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MEDICAL ASPECTS OF COLD WEATHER OPERATIONS: A Handbook for Medical Officers

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Preface

This handbook is one of a series prepared at the U.S. Army Research Institute of Environmental Medicine (USARIEM) for medical officers supporting units operating and training in harsh climatic conditions. It is intended for health care personnel as a review of those aspects of military medicine important for cold weather operations.

In addition to this medical handbook, USARIEM has prepared another: Sustaining Health and Performance in the Cold: Environmental Medicine Guidance for Cold Weather Operations intended for all personnel, medical and non-medical, who may have to deploy to cold regions. This handbook is available in two formats, 8.5 *x* 11 (USARIEM Technical Note 92-2, DTIC No. AD A254-328) and pocket size USARIEM Technical Note 93-2, DTIC No. AD A259-920) . The two handbooks are complementary We believe medical personnel will find useful information in both.

Most current practice in the management of illness and injury due to cold exposure depends on limited clinical series and personal anecdote. It has little basis in modern clinical investigation. To address the need for new contributions, we welcome advice, anecdote, opinion, correction and clarification.

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The material in this handbook should not be taken to be doctrinal guidance of the Department of Defense, the U.S. Army or the U.S. Army Medical Department, unless so designated by appropriate authority.

Introduction

Of all the natural environmental hazards soldiers confront in the course of their duties, cold is arguably the most difficult and dangerous. Every medical officer has heard of the catastrophic withdrawal of Napoleon' s Grand Army from Moscow in the winter of 1807 during which illness and injury due to cold caused the death of thousands of French troops.

Casualties from cold exposure occur in all types of operations. Cold is an effective weapon on the side of a military force with initiative. Forces including the U.S. Cavalry, the Russians and the Finns used cold in this way by displacing their opponent from shelter and allowing the environment itself to force surrender. In the offense, rapid paced operations 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 U.S. Army suffered cold weather casualties in almost all of our conflicts from the American Revolution to Korea. Cold injuries remain a problem in military operations and training exercises today (Barat, A. K., Puri, H.C., Ray, N., 1978; Marsh. A.R., 1983; Orr, K.D. and Fainer, D., 1952; Tek, D. and Mackey, S., 1993).

Because the U.S. Army is evolving into a CONUS-based rapid deployment force, U. S. ARMY Medical Department officers must be prepared to assist commanders to deal with any deployment contingency. Many cold regions throughout the world are potential deployment destinations for the U.S. Army. This handbook was written to help medical officers prepare for, and manage the medical needs of units confronted by deployment and operations in cold environments.

This handbook is divided into four sections. The first section is a review of human physiologic and pathophysiologic reactions to cold. The second is an approach to the prevention of illness and injury during operations in the cold. The third is a clinical review, from the military medical perspective, of the presentation and management of the various forms of illness and injury caused by cold exposure. The fourth sect ion summarizes particularly important or useful points to allow easy extraction for training.

Section 1.

Physiologic Effects of Cold Exposure

1.1 Heat Balance and Core Temperature

Humans require that the body core temperature stay within narrow limits for optimum functioning. Even small (2-3^{0}F) deviations from the average 99^{0}F (37^{0}C) temperature will impair physical and mental performance.

Core temperature stability is maintained by balancing heat gain from the environment and metabolism with heat loss. 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 following way: **S=M±R±C-E**

where: **S. =**net change in heat content **. .**

M = metabolic heat production; always positive.

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 80-90 Cal per hour (roughly equivalent to a 100 W bulb). Maximum aerobic exercise increases metabolic heat production to about 10 times the resting rate (about 1000 Cal per hour) and can be sustained by most individuals for only brief periods. Individuals performing sustained hard physical work (digging, marching under a load) who can control the rate of exertion will usually work at about 4-5 times the resting metabolic rate. For comparison, shivering can increase metabolic rate up to six 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 we

sense standing in direct sun or near a hot surface is produced by radiative heat gain. As “warm-blooded” mammals in a cold environment we are relatively warm objects, 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 due to physical contact between two materials of different temperatures. Conductive heat transfer occurs when one material warms another adjacent material. Conductive bodily heat gain or loss is particularly significant when lying on hot or cold ground. If one of the materials is a fluid (water or air), then convection can occur which moves the warmed fluid away and replaces it with unwarmed fluid sustaining the temperature differential and conductive cooling. Wind increases convection and is responsible for the greater apparent coldness of moving air. Convection is also an important component of cooling during cold water immersion

Evaporation (E) of sweat causes heat loss. Each liter of sweat evaporated transfers 540 Cal to the environment. Sweat evaporation does NOT depend on the relative humidity, but rather upon 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 that has been warmed by contact with skin. Sweating in the cold is an important form of heat loss.

1.2 The Acute Response to 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.

When the body is exposed to cold, blood is diverted away from 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 light business suit. In contrast, heavy arctic clothing provides six to eight times as much insulation.

The diversion of blood away from the skin and extremities is caused by arteriolar and venous vasoconstriction mediated by both cutaneous and central temperature responsive reflexes. Direct cooling of the skin or the core causes generalized cutaneous vasoconstriction. Central and peripheral receptors act synergistically, so, the degree of vasoconstriction depends on both skin and core body temperature. The lower the core temperature, the more intense and generalized the vasoconstriction.

Skin cold sensory receptors respond both to 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. (Who hasn't experienced the brief frisson of shivering when going out into the cold?) As cold exposure continues, the skin equilibrates at a 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 two to three hours) accommodates to the cold and the sensation of cold becomes less uncomfortable. Conversely, if cold skin is warmed, the reflex response can produce moderation of the insulating vasoconstrictive response and increase heat loss from skin and extremities.

As a consequence of reducing blood flow and volume in skin and extremities, peripheral vasoconstriction causes an expansion in central blood volume. Although the effect is antagonized by upright posture, the expanded central volume can produce "cold diuresis." Cold diuresis can contribute to dehydration during prolonged cold exposure, and particularly, to hypovolemia in victims of accidental hypothermia and immersion.

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 twofold.

However, if temperature falls further, the muscular activity changes to cycles of contraction and relaxation, producing visible shivering. The core temperature at which maximal shivering develop s differs among individuals, but is almost always between 94 and 97^0F. Maximal shivering increases heat production to six times the resting level. Shivering, although an important mechanism of emergency metabolic heat production in the cold is sustained only at considerable cost. First, it disables by interfering with purposeful, coordinated muscular activity. Second, the increased muscular activity requires an increase in extremity blood flow, which diminishes the insulating effect of vasoconstriction.

1.3 Cold Induced Vasodilatation (CIVD)

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 included of the body at risk of severe cooling and injury. “Cold induced vasodilatation” is aphysio that appears to reduce the risk of injury physiologic mechanism are exposed to water below 50^0F and air below 32^0F.

As the hands or feet cool, vasocontriction initially reduces blood flow and volume. After a few minutes of low digital temperature, arteriovenous anastomoses in the distal phalanx open and allow a rapid increase in digital blood flow **(**bypassing the constricted precapillary arterioles). While the arteriovenous anastomoses remain open, the digits remain warm the phenomenon is usually cyclic, producing alternating periods of vasoconstriction and vasodilation 10-20 minutes long. Individuals vary in the magnitude of their CIVD response. Occasionally, individuals will sustain CIVD without cyclicity; other individuals have very limited CIVD and seem to have poor cold tolerance.

