frostbite by a combination of effects: impaired self-protective behavior, reduced shivering and heat generation, reduced pain of cold exposure, dehydration from diuresis, and inhibited gluconeogenesis. There are no known beneficial effects of alcohol in the prevention or management of cold injury.

MOUNTAIN ENVIRONMENTS

The Environment

The stressor most characteristic of the high terrestrial altitude environment is hypobaric hypoxia and an equation explaining its relationship to environmental strain is shown here:

$$(3)\ \text{Strain} = f\,\frac{\text{Time} \bullet \text{Hypoxia}}{\text{Altitude Tolerance} \bullet \text{Technology}}$$

US military forces have trained for altitude operations since the activation of the 10th Mountain Division in World War II. However, it has never actually had to conduct conventional combat operations at an altitude where hypoxia was an important limiting factor. Rather, as exemplified by the World War II campaign at Monte Cassino, Italy, the other difficult and dangerous features of mountain environments were the challenges that needed to be overcome.

Military operations at significant terrestrial altitudes are usually considered unlikely. At present, however, military forces are stationed and in conflict at altitudes up to 6,000 m (20,000 ft) in the Himalayan mountain range, and guerillas operate in the 4,000 to 5,100 m (13,000–17,000 ft) altitudes of the altiplano in Bolivia and Peru. US Army and Marine Corps units have deployed for training and humanitarian assistance to these attitudes, as have special warfare forces (Table 19-2).

Even when hypoxia is not a significant stress, mountain environments present significant and unique hazards. These hazards include irregular and steep terrain, extremes of heat and cold, intense ultraviolet radiation, lack of water, flash floods, lightning, and difficult supply and evacuation. For example, solar heat load in the mountains exceeds that of equatorial deserts at the same latitude, and dramatic day-to-night changes in temperature are typical (40°C–70°C [72°F–126°F]). These hazards, which are at least as great a source of illness and injury as illness due to hypoxic exposure, are magnified when combined with the physical and psychological effects of hypoxia. These must be appreciated and addressed in a preventive medicine plan.

Mountains are difficult operational environments, and this will affect the health service support function. Supply and evacuation routes are long and treacherous. Aircraft and land vehicles may not be able to approach bivouac and operational areas. Cold will freeze equipment and supplies; water supply and storage equipment is particularly vulnerable. Mountain environments are usually very dry, so natural water supplies are limited. All personnel, medical staff included, are likely to be impaired by hypoxia or acute mountain sickness.

Physiological Response and Acclimatization to Hypobaric Hypoxia

The immediate physiological response to hypobaric hypoxia is hyperventilation and tachycardia.[204] Both reflexes originate in the oxygen-sensing cells of the carotid body. The hyperventilation reduces the oxygen tension gradient between inspired and alveolar air and counteracts the reduction in arterial oxygen tension. The tachycardia increases the delivery of blood to the periphery, which reduces the oxygen tension gradient between the capillary blood and tissues. The respiratory alkalo-

TABLE 19-2

PHYSIOLOGICAL CORRELATES OF ALTITUDE

Altitude (m/ft)	Atmopheric Pressure (mmHg)	PaO_2* (mmHg)	O_2 Sat† (%)
0	760	96	96
1,600/5,280	627	69	94
3,100/10,000	522	57	89
4,300/14,000	448	40	84
5,500/18,150	379	35	75

*partial pressure of oxygen in arterial blood
†% of hemoglobin saturated with oxygen
Adapted from US Army Research Institute of Environmental Medicine. *Medical Problems in High Mountain Environments: A Handbook for Medical Officers*. US Army Medical Research and Development Command: Fort Detrick, Md; 1994: 4. USARMIEM Technical Note 94-2.

sis of the acute hyperventilatory response inhibits hyperventilation somewhat and does not allow the maximum compensation that ventilation could achieve.