The magnitude and duration of the CIVD ace dependent on core temperature. When core temperature is low, the phenomenon is substantially less prominent and, presumably, affords less protection to the hands and feet from cold injury

Unless a fall in core temperature inhibits CIVD, it will increase heat loss, particularly in cold water. Loss rates oh 40 Cal per hour have been reported.

1.4 Human Adaptation to Cold.

Although apparently adaptive changes in metabolic rate core temperature, shivering threshold, vasoconstriction and CIVD have been demonstrated in experimental cold exposures, the magnitude of these effects is small. Physiological adaptation to cold is not a phenomenon of the same significance as adaptation to heat or high altitude. 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 appears to be no practical means of significantly enhancing physiological cold tolerance through training or predeployment exercises.

Some component of the cold tolerance seen in soldiers during cold weather exercises may be due to physiologic change, but familiarization and skill-development are at least as important to maintaining their well being.

Section 2.

Prevention of Illness and Injury in the Cold

2.1 Preventing Cold Injuries: Role of the Medical Officer.

Commanders are responsible for the prevention of cold injury in their units. They take this responsibility seriously, but need constructive advice from their medical staff to develop effective preventive measures compatible with mission accomplishment. Commanders, not medical officers, control the exposure of soldiers to cold. The principal role of the medical officer in the prevention of injury is to advise his/her commander and unit, train unit leaders and members, and conduct injury surveillance.

2.2 Advice for the Commander.

During operations planning, medical officers have the wide-ranging responsibility of preparing analyses, recommendations and plans for the entire spectrum of health service support. Part of that responsibility consists of considering the risks due to the environment, recommending countermeasures to reduce the risk of environmental injury and preserving the health and performance of the unit.

It is useful to analyze the risk of environmental injury as an interaction of three components: the soldier, the environment and the mission. Each component has unique risk factors, which must be assessed and controlled.

A careful consideration of the three risk components of environmental injury will assist the medical officer in the preparation of the health service estimate which should contain recommendations and plans to reduce injury risk. Since most of the components of risk can be modified, explicit consideration of each risk component will allow the implementation of countermeasures to preserve the health of the unit.

2.2.1 Soldier Related Risk Factors

Characteristics of soldiers that are generally accepted to be risk factors for cold injury include: fatigue, dehydration, undernutrition, lack of cold weather training and experience, lower rank, black race and tobacco use. The degree to which these factors are present in a unit will vary depending on the composition and circumstances of the unit. The medical officer must be completely familiar with the unit and monitor it carefully in order to judge when particular risk factors are of sufficient magnitude to require control measures.

Some of the soldier-related risk factors for cold injury are common consequences of operating in the field. Soldier fatigue, undernutrition and dehydration are encountered in every deployment. These risk factors are controlled by ensuring command attention to scheduled rest periods, complete consumption of rations and enforced hydration. Nutrition and hydration are considered in more detail in 2.5.2 and 2.5.3.

With all the other training and readiness demands, units that do not routinely train in cold environments will have fewer opportunities to maintain or develop their cold-weather skills. Before operating in the cold, military units should plan to acquire appropriate skills and equipment. Units that train regularly in the cold should ensure that new members of the unit are given individual cold-weather training and equipment to allow them to function safely in the field.

Soldiers at particular risk of cold injury are: 1) those of lower rank who are exposed to cold most, 2) those with black skin, which seems more susceptible to cold injury than white skin and 3) those who use tobacco, which vasoconstrictors and may limit the protective vascular response to cold. Unit leaders must be aware of the increased risk to these individuals and monitor them during cold exposure.

2.2.2 Environmental Risk Factors

Cold environments are dangerous; however, with the one exception of the wind-chill Index, the risks have been hard to express quantitatively. The medical officer must remain aware of the current and forecast conditions to enable him to estimate risk of exposure and recommend countermeasures.

Cold exposure can occur on land, in water or in aircraft. Cold land environments are generally classified as either wet-cold or dry-cold. Wet-cold environments have ambient temperatures above freezing to about 60^0F (16^0C) with wetness ranging from fog to heavy rain. They are associated with non-freezing peripheral 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 (32^0F, 0^0C). Precipitation, if present, is in the form of dry snow. Dry-cold environments are associated with freezing

peripheral injuries, which can develop in a few minutes to hours. Aircrew exposed to the airstream around a flying aircraft through an open hatch or port in the fuselage risk severe dry-cold exposure and can incur serious freezing injuries in seconds.

Accidental hypothermia can occur in any cold environment, water, wet-cold or dry-cold.

A quantitative index of risk to exposed skin, the “Wind-Chill Index”, has been developed as an important tool for judging environmental risk. A Wind Chill Table is included in this handbook as Appendix A. It is important to remember the Wind Chill Index does not provide an index of thermic risk or of risk to covered skin. Effective cover will protect skin even in conditions of very “cold” wind-chill.

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 y warmer, but still cold, temperatures do not produce their usual sensation of cold.

2.2.3 Mission Related Risk Factors

The mission will determine the exposure of soldiers to the environment and is an important component of the environmental injury risk. Risk factors associated with the mission include: prolonged cold exposure, hasty preparation, limited resupply and inadequate equipment.

Mission-related operations should be planned to protect soldiers against environmental injury. In the cold, operational plans should provide for adequate resupply of water, food and dry clothing. Operational schedules must take into account the difficulties of movement in the cold and provide for adequate opportunities for rewarming, rest and foot care. The possibility of weather change should be anticipated and appropriate alternatives planned.

2.3 Training.

Successful injury prevention ultimately rests on the skill and knowledge of the soldiers conducting operations in cold environments. The medical officer is responsible for providing some of that skill and knowledge, which addresses the cause, prevention, recognition and first aid of cold injury. Other members of the unit will be responsible for providing training on

cold weather survival, use of equipment and unit procedures in the cold. The medical officer needs to provide his medical training for three groups in the unit: unit medics, unit leaders and the soldiers themselves.

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 the severity of injury and to reduce the number of soldiers affected. In addition to their usual medical supplies, their medical support plan should include adequate equipment to protect casualties from the cold during evacuation.

Unit leaders should understand the causes and manifestations of cold injury, both for their own benefit and that of their soldiers. They should understand that cold injury is insidious, that affected soldiers are often unaware of their injury and that cold can adversely affect performance. They should understand the importance of potable water, adequate rations, the “buddy” system and cold weather clothing on preserving unit function in the field. They should understand the importance of adequate predeployment preparation for cold by conducting cold weather training, cold weather clothing and equipment inspections and assuring appropriate health maintenance and medical screening measures .The USARIEM handbook “Sustaining Health and Performance in the Cold” is intended for small unit leaders and can be used both as a training aid and field guidance.

Leaders should understand the importance of adequate materiel support in cold weather operations. Functional cold weather clothing, particularly dry warm socks, cold weather footwear and underwear are essential for adequate cold weather protection. Opportunities for exchange or cleaning and drying socks and underwear are essential. A supply of cold weather boots must be available to replace immediately boots that become defective. If long intervals between resupply are anticipated adequate supplies of these items must be included in the initial unit issue.

The most important control measure for preventing cold injury during cold exposure is the provision of adequate time and facilities for rest, rewarming and inspection of the face and extremities. The interval between rest and 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.

Soldiers should know the signs and symptoms of cold injury in themselves and their buddy, and know what to do if they suspect a medical problem. They should be alert to the consequences of dehydration, undernutrition, fatigue and alcohol.

They should understand the importance and the techniques for foot care in the cold.

Effective techniques have been developed for preventing cold injury in the lower extremities. Soldiers should be trained in these techniques and leaders should understand the importance of assuring that they are carried out when soldiers are exposed to cold. They are summarized below:

1. Assure the best possible fit of the boots with heavy socks.

2. Keep the body as warm as possible; avoid chilling.

3. Remove boots and socks at least twice a day; three times is better. Wash, dry, massage and move the feet to restore circulation and feeling. Al low enough time and provide appropriate shelter to complete this task. After massaging and warming the 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.

4. 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.

5. 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.

6. In fixed positions, stand on rocks, boards or brush to keep the feet out of water and mud.

7. 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.

8. Watch carefully for numbness or tingling. These are early symptoms of injury. If these develop, immediately take measures to warm the feet.

9. Keep the clothing and footgear loose enough to permit easy circulation.

2.4 Medical Surveillance.

Medical surveillance of the unit is the third component of the medical officer’s role in preventing cold injuries. The goal of surveillance is 1) to identify risk factors for illness or

injury to permit preventive intervention and 2) detect illness and injury as early as possible to allow interruption of the disease process.

The medical officer should implement a formal medical surveillance plan tailored to the needs and mission of the unit. The surveillance is intended to monitor selected information about the health of the unit in order to detect emerging risk factors and early manifestations of illness and injury.

A medical surveillance plan to support a program of cold injury prevention starts before deployment by assuring that all members of the unit are optimally prepared for cold exposure. The plan should periodically assess the following medical data on all unit members: immunization status, past history of cold injury current coincident or cold-related illnesses. A medical record review is important to identify individuals' coincidental medical conditions that increase the risk of cold injury. Examples of these latter conditions are Raynaud's Disease or diabetes mellitus.

In the field, medical surveillance should assess frequently hydration, fatigue, nutrition, coincident illnesses and the intensity of cold exposure. Cold injuries should be reported immediately and the circumstances of the injury determined. Inspection of unit work schedules for adequate rest periods, morning urine checks for hydration, and inspect ion for discarded rations can provide evidence of fatigue dehydration or undernutrition.

Medical surveillance should be performed in an organized fashion by all members of the health service support team. Regular written reports are an essential part the discipline that successful medical surveillance requires. Without organized data collect ion, surveillance becomes anecdotal and loses a significant amount of its sensitivity for early detection and successful intervention.

2.5 Sustaining Health and Performance.

Predeployment immunization, nutrition and hydration are critically important to preserving health in cold environments. Each is reviewed in more detail below.

2.5.1 Medical Predeployment Preparation

The unit should have received appropriate immunization and prophylaxis. Influenza and tetanus vaccinations are particularly important preparations for cold weather operations. In some cases, some soldiers may benefit from pneumonia vaccination. If

the unit has been exposed to streptococcal infection, bicillin prophylaxis should be provided **.**

Unit members who develop conditions that increase their risk of cold injury should be formally evaluated for deployability. Medical conditions that are considered to increase the risk of cold injury are: a history of prior cold injury, Raynaud's phenomenon or Raynaud's disease, obstructive vasculopathies (venous and arterial), chronic pulmonary disease and peripheral neuropathies.

2.5.2 Nutrition

Maintaining nutrition is a difficult challenge in the field. Appetite and food intake decreases in the field, while energy requirements increase. Voluntary undereating for weight control, dehydration, cold rations and unpredictable meal schedules compound the problem. Consequently, weight losses of two to three pounds per week are common for the first few weeks of a cold weather deployment.

Countering the propensity to weight loss requires that soldiers eat sufficient rations to provide 4,000 to 5,000 Cal per day. Command emphasis on complete ration consumption is required. Providing warm rations (A's or B's) at least once a day and a warm drink at each meal will enhance food consumption. A modified MRE ration specifically tailored to support cold weather operations (Ration - Cold Weather) has been developed and, if available, should be used when units cannot be provided daily "A", "B" or "T" rations.

2.5.3 Hydration

Maintaining hydration is as difficult and important as maintaining food intake. A number of factors contribute to dehydration. In the field, soldiers exhibit voluntary dehydration, which is a moderate degree of dehydration (2% below normal) that seems insufficient to trigger thirst. In addition, despite thirst, soldiers may further restrict water intake to reduce the need for urination in the cold. Since most water is taken with meals and is related to the amount of food eaten, incomplete consumption of rations tends to reduce water intake. The logistic problems of providing potable liquid water in the cold often restrict supply. Even if water is freely available, it is often cold and unpalatable.

Water requirements in the cold are high. Cold air is dry and increases respiratory losses. High work rates required in cold weather operations increase water requirements. All this adds to the dehydration due to restricted water intake.

Soldiers require a minimum of four quarts of liquid per day to assure adequate hydration. The effort to supply water in a cold environment requires the same attention as water supply in the desert. Once adequate water is provided, unit leaders must ensure that it is consumed. Squad leaders should check urine color daily to detect dehydration early and to emphasize the commander's concern about adequate hydration. Command directed drinking may be required.

2.6 Medical Soldiers Are Role Models.

Soldiers will watch their medics to see if they put into practice what has been provided in the unit ' s cold weather medical training. The medical officer should assure that the medics understand that they will provide an important example to the rest of the unit and that their behavior can either reinforce or weaken the training which the unit has received in sound cold weather preventive practice.

2.7 Prevention of Illness and Injury from Other Hazards.

Cold environments present hazards other than that of cold exposure such as: icy surfaces, long hours of darkness, open flame heating equipment and heavy clothing. The medical officer must be as vigilant to these other hazards as to cold itself. Several of these hazards and measures to control them are briefly reviewed in this section.

2.7.1 Traumatic Injury and Falls

Wet or icy surfaces long hours of darkness and heavy clothing combine to make work and movement slow, difficult and uncertain and increase the risk of falling. Darkness and snow conceal trip and fall hazards during cross-country movement. Vehicles are more difficult to control in ice, snow and mud. Their direction of travel is less predictable for both driver and pedestrian increasing the risk of accident.

Control measures include 1) careful p reparation, maintenance and marking of paths, roads and load-handling areas, 2) separation of pedestrian and vehicular traffic, 3) one-way vehicular traffic, 4) extra help for work-details and 5) work-area illumination.

2.7.2 Carbon Monoxide, Fires and Fumes

Fuel-fired heaters are ubiquitous equipment in cold environments and create a risk of carbon monoxide (CO) poisoning, burns, eye injuries and tent fires. In the cold, motor vehicles are kept running for long periods, and are often close to troop

bivouacs. Soldiers sleeping in or near running vehicles are at risk of CO poisoning.

Control measures include unrelenting attention to training soldiers in the operation and maintenance of heaters, mandatory firewatch, placement of vehicle areas downwind from bivouacs and prohibition of sleeping in operating vehicles.

2.7.3 Crowding and Infection

Soldiers sleeping in close, poorly ventilated quarters provide an environment for the rapid dissemination of respiratory illness. Viral and streptococcal respiratory infections are particular hazards.

Control measures include appropriate predeployment immunization and prophylaxis, early detection of respiratory infection by examination and culture, head-to-toe sleeping and adequate ventilation.

2.7.4 Sun Exposure

Sunlight, particularly when reflected of f snow and ice, can produce serious burns to exposed skin and eyes from its ultraviolet component. The risk of burns is increased in moderate weather or at high altitude because the warming effect of the sun is particularly noticeable and inviting in these circumstances.