If exposure continues, further physiological adaptation occurs during the next 2 to 4 days. First, the inhibitory effect of alkalosis and hypocarbia on ventilation moderates and permits some further increase in hyperventilation. This increases alveolar oxygen tension and oxygen delivery to the blood. Second, a diuresis develops that reduces blood volume and increases hematocrit. This improves the oxygen-carrying capacity of blood and permits some moderation of the tachycardia of acute exposure. During this time, the kidneys have responded to hypoxia by increasing the secretion of erythropoietin. Its stimulatory effect on the red blood cell mass and hematocrit will not become apparent, however, unless exposure continues for several weeks.

There is some evidence for additional adaptations at the organ and cellular level to chronic hypoxia. Changes have been reported in capillary and mitochondrial density, adaptation of aerobic pathways, and myoglobin concentration. The significance of any of these changes to altitude adaptation is not well understood.

Effects of Hypobaric Hypoxia on Performance

Physical Performance

High altitude is well known to have significant effects on physical performance.[205,206] The oxygen requirement of activity at altitude is the same as at sea level. However, since the oxygen available with each breath at altitude is reduced and this deficit is incompletely compensated for by hyperventilation, minute ventilation must be higher relative to that at lower altitude to provide sufficient oxygen for any given activity. The increase in ventilation is perceived as breathlessness, even at relatively light workloads, and produces early fatigue. Hypoxia also limits the oxygen delivery at maximum ventilation, so maximal aerobic exercise capacity is reduced proportionally as altitude increases.

Acclimatization improves exercise tolerance primarily through the enhanced oxygen carrying capacity of blood due to the elevated hematocrit. Consequently, oxygen transport to sustain any given amount of activity can be accomplished with less cardiovascular strain as acclimatization progresses. However, acclimatization does not reduce the amount of oxygen required for a particular task or the requirement for increased ventilation.

Psychological Performance

Altitude exposure also has well-known psychological effects, which are of consequence to the conduct of military operations and to the prevention of accident and injury. Night vision is impaired, even at relatively modest altitudes (1,500 m [5,000 ft]).[207–209] Cognitive changes have been found at these relatively low altitudes, although the changes were subtle. Changes that are more evident occur at higher altitudes.[210] Altitude exposure slows learning of complex mental tasks and fine psychomotor performance.[211] Individuals compensate for these changes by slowing performance to preserve accuracy. Mood and judgement are impaired on acute exposure to higher altitudes (> 3,000 m [10,000 ft]), and this is often manifested as a dangerous euphoria or indifference to danger.

Sleep disturbance and sleep loss are common at altitude.[212–218] Sleep loss will aggravate mood and cognitive changes due to hypoxia. The normal reduction of minute ventilation that occurs during sleep aggravates hypoxia and its effects on sleep.[219–221] Typically, as ventilation falls with the onset of sleep, hypoxia worsens and causes reawakening. The cycle repeats throughout the sleep period, and adequate sleep becomes difficult to obtain. Furthermore, the symptoms of acute mountain sickness (eg, headache, nausea, breathlessness) will aggravate the sleeplessness caused by hypoxia. Sleep deprivation is an important additional factor reducing psychological performance at altitude.

PREVENTION OF HIGH ALTITUDE ILLNESSES

The classic acute high altitude illnesses include acute mountain sickness (AMS), high altitude cerebral edema (HACE), and high altitude pulmonary edema (HAPE). They share a common etiology in hypoxia and probably share a common pathophysiological mechanism involving a breakdown in the regulation of water and electrolyte movement across capillar-tissue interfaces.[222]

Primary prevention of all the classic illnesses of high terrestrial altitude follows a common strategy, which includes preexposure screening, gradual exposure to hypoxia to allow time for acclimatization, use of prophylactic drugs, and control of activities such as overexertion that increase risk.

Mitigating Hypoxic Stress

The risk of high altitude illness is directly related to the rate of exposure to hypoxic stress. The principle factor is rapid ascent to unaccustomed altitudes. Mild symptoms of altitude illness appear in individuals from sea level at altitudes as low as 2,200 m (7,000 ft). Almost everyone exposed acutely to altitudes in excess of 3,500 m (11,000 ft) will become frankly ill with AMS after a number of hours.[223] Gradual exposure to hypoxia will allow time for altitude tolerance to develop and reduce the risk of illness. Several guidelines for managing exposure have been offered: no faster than 300 m (1,000 ft) per day at altitudes over 2,700 m (9,000 ft) with a rest every 2 to 3 days or a rest day at 2,400 m (8,000 ft), then a rest day for every 600 m of further ascent. In any case, no one with AMS symptoms should ascend further until well.

Maintaining Altitude Tolerance

Some individuals can be predicted to be at risk of serious illness on exposure to hypoxia at altitude. These include those with previous episodes of HAPE, HACE, and heterozygous or homozygous S hemoglobinopathy.[224,225] In addition, the appropriateness of exposing to altitude any individual with a condition that can be worsened by hypoxia (eg, coronary or cerebrovascular insufficiency, pulmonary disease)[226,227] should be carefully considered.

Physical exertion during the first few days of altitude exposure increases the risk of HAPE. Exertion increases pulmonary blood flow and pressure, which synergizes with the reduced integrity of the pulmonary capillary-alveolar interface to permit the increased flow of water and electrolytes into the alveolar space, causing pulmonary edema. Primary prevention of HAPE requires the recognition of this association and planning work schedules to accommodate the risk. The roles of hydration, nutrition, and fitness in prevention of altitude illness are unclear but do not seem to be substantial.[228–233]

Technology

Drugs can be used for the prophylaxis of AMS. Since HACE is considered, at least in part, to evolve from AMS, then the use of these drugs is expected to reduce the risk of HACE as well. The principal prophylactic drug for AMS is acetazolamide.[223,234–240] It inhibits carbonic anhydrase and produces a mild metabolic acidosis. Its seems to stimulate respiration directly through medullary chemoreceptive neurons and to moderate respiratory inhibition by hypocapnic alkalosis. The experience of the US Army and Marine Corps in Exercise Fuertos Caminos 1990 in Bolivia indicated that troop populations that take acetazolamide will suffer less disability on altitude deployments.[241] Acetazolamide prophylaxis is appropriate for deployment to any altitude where AMS is a likely risk (> 2,700 m [9,000 ft]). It is begun 1 to 2 days before exposure and continued for 4 to 5 days, if exposure lasts that long. Other drugs that have been used successfully for prophylaxis include dexamethasone and spironolactone.[242–249] Coca leaf, used by residents of the altiplano in the Andes mountains, also appears effective in promoting altitude tolerance.

Nifedipine,[250–253] which reduces pulmonary artery pressure, is effective prophylaxis for HAPE among individuals who have had a previous episode. However, military personnel who have experienced HAPE should not be reexposed to altitude.

Technological means of increasing oxygen tension are effective in preventing the health and performance decrements of altitude exposure. Compressed oxygen cylinders are used by climbers at extreme altitudes but are impractical except for emergency medical treatment in most military circumstances. Fixed facilities can use oxygen concentrators[254] to increase ambient oxygen tension. Intraalveolar pressure and oxygen tension can be increased by portable hyperbaric chambers[255–257] or expiratory positive airway pressure masks.[258]

HIGH ALTITUDE ILLNESSES

Acute Mountain Sickness

This is the most serious operational threat to military operations at altitude.[259] It is common, and its incidence (up to 100%) increases with altitude and rate of ascent.[260] AMS can occur at any altitude over 2,500 m.[261] It can be profoundly debilitating and cost a unit suddenly deployed to altitude a significant proportion of its members.

AMS develops after several hours (usually 8–24) of altitude exposure and lasts 1 to 2 days. AMS does not develop in short exposures (less than 4 hours). It usually resolves spontaneously. The mechanism of AMS is unknown, but most think it is caused by a combination of increased cerebral blood flow, capillary permeability due to hypoxia, and water and sodium retention, which together cause subclinical cerebral edema and cerebrospinal fluid hypertension. Besides rate of ascent and altitude achieved, possible additional risk factors include young age and male sex. Those who have had AMS in a particular exposure may be at increased risk of a recurrence with reexposure. Prudent preventive medicine practice would respond to the potential risk by emphasizing the particular importance of acclimatization and prophylaxis in those individuals.