Solar keratitis (snow blindness) is profoundly disabling. Affected soldiers' vision is blocked or restricted for two or three days by required protective eye covering and eye pain may produce substantial discomfort. They may not be able to tolerate full sun exposure for several days after the injury.

Sunburn is not a minor problem! It can prevent the wearing of cold weather uniform items, and consequently, significantly limit availability for normal duty.

Control measures include clothing and sunscreen to protect skin, and sunglasses to protect the eyes. If sunglasses are not available, eye covering (e.g., tape-covered eyeglasses) with narrow horizontal slits provide adequate field expedient eye protection.

2.7.5 Contact Freezing Injury

In the cold, all materials in the environment should be considered to be at the ambient temperature. If cold materiel comes in contact with unprotected skin, it can produce local frostbite in seconds. Particular hazards are: 1) vehicles (door handles, steering wheels), 2) metal surfaces (utility poles,

armored vehicles, weapons), 3) POL, antifreeze and alcohol (these can be liquid at temperatures many degrees below zero. Splashes on skin or mucous membrane can produce immediate severe injury) and 4) ice and snow.

2.7.6 Alcohol

Alcohol consumption increases the risk of all forms of illness and injury in the cold. It impairs judgement and coordination, which increases the risk of traumatic injury. Its hypnotic effect reduces the alertness to fire and carbon monoxide poisoning. Alcohol 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 arid inhibited gluconeogenesis.

There are no known beneficial effects of alcohol in the prevention or management of cold injury.

2.8 BE ALERT!

Responses to operational contingencies will generate new hazards. For example e, during one winter exercise, when unexpected subfreezing temperatures developed, soldiers training to conduct mass decontamination responded by preparing a field-expedient freeze-resistant shower solution by adding ANTIFREEZE. Only the last minute recognition that this shower solution was now actually below freezing itself prevented a large number of acute cold injuries.

Section 3.

Illness and Injury Due to Cold

3.1 Introduction.

The following sections describe injuries and illnesses due to cold exposure. Sections 3.2, 3.3 and 3.4 follow the traditional classification of these conditions into peripheral injuries of the freezing (3.2) and non-freezing types (3.3) and the systemic injury of accidental hypothermia (3.4). Section 3.5 discusses a variety of other medical conditions that commonly occur during military operations or training in the cold.

3.2 Freezing Injury (Frostbite).

Frostbite is currently the most common cold-induced injury encountered in the U.S. Army. It is endemic at a low rate in cold environments. Isolated episodes are usually associated with an episode of carelessness or sudden weather change. Clusters of frostbite injuries occur in exercises and operations, frequently the result of poor planning or inattention to control measures. Fortunately, most frostbite injuries occurring during training are of low degree of severity and do not usually result in permanent tissue loss. However, the prolonged recovery usually means the loss of the injured soldier 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.

Remember that freezing injuries are preventable. Prevention is a command responsibility that depends on advice, training and surveillance by unit medical personnel (see Section 2).

Most freezing injuries occur in the field, consequently, the medical officer is often the last person to see and manage frostbite injuries. Most freezing injuries will be recognized and initially managed by unit medics and other non-physician medical providers. Because the outcome of the frostbite depends on early detection and management, the medical officer must assure that the unit medics are well trained in the recognition and management of these injuries.

3.2.1 Pathogenesis of Freezing Injuries

Frostbite injury results when tissue is cooled sufficiently to freeze. Tissues with large surface to mass ratios (ears) or with restricted circulation (hands and feet) are particularly susceptible to freezing. However, any tissue exposed to severe cold can freeze.

Tissue does not freeze at 32^0F; the high concentration of electrolytes and other solutes prevents freezing until tissue is cooled below 28^0F. 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, upon restoration of blood flow, hyperfusion tissue injury occurs. Second, marked endothelial swelling develops in the thawed tissue causing secondary loss of perfusion, ischemia and infarction of tissue. Despite freezing and reperperfusion injury, some frostbitten tissue is able to recover. However, refreezing of injured tissue causes irredeemable injury, a phenomenon used therapeutically in cryosurgery.

Pigmented cells seem to have greater susceptibility to freezing than non-pigmented cells. The mechanism is not known but may be related to lower cytoskeletal tolerance to cold in pigmented cells (Post, P.W., Daniels, P. Jr. and Binford R.T. Jr., 1975). To some extent, the higher incidence of freezing injury among Afro-American soldiers may be explained by this phenomenon.

3.2.2 Clinical Manifestations and Classification

Initially, all frozen tissue has the same appearance: cold, hard and bloodless. Except in minor and severe cases, the degree of injury will usually not become clear for 24-72 hours. Most significant injuries include areas with different degrees of significant injuries include areas with different degrees of frostbite, usually more severe distally. Digits, ears and exposed facial skin are the most commonly used injured areas.

Frostbite is classified by depth of injury into four degrees of severity. The depth of the injury depends on the duration and intensity of the cold exposure. Very intense cold for a few seconds will produce a superficial injury whereas prolonged moderate freezing 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 (e.g., 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 7-10 days.

Second degree frostbite involves the whole epidermis and may also affect superficial dermis. The initial frozen appearance is the same as 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 nobility and appearance of pain in affected areas. A bulla, with clear fluid, forms in the injured area over several hours after thawing. The blister fluid is extravasated from the dermis. Usually, the upper layers of dermis are preserved which permits rapid re-epithelialization after injury. Second degree injuries produce no permanent tissue loss. Healing is complete but takes three to four weeks. Some amount of first degree injury is frequently present in the immediate vicinity of second-degree frostbite. Frostbite should be looked for on all other exposed areas. Following second degree frostbite, cold sensitivity may persist in the injured area.

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

Fourth degree frostbite involves the full thickness of the skin and underlying tissues, even including bone. Initially the frozen tissue has no nobility. Thawing restores passive nobility, but intrinsic muscle function is lost. Skin reperfusion after thawing is poor. Bullae 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.

Corneal frostbite is a rare, but profoundly disabling injury. The evolution is similar to any deep ocular keratitis. Permanent corneal opacification requiring corneal transplant is a common outcome.

3.2.3 First Aid and Field Management

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

When frostbite is suspected, the injured area must be immediately protected from further cold exposure and trauma by the best available means. After protection is assured, a decision can be made regarding the method of warming.

It is important to remember that active warming of frozen tissue should be deferred until there is no risk that the injured tissue can be reexposed to freezing cold. Because refreezing of a frostbite injury aggravates the injury so severely, current practice recommends that frozen parts not be actively rewarmed until protection from refreezing can be assured. This recommendation should not be interpreted to mean that the injured part should be deliberately kept frozen by packing in snow or continued cold exposure. To do so would extend the injury to adjacent tissue. Rather, expeditious evacuation and protection of the casualty from further cold exposure is required. If adequate shelter and wraps are available, passive rewarming will occur during a long evacuation. Once tissue has thawed, it is absolutely essential that it be protected from reexposure to cold.

If refreezing can be prevented during evacuation, then frozen tissue can be immediately warmed by contact with warm skin. The groin or axilla are particularly effective areas for warming frozen tissue.

It is also important to remember that since the injured tissue is as vulnerable to heat as to cold, whatever emergency technique is used for warming, the tissue must not be exposed to temperatures in excess of 102-103^{0}F, which will aggravate the injury. Exposure to motor engine manifolds or exhaust, hot water, open flames, stove tops, or incandescent bulbs is particularly dangerous. Many frostbite injuries have been substantially worsened by exposure to inappropriate warming techniques.