The manifestations of AMS include malaise, fatigue, irritability, sleep disturbance, frontal headache that is worse with exertion, anorexia, nausea, vomiting, photophobia, orthostatic vertigo, breathlessness even at rest, and peripheral dependent edema. Formal systems for scoring the severity of AMS have been developed.[262–265]

AMS can be treated by descent (which is curative), mild nonnarcotic analgesics, antiemetics, dexamethasone, supplemental oxygen, voluntary hyperventilation, 3% carbon dioxide, and portable hyperbaric chambers.[223,245,256,266,267] An episode of AMS does not preclude reexposure to altitude but suggests gradual ascent and prophylaxis are advisable.

High Altitude Cerebral Edema

HACE usually evolves from AMS when subclinical cerebral edema becomes clinically manifest with frank changes in cerebral function.[268–270] Occasionally, HACE will appear 2 to 3 weeks after arriving at altitude. HACE will not spontaneously resolve and will be lethal unless successfully treated. It is relatively uncommon (about 1% of altitude exposures). One study[260] demonstrated an incidence of 1.8% among people trekking to 4,200 m (14,000 ft), equivalent to the altitude at the summit of Pikes Peak.

Typically, casualties have symptoms of AMS that evolve over 1 to 3 days to increasing deterioration in mental status, including thought disorders, hallucinations, ataxia, obtundation, and coma with signs of intracranial hypertension.

HACE requires immediate evacuation from altitude exposure. Emergency therapies to use while awaiting evacuation include use of oxygen, a portable hyperbaric chamber, dexamethasone, and diuresis. Severely affected casualties may not respond to treatment even after descent. An episode of HACE should preclude future altitude exposure.

High Altitude Pulmonary Edema

HAPE is a noncardiogenic pulmonary edema probably caused by a combination of high pulmonary vascular flow and vascular leak due to the reduced integrity of the pulmonary capillary-alveolar interface[253,271–274] (Figure 19-16). HAPE worsens hypoxia by interfering with oxygen exchange

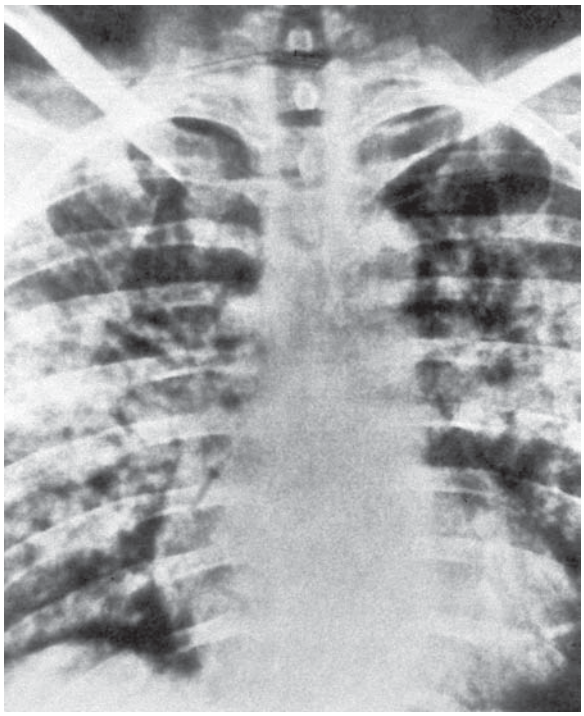

Fig. 19-16. A radiograph of acute high altitude pulmonary edema.
Photograph: Courtesy of Colonel Paul Rock, Medical Corps, US Army, USARIEM, Natick, Mass.