Frostbitten tissue is vulnerable to trauma and should be carefully protected from physical injury during evacuation. In desperate circumstances, frostbite casualties may need to walk on frozen feet. In these circumstances, any available means should be used to prevent additional injury to the frozen tissue (e.g., extra wrapping, crutches or buddy assistance).

Victims of frostbite often have other, coincident medical problems (e.g., hypothermia, traumatic injury, dehydration and hypovolemia). To be sure other problems are found and managed, every frostbite casualty must be thoroughly examined and evaluated.

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

3.2.4 Hospital Management

At the Medical Treatment Facility (MTF), management includes warming of still-frozen tissue, treatment of various phases, of the injury as it evolves, and evaluation for coincident injury and illness. Frostbite injuries require meticulous record keeping, which should include careful and complete descriptions of the circumstances of the injury, its prehospital management, its extent, and all phases of its management. This documentation should begin from the first contact with the casualty.

If tissue is still anesthetic and cold upon arrival at the MTF active warming is usually appropriate. If the tissue has already thawed on arrival at the MTF, additional active warming should not be done.

Digits or entire hands or feet can be warmed in a temperature monitored water bath kept at 102-105^{0}F. Facial tissue or the ears can be warmed by towels kept wet with water warmed to 102-105^{0}F. Warming should be continued until no further improvement in the return of circulation and mobility is noted. This usually requires 15 to 45 minutes depending on the initial temperature and size of the injured part. After warming, the frostbitten tissue should be carefully and atraumatically dried, completely covered in bulky dry dressings, and kept slightly elevated to moderate swelling.

In addition to the immediate local treatment of the injured part, a complete examination of the casualty is required to detect other frozen tissue (remember, while below 50^{0}F these areas are anesthetic), other traumatic injuries, hypovolemia, and hypothermia. Temperature should always be taken using a low reading thermometer at a “core” (rectal or esophageal) site. Since dehydration and hypovolemia are so common with significant cold injuries and cold exposure, IV access should be established early and rehydration begun.

After whatever stabilization is necessary and warming has begun, early management includes: tetanus prophylaxis

as appropriate, and analgesics. Frostbite is a tetanus-prone wound. If immunization status is unknown or incomplete, both tetanus toxoid and tetanus immune globulin should be given. If tetanus immunization has been completed but no more than five years have passed since the last “booster”, only the toxoid should be given. During warming, pain appears and is often intense. Analgesia should be provided with NSAIDs and narcotics as needed. In addition to pain control, NSAIDs are believed by scene to reduce the release of eicosanoids that may aggravate the post-injury ischemia. Ibuprofen is the NSAID commonly used.

Prophylactic antibiotics are frequently used as a routine part of early management. Because wound anaerobes and streptococci appear to be early causes of post-injury infection, penicillin is recommended (2-4 M units IV OD or 500 mg po QID). For the penicillin allergic, clindamycin is an alternative. There is no consensus on the appropriate duration of prophylactic antibiotic treatment. Courses longer than a few days clearly carry the risk of selecting resistant organisms.

The continuing care of frostbite 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 injury must be absolutely protected against exposure to excessive cold or heat and from physical trauma. When the casualty does not have adequate housing or warm transportation, hospitalizations may be required for injuries involving only small amounts of tissue. In the case of third or fourth degree injuries, particularly in cold climates, months of hospitalization may be needed to provide appropriate care and protection of the injury.

In second, third and fourth degree injuries, devitalized tissue is usually removed by whirlpool debridement once or twice daily in skin temperature saline or dilute betadine. During whirlpool treatment, gentle active range of motion exercises are done to minimize functional loss. Careful atraumatic drying and redressing is done after each whirlpool treatment. Some centers have begun to apply aloe vera ointments to the surface of bullae in an attempt to reduce the production of vasoconstrictive eicosanoids and facilitate healing.

In injuries that appear severe, radioisotope scanning of bone or soft tissue perfusion three to five days post-injury will provide some evidence of the extent to which the deep tissues are involved and may allow earlier prognostic judgements. Digital subtract ion angiography has been used as well, but it is not clear that it offers sufficient additional information to offset its increased risk relative to scanning. In any case, since current practice permits the injury to evolve with minimal intervention, these diagnostic techniques would not be expected to influence management or hospital stay.

Occasionally, significant arterial vasospasm may be present in the first several days after injury. As a rule, use of tobacco is prohibited during healing of an cold injury to avoid any vasoconstriction due to nicotine. Alpha-blockade has been reported anecdotally to reduce post- injury vasospasm. Based on this experience, when vasospasm seems to be 1imiting perfusion after injury, a trial of alpha-blockade should be considered. The vascular response should be monitored with objective techniques such as skin temperature or Doppler flow measurements. The dose can be titrated upward to the appearance of significant non-cutaneous side effects such as orthostatic hypotension or nasal stuffiness. If alpha-blockade appears to enhance flow, it should be continued in the minimally effective dose for several days and, then, tapered over one to two days while watching for return of vasospasm. The practice of early sypathectomy or intraarterial injection of vasodilators has largely disappeared.

Heparin and aspirin have both been used in attempts to prevent microthrombi in injured arterioles without notable success. Neither measure is used routinely in current practice. One anecdotal report of early use of thrombolysis has been published. At this time, the role of thrombolytic therapy in the early management of freezing injury is not clear and is a matter for clinical investigation.

Infection during healing of deep frostbite injuries is an extremely serious complication. If uncontrolled, the infection can disseminate systemically or precipitate gangrene. Even if the infection does not disseminate or produce gangrene, local treatment of infection often necessitates surgical debridement of the injury and loss of tissue that might otherwise have been preserved. At the earliest evidence of infection, after culturing the wound, aggressive antibiotic therapy should be initiated. The antibiotics should be directed at usual wound pathogens, including anaerobes, and the nosocomial organisms characteristic of the particular MIT. If prophylactic antibiotics have been used, antibiotics should be selected on the assumption that the infection is due to organisms resistant to the prophylactic antibiotics.

The principal late complications of frostbite include tissue loss, contractures, persistent pain, cold sensitivity, susceptibility to reinjury and hyperhidrosis. The evaluation and management of post-injury symptoms may be complicated by secondary gain. Contractures and tissue loss should be managed by mechanical means such as orthotics and prostheses. There are no uniformly beneficial approaches to the management of persistent pain. Referral to a specialty pain management center as early as possible is appropriate. Cold sensitivity can be managed by extra protection of the injured area. However, occasionally cold exposure of any portion of the skin can 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. Post-frostbite hyperhidrosis of the feet can increase the incidence of dermatophyte infect ion and maceration.

3.2.5 Return to Duty and Physical Profiling

Recovery from frostbite is slow; Soldiers with frostbite are, as a rule, unable to participate in outdoor duties for at least the remainder of the cold season. Even though most frostbite injuries are only first or second degree, persistent post-healing functional symptoms are frequent and may be a cause for separation from military service.

Physical profiles should be based on each individual circumstance and any established local, Department of the Army (DA) or U.S. Army Medical Department (AMEDD) policies. If no specific policies have been established, the following profiling guideline is suggested:

1. If the frostbite injury has caused significant loss of tissue or persistent disabling symptoms, the soldier should be evaluated by a Medical Board (MEBD) and referred, as appropriate, for further consideration.