EXHIBIT 19-10

HIGH ALTITUDE PULMONARY EDEMA AND EXERCISE FUERTOS CAMINOS IN BOLIVIA, 1990

- 364 US soldiers and Marines deployed to Potosi, Bolivia (4,200 m [13,800 ft])
- No Marines taking acetazolamide had significant HAPE
- 14 cases of HAPE among Army troops (3.8%)
- Most treated with rest, supplemental oxygen, diuresis
- 5 treated in Gamov Bag
  - 1 primary failure: evacuated
  - 4 had symptomatic relief in 20 minutes
  - 2 secondary failures (relapse): evacuated
- Three evacuations (0.8%)

and introduces a vicious cycle of hypoxia worsening edema and edema worsening hypoxia. The incidence of HAPE, like that of AMS and HACE, depends on the intensity of the hypoxic exposure. In the 1990 Fuertos Caminos exercise at 4,200 m, it was 3.8% (Exhibit 19-10). The risk is substantially increased by exertion,[275] particularly in the first 3 to 4 days of altitude exposure, but physical exertion is not required to precipitate HAPE.[276]

The manifestations of HAPE include breathlessness, cyanosis, orthopnea, rales, chest pain, cough, frothy sputum, hemoptysis, and tachycardia. Fever is occasionally present. An individual with AMS or HACE can also develop HAPE; this complicates the management of the casualty.

Mild degrees of HAPE can be resolved with treatment at altitude,[277] but it can be lethal and is generally more fulminant than HACE. Severe cases die within a few hours of presentation if not treated. Treatment includes descent, portable hyperbaric chambers,[266] (Figure 19-17) oxygen, nifedipine and other pulmonary vasodilators,[252,278] expiratory positive airway pressure,[258] and dexamethasone. With proper treatment, clinical recovery is usually rapid, but severe acute respiratory distress syndrome that requires assisted ventilation can occur. Subtle abnormalities in pulmonary function can persist for a number of weeks after recovery.[279] Although nifedipine can be used for prophylaxis against recurrence of HAPE, an episode of HAPE should preclude future altitude deployment for the service member.

Other Medical Issues at Altitude

Thromboembolic Disease

Thromboembolic disease is more common at altitude.[280–283] Its increased incidence seems related to hemoconcentration, dehydration, alteration in clotting mechanisms, and enforced inactivity during bad weather. The types of thromboembolic disease include (*a*) thrombosis of the deep veins of the legs complicated by pulmonary embolism (which may present like HAPE[284]) and cerebral thrombosis and (*b*) stroke. Although epidemiological data are very sparse, the risk seems small at moderate altitudes (< 3,000 m) and increases with the duration and intensity of altitude exposure.

High Altitude Retinal Hemorrhage

High altitude retinal hemorrhage (HARH) is a usually benign condition with startling ophthalmoscopic findings. HARH is caused by hemorrhage from retinal vessels that dilate in response to hypoxia.[285,286] The hemorrhages can interfere with vision if they involve the macula. One case of central retinal vein occlusion with marked visual loss and retinal hemorrhage has been described.[287] As a

Fig. 19-17. A portable hyperbaric chamber (a Gamov bag) used to treat high altitude pulmonary edema during Operation Fuertos Caminos in Potosi, Bolivia.
Photograph: Courtesy of COL Eugene Iwanyk, Medical Corps, US Army, USARIEM, Natick, Mass.

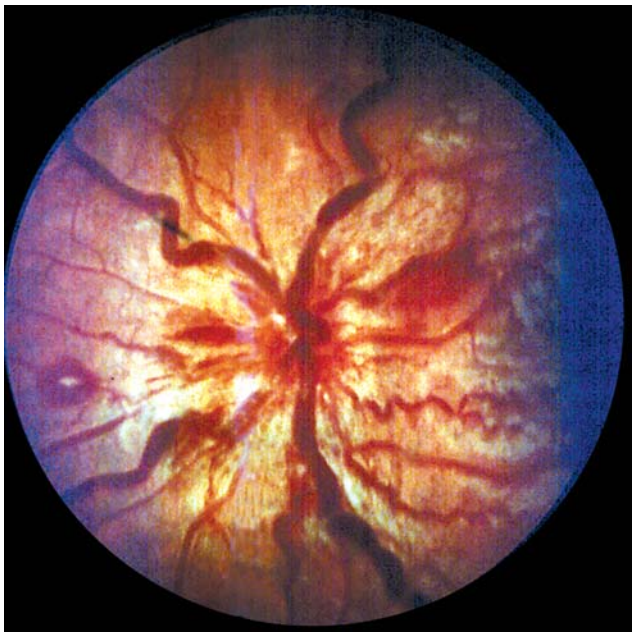

Fig. 19-18. A photograph of a radiograph of high altitude retinal hemorrhage.
Photograph: Courtesy of COL Paul Rock, Medical Corps, US Army, USARIEM, Natick, Mass.

rule, HAPH resolves spontaneously on return to lower altitude, with no permanent visual effects (Figure 19-18).