2. For healed first degree frostbite: Following complete healing, issue a permanent P-2 profile permitting the use of additional cold weather protective clothing as needed. Non regulation clothing items are permitted under the regulation outer uniform.

3. For healed second, third and fourth degree frostbite: Initially, issue a temporary P-3 profile prohibiting both exposure to temperatures below 32^0F and any physical activities limited by the injury itself. This temporary profile is renewed at three-month intervals for the remainder of the cold season. At the end of the cold season, issue a permanent P-2 profile similar to that for first-degree injury providing there is no apparent residual difficulty. Reappearance of symptoms upon the return of cold weather may require evaluation by a MEBD and appropriate further evaluation.

3.3 Non-Freezing Cold Injury (NFCI).

Non-freezing cold injury is the result of prolonged (many hours) exposure of the extremities to wet-cold; but above freezing, conditions. The feet are the most common area of injury which is reflected in the common names of the two principal types of non-freezing injury: trench foot and immersion foot. Trench

foot occurs during ground operations and is due to 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 venture on cold ocean.

3.3.1 Pathogenesis

NFCI is the result of prolonged (many hours) cooling of the lower extremities to temperatures above freezing but below 65^0F. The prolonged cooling produces some damage to all the soft tissues; but peripheral nerves and blood vessels suffer the greatest injury. The vascular injury causes secondary ischemic injury which aggravates the direct effect of cold on other tissues. NFCI is initially reversible, but if cooling is sustained, it becomes irreversible. Wet conditions increase the risk and accelerate the injury both because wet clothing insulates poorly and 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 to early infection.

3.3.2 Clinical Manifestations

When first seen, the injured tissue is pale, anesthetic, pulseless and immobile, but not frozen. Trench foot or immersion foot (depending on the environmental medium causing the injury) can be diagnosed when these signs DO NOT CHANGE AFTER WARMING. In addition, the skin is frequently macerated and slightly edematous. The degree of the injury is usually not completely apparent early.

Initially, despite rest and warmth, the injured part remains pale, anesthetic and pulseless. After several hours (occasionally as long as 24-36 hours), a marked hyperemia develops associated with severe burning pain and reappearance of sensation proximally, but not distally. The hyperemia represents a passive venous vasodilatation 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.

Recovery from NFCI is slow due to its neuropathic component. Except in minor injuries, sharp, intermittent “lightning” pains develop in the second week after injury, which are in addition to the burning and are described as reminiscent of tabes dorsalis. Improved sensitivity to light touch and pin in the area of anesthesia within four to five weeks suggests reversible nerve injury and less likelihood of persistent symptoms. Persistence of anesthesia to touch beyond six weeks suggests neuronal degeneration. Injury of that degree takes much longer to resolve and has a greater likelihood of persistent disabling symptoms.

Hyperhidrosis is a carrion and prominent late feature of NFCI and seems to precede the recovery of sensation. A distinct advancing hyperhidrotic zone can develop which is presumed to mark the point to which sudomotor nerves have regenerated (Guttmann. L., 1940). The excessive sweating may be permanent. It predisposes to blistering, maceration and dermatophyte infection.

3.3.3 Classification

NFCI has been classified into four degrees of severity. Two schemes of classification have been used based on clinical series from World War II. The Webster classification (Webster, D.R., Woolhouse, F.M. and Johnston, J.L., 1942) is based on the clinical appearance of the foot two to three days after injury. The Ungley classification (Ungley, C.C., 1949) is based on the distribution of anesthesia seven days after injury. These two systems correlate well and provide useful prognostic information.

Minimal NFCI (Webster) or Grade A (Ungley). Hyperemia and slight sensory change remain two to three days after injury. At seven days after injury, no objective findings or anesthesia remain. Rapid re-ambulation is the rule with only occasional residual cold sensitivity. Return to duty is possible within one to two weeks.

Mild NFCI (Webster) or Grade B (Ungley). Edema, hyperemia and definite sensory change are still present two to three days after injury. At seven days after injury, anesthesia is found only on the plantar surface of the foot and tips of the toes, and lasts four to nine weeks. This degree of injury is associated with one to three weeks of edema, two to four weeks of neuropathic pain and three to seven weeks of hyperemia. Bullae and skin loss are not seen. Hyperhidrosis and residual cold sensitivity occur in about half the injuries, although not necessarily in the same individuals. Re-ambulation is possible when walking does not increase pain. Return to duty usually requires three to four months.

Moderate NFCI (Webster) or Grade C (Ungley). Edema, hyperemia, bullae and mottling are p resent two to three days after injury. At seven days, anesthesia to touch is present on the dorsum of the foot as well as the plantar surface and toes. In addition to anesthesia to touch, vibration and position sense are diminished and the intrinsic muscles of the foot become paretic and atrophic. Edema persists two to three weeks, pain and hyperemia up to 14 weeks. Some of the skin affected by blistering sloughs, but loss of deep tissue does not occur. Hyperhidrosis and cold sensitivity occur in the majority of cases. A significant number of cases will become permanently disabled and these soldiers will not be able to return to duty.

Severe NFCI (Webster) or Grade D (Ungley). Severe edema, extravasation of blood and incipient gangrene are present two to three days after injury. At seven days, complete anesthesia is found in the entire foot with paralysis and wasting of the intrinsic muscles. The injury often extends proximal to the foot. The injury produces significant loss of tissue through autoamputation. Deep tissue, as well as superficial tissue is lost. Gangrene is a constant risk until tissue loss is complete. Edema in tissue that is not lost lasts three to seven weeks; hyperemia and pain last up to four months. Prolonged convalescence and permanent disability are the rule. Soldiers with these injuries rarely are able to return to duty.

3.3.4 First Aid and Field Management

NFCI, like frostbite, is an insidious injury because the affected tissue is cooled to the point of anaesthesia while the injury is occurring. So, like frostbite, the first essential of management is detection. Foot inspection and care every eight hours under cold-wet condition 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. Soldiers who suspect a NFCI should inspect and warm their feet immediately.

If NFCI is suspected to have occurred, 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. Dry covering of the injured part and protection of the casualty from cold may permit spontaneous warming of the injured tissue during evacuation. If warming does occur during evacuation, severe pain may develop before arrival at a MTF. Consequently, if a prolonged evacuation is anticipated in a particular case, the soldiers 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. Massaging the injury “to restore circulation” may worsen it.

NFCI, even in its mildest expression, evolves slowly, and so, requires time (> one week) for evaluation and recovery. Therefore, when a casualty is considered to have a NFCI, he should be evacuated to a rear echelon hospital. Nothing is to be gained from observation in forward echelons.

3.3.5 Hospital Management

The principal requirements of initial hospital management are tetanus prophylaxis, management of concomitant hypothermia and dehydration, and pain relief. The casualty should be warmed and rehydrated, but the injured extremity should not be actively warmed. Bed rest in a warm room is sufficient.

The injured extremity should be kept at the temperature a fan some burning pain. External warming (as, e.g., a vascular light box) 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 with acetaminophen, NSAIDs and opiate analgesics 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 a 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. Open bullae, ulcers and areas of necrosis should be periodically monitored with surveillance aerobic cultures. If infection appears, it should be treated immediately with antibiotics selected to be effective

against organisms detected by surveillance cultures. In severe injuries, infection of deep tissues may develop. In this circumstance, the antibiotics selected must also be effective against anaerobes and pseudomonas (e. g., ampicillin sulbactam or imipenen cilistatin).

3.3.6 Return to Duty and Physical Profiling

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

Casualties with minimal or mild degrees of injury are usually able to return to duty. They should receive a medical fitness profile appropriate for their symptoms and functional limitation. Casualties who have suffered moderate or severe injury should be evaluated by a MEBD at the completion of clinical healing. Soldiers with persistent symptoms or functional impairment should be referred to a Physical Evaluation Board (PEB).

3.4 Accidental Hypothermia.

3.4.1 Pathophysiology of Hypothermia

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. With continued fall in core temperature, shivering, tachypnea, tachycardia and hypertension develop. These became maximal when the core temperature is about95^0F (35^0C).

Below a core temperature of 95^0F (35^0C)**,** the depressant effect of hypothermia begins to offset the metabolic activation. As core temperature falls from 95^0F to 86^0F (3000), metabolic rate, shivering, respiratory rate, heart rate and cognitive function all decline. The individual may become quiet and withdrawn or confused and combative initially, but eventually becomes obtunded. Furthermore, since the metabolic depression of hypothermia stops the hypermetabolic response to cold, the individual loses a substantial defense against additional fall in core temperature. As core temperature declines, the rate of its fall can accelerate.

Below 95^{0}F (35^{0}C) core temperature, heart rate, blood pressure and respiratory rate decline roughly in parallel. Metabolic rate, oxygen consumption and cardiac output are about half normal at 85^{0}F (29^{0}C) and about 20% normal at 68^{0}F (20^{0}C). At these lower temperatures, ventilation and perfusion do not quite keep up with metabolic requirements and a mixed respiratory and metabolic acidosis develops.

When the core temperature falls below 86^{0}F (30^{0}C), atrial tachyarrhythmias and repolarization abnormalities ("Osborne waves") appear. Below 82^{0}F (28^{0}C), the ventricular fibrillation threshold declines, presumably due to inhibition of Purkinje fiber conduction velocity.

Peripheral voluntary muscle activity and reflexes disappear about 80^{0}F (27^{0}C). Brainstem reflexes disappear about 73^{0}F (23^{0}C). Below 68^{0}F (20C), 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.

3.4.2 Accidental Hypothermia

Hypothermia is the clinical syndrome that results from reduced core temperature. By definition, hypothermia is considered to be present when the "core" temperature (clinically usually taken to bet he same as rectal temperature) is below 95F (35^{0}C). Hypothermia is always the product of loss of heat to the environment in excess of the rate of heat production by the body.

Hypothermia that occurs outside the therapeutic setting is called accidental hypothermia to distinguish it from hypothermia deliberately induced for therapeutic purposes. Individuals develop accidental hypothermia when the cooling effect of their environment is greater than their thermoregultory capacity. Individuals who have poor thermoregulatory responses to cold may become hypothermic even in relatively mild cold stress. Poor thermoregulatory responses to cold are found in individuals with metabolic illnesses (e.g., hypothyroidism or hypoadrenalism), malnutrition, severe trauma, extensive burns (which accelerate heat loss despite hypermetabolism), CNS lesions of thermoregulatory centers, depressant intoxications (barbiturates, opiates, ethanol) and others.

Soldiers, unless injured, generally are healthy and have normal thermoregulatory responses. In soldiers, accidental hypothermia is usually the result of severe cold exposure. In soldiers, accidental hypothermia is usually the result of either immersion in cold water (immersion hypothermia) or prolonged

exposure in cold-wet ground environments (exhaustion hypothermia).

Immersion hypothermia is usually the result of boating, pond skating arid automobile accidents. Airplane accidents and shipwreck 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:

1) water temperature.

2) protective clothing.

3) body posture and movement. Increased exposed surface area and movement increase the rate of heat loss.

4) body size and adiposity. Smaller, leaner individuals cool heat more rapidly.

5) thermoregulatory aggressiveness. There is individual variation in the 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 are unable because of fatigue or injury to sustain a metabolic rate sufficient to balance the loss of heat to the environment. Factors that influence the rate of temperature during exposure to cold land environments are:

1) ambient temperature and wind.

2) clothing.

3) precipitation. Precipitation reduces the insulating value of clothing and adds an additional source of cooling.

4) rate of physical activity. In contrast to immersion, physical activity is an important mechanism of maintaining core temperature in cold conditions on land. The benefit of physical activity lasts only as long as activity is maintained.

5) shelter. Dry shelter moderates the cooling effect of wind and precipitation, and may allow an opportunity to rest.

The lethality and difficulty of management of hypothermia depends on the degree of temperature depression. Consequently, hypothermia is classified into mild, moderate and severe, based on core temperature. Mild hypothermia is defined as core

temperature between 90 and 95^{0}F (32 to 35^{0}C). Casualties with mild hypothermia usually retain the ability to rewarm spontaneously and do not develop cardiac arrhythmias. Between 90^{0}F and 82^{0}F (320 to 28^{0}C), the range of moderate hypothermia, atrial arrhythmias become common and metabolic rate is sufficiently depressed to significantly slow the rate of spontaneous rewarming. Below 82^{0}F (28^{0}C), the range of severe hypothermia, spontaneous rewarming is markedly depressed and the risk of ventricular fibrillation becomes substantial.

3.4.3 Clinical Manifestations of Hypothermia

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 95^{0}F (35^{0}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. Persistent shivering is evidence of incipient hypothermia and should always be taken seriously. Shivering will diminish as hypothermia worsens. The other significant early manifestation of hypothermia is a change in mental status. 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 below 80^{0}F(27^{0}C).

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 in the 80-90^{0}F (27-32^{0}C) range.

At core temperatures between 70 and 80^{0}F (21-27^{0}C), reflexes and vital signs become imperceptible, the skin is cold and waxy and muscular rigor may be present.

The brain and the heart become electrically silent at core temperatures between 60 and 70^{0}F and the hypothermic casualty appears clinically dead.

3.4.4 First Aid and Field Management of Accidental Hypothermia

The diagnosis of hypothermia is confirmed by core temperature measurement. However, clinical thermometers are

often unavailable in the field and first responders have to rely on their judgement of the clinical state of the casualty.

Anyone suspected of hypothermia should be considered to be at risk of sudden death from ventricular fibrillation or hypotension and steps should be taken to prevent those complications. 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. Protection from wind and wet is important. The insulation under the casualty should be incompressible, otherwise it will rapidly become ineffective under the casualty's weight and allow significant heat loss. Airway heat loss should be prevented by any means available even if only a scarf or non-occlusive bandage is available.

If necessary, ventilation can be assisted by mask and bag. When indicated, endotracheal intubation is safe as it does not seem to increase the risk of ventricular fibrillation. Oxygen supplementation is usually not needed because of the low oxygen requirements in hypothermia.

Since dehydration and hypovolemia are common in hypothermic casualties, an IV should be started with warmed fluid (LR, NS, D5/0.5NS). If hypoglycemia, alcoholism or opiate intoxication are possible causes of hypothermia, naloxone (1-2 mg), thiamine (100 mg) and glucose (10-25 grams) should be administered IV.

An injury survey should be done and appropriate dressing, splinting and stabilization provided before transport/evacuation.

3.4.5 Cardiopulmonary Resuscitation

Victims of severe hypothermia often appear to be in cardiac arrest; they are unconscious and without perceptible signs of life. In the hypothermic individual with these signs, CPR would be initiated without hesitation because there is no significant risk of doing harm in excess of benefit. In the hypothermic individual, however, there is a real risk of converting a perfusing quiet bradycardia into ventricular fibrillation by external massage. The lowered ventricular fibrillation threshold is likely due to reduced conduction velocity in both the Purkinje system and myocardium allowing circus movement propagation of action potentials. Ventricular fibrillation greatly complicates and reduces the likelihood of successful resuscitation from hypothermia.

If possible, cardiac monitoring and Doppler flow probes should be used in the field to determine the need for CPR. (Be sure to warm metal electrodes before applying to avoid freezing injury to the skin.) If monitoring shows a regular ventricular

rhythm and arterial flow can be appreciated by Doppler, CPR should not be performed, even if pulse and/or blood pressure are otherwise imperceptible. Ventricular fibrillation requires external massage. Wide complex ventricular rhythms without detectable flow should probably also be given external massage.

If cardiac monitoring or flow probes are not available, the following suggestion can be considered for implementation.

Remember to take more time for assessment in hypothermic casualties. The slow pulse may be difficult to appreciate. If the casualty meets the criteria for CPR, initiate CPR unless:

1) Any signs of life (respiratory effort, spontaneous muscular movement) are present. These signs must be continuously reassessed to assure they persist. Their disappearance may represent a failure of perfusion due to ventricular fibrillation.

2) There are pre-existing orders not to resuscitate or a valid living will.

3) There are other obvious lethal injuries.

4) The chest is incompressible.

5) Performing CPR would be dangerous to the rescuers.

6) Core temperature is less than 42^0F (6^0C).

7) Serum potassium is greater than 10 meq/l. (This may not be the easiest measurement to obtain in the field, but is a reliable criteria for resuscitative futility).

CPR should be continued until core temperature exceeds 90^0F and no evidence of effective cardiac function is present (i.e., pulseless electrical activity, asystole or agonal rhythm).

Guidelines for discontinuing resuscitation after prolonged arrest in severe hypothermia have been developed at Emanual Hospital in Portland, Oregon. These are:

Resuscitation can be discontinued if:

1) All of the following are true: Core temperature (measured at the right or left atrium) is between 42 and 79^0F (6-26^0C), the victim has no vital signs, electrical asystole is present and the documented period of arrest has been greater than five hours.

OR

2) All of the following are true: Core temperature (measured at the right or left atrium) is between 79 and 90^0F(26-32^0C), the victim has no vital signs, electrical asystole is present and the documented period of arrest has been greater than12 hours.

3.4.6 Sudden Death in Accidental Hypothermia

Sudden death is a common complications of rescues of hypothermic individuals. Examples abound of hypothermic victims alive and alert at the start of crevasse extraction who died while being moved. Even more striking are the reports of mass deaths among shipwreck survivors, who had been able to climb aboard rescuing ships, only to be found dead shortly thereafter.

Three factors are believed to contribute to sudden death in hypothermic individuals: ventricular irritability, hypovolemia and orthostasis, and sudden intraventricular cooling.

The increased risk of ventricular fibrillation seems to be due to changes in ventricular impulse conduction. The hypothermic ventricle responds to mechanical stimuli with local action potentials. Because the Purkinje system is relatively more depressed than myocardium, action potentials are propagated directly by myocardium allowing establishment of circus movement arrhythmias.

Victims of accidental hypothermia are usually hypovolemic due to minimal food or water intake and cold diuresis. Immersion victims have had an immersion diuresis (due to the displacement of blood volume into the central circulation by the outside pressure on the legs and abdomen) in addition to cold diuresis. Hypothermia also depresses orthostatic reflexes causing venous pooling to occur in any upright posture. Muscular exercise increases blood flow to the exercising muscle, further reducing effective blood volume. The ultimate consequence of the hypovolernia, orthostasis and redistribution of blood flow is hypotension.

Finally, exercise and warming of the skin in hypothermic individuals increase blood flow to areas that have been underperfused during cold exposure. The stagnant blood in these areas is acidotic, hypekalemic and cold. If large amounts of this cold peripheral blood enter the central circulation, the ventricle is exposed to metabolic and temperature conditions (called post-exposure cooling or “after-drop”) that further aggravate the risk of fibrillation.

The risk of sudden death can be moderated during rescue by assuring that each of the risk factors above are controlled. Casualties should be kept quiet and supine. Sudden movements

during rescue should be avoided. The casualty should not be allowed to exert during rescue. Hypovolemia should be treated early.

3.4.7 Hospital Management

The key to 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 consist of providing sufficient insulation to both the body and the airway to prevent further heat loss. Passive rewarming is effective even from core temperatures as low as 80^0F (27^0C). Depending on the effectiveness of the insulation, core temperature increases from 0.25 to 1^0F per hour. Passive rewarming is appropriate only as long as temperature is rising even though it may take 24 - 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 alliance 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 (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. However, there are three 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.

One technique of active surface rewarming is used to warm patients after induced surgical hypothermia. In this technique the scalp is warmed by application of warm (104^0F) water soaked towels. The scalp has a low blood volume but high blood flow and rapid entry into the central circulation. Consequently, heat can be delivered to the core without increasing the delivery of large

amounts of cold peripheral blood. There are no reports yet of its efficacy in accidental hypothermia.

Active “core” rewarming is required for resuscitation of hypothermic cardiac arrest and for most severely hypothermic patients. There are four categories of core rewarming techniques: 1) intraluminal lavage, 2) heated air, 3) direct vascular warming, and 4) radiant energy.

Although virtually every cavity in the body has been lavaged with warm fluid for the treatment of hypothermia (stomach, urinary bladder, colon, abdomen and chest), the two in most common use are gastric lavage and intraperitoneal lavage using peritoneal dialysis equipment. These techniques are effective and ample. They raise core temperature 0.5 to 1. 5^{9}F per hour. The principal risks of lavage are the procedures required to gain access (gastric intubation, insertion of intraperitoneal catheters, etc.) and aggravation of edema due to fluid overload. Direct myocardial warming by mediastinal lavage using either tube thoracostomy or open techniques has been reported to have resolved ventricular fibrillation and permitted resuscitation when less invasive techniques of core rewarming had failed.

Ventilation with heated air has been developed as an effective technique for central rewarming. A variety of apparatus have been developed for use in the field and in the hospital. An excellent recent review of the technique and available apparatus is found in the Journal Of Wilderness Medicine (Lloyd, E.L., 1990).

Hemodialysis and cardiopulmonary bypass are very effective rewarming techniques. Relatively rapid rewarming is possible with both. Cardiopulmonary bypass offers the additional advantage of assured perfusion during resuscitation. Because these techniques are only available in specialized centers, they are usually only applied in extraordinary circumstances of profound hypothermia when other methods have failed.

Experimental devices for direct core rewarming using electromagnetic radiation in the microwave range have been reported. Such devices have not yet become available for clinical practice.

The choice of technique for rewarming depends on: (1) the state of the circulation and (2) the degree of hypothermia

Casualties who have stable circulation and only mild to moderate degrees of hypothermia can be given the chance to rewarm spontaneously. If they fail to rewarm spontaneously, then active rewarming should be started. They should be admitted to an intensive care unit, be given warmed, humidified oxygen and gradual re-hydration. They should have continuous cardiac and

temperature monitoring (rectal or esophageal). A thorough search for contributing causes of hypothermia should be done including evaluations for intoxication (alcohol and depressants particularly