Chronic Mountain Sickness

Chronic mountain sickness (Monge's disease) appears in some individuals after long residence at altitude.[288] It is rare below 3,000 m. It manifests as extreme secondary polycythemia and hyperviscosity, cor pulmonale, and reduced exercise tolerance. Individuals who are affected either have some additional pulmonary risk factor (eg, smoking, pneumoconiosis) or an abnormality in the regulation of ventilation that aggravates altitude hypoxia (eg, sleep apnea). Premenopausal women are almost never affected, presumably due to the stimulatory effects of progesterone on ventilation.[289] Treatment is directed at correcting hypoxia or polycythemia by relocating to lower altitude, oxygen therapy, respiratory stimulants (medroxyprogesterone), or venesection.

Exposure to Extreme Altitudes

Residence at altitudes over 5,100 m (17,000 ft) leads to a gradual physical and mental deterioration in addition to the debilitating effects of dehydration, fatigue, and weight loss.[290–293] The mechanism is unknown but may be a direct consequence of chronic hypoxia. Descent is required to ameliorate the process. Several studies have shown that exposure at these extreme altitudes has demonstrable neuropsychological sequelae.[294,295]

SUMMARY

Soldiers and campaigns have been victims of hot, cold and high altitude environments for as long as civilizations have conducted military campaigns. These three environments are encountered in all parts of the world and are still major obstacles to the successful conduct of military operations and significant causes of casualties. Casualties are due to the additive effects of climate and the entire suite of physiological and psychological stresses encountered in military operations.

There is a common pathophysiologic mechanism of illness and injury, which, if understood, permits a broader and more effective approach to preservation of unit effectiveness and prevention of casualties in the field. The risk of disease and injury is a strong correlate of the physiological and psychological strain experienced by deployed service members. Any measures to mitigate that strain will reduce the risk of illness and injury.

Hot environments expose service members to the threats of high ambient temperature and humidity, dehydration, sunburn, and acute heat illnesses, among others. Mitigation of these threats requires attention to heat and sun exposure by implementation of work schedule controls and technologies to reduce heat strain. In addition to direct approaches to moderating heat strain, heat tolerance can be increased by provision of adequate nutrition, rest, and other measures to optimize physiological and psychological hygiene. Service members are at risk for a wide spectrum of heat illnesses, from heat exhaustion to life-threatening conditions such as heat stroke or rhabdomyolysis. Experience has shown that most of these conditions are preventable, but medical officers still need to be prepared to manage them competently if they occur.

Cold environments expose service members to low ambient temperatures, wet conditions, long nights, intense ultraviolet radiation, difficult terrain, toxic exposure from carbon monoxide, and the burden of cold-weather clothing. Unlike hot environments, where a reduced pace of operations reduces risk, in the cold, passivity is dangerous. Consequently, effective mitigation of the threat is challenging and depends on careful planning, robust logistics, and well-trained and experienced

personnel. Cold injuries are generally insidious and epidemic. They are disabling and slow to heal, usually requiring evacuation for clinical care and recovery.

High altitude environments are infrequent settings for US operations and training. In addition to their dominant environmental characteristic, hypobaric hypoxia, they present threats of heat, cold, rough terrain, dehydration, and cognitive dysfunction. The most common altitude illness, acute mountain sickness, can disable an entire unit soon after deployment to altitude. The other two altitude illnesses, pulmonary edema and cerebral edema, are fatal if not successfully managed.

Because of their ubiquity, potential for causing casualties, and susceptibility to intelligent intervention, environmental stresses must be a core part of the skill and knowledge repertoire of medical officers.

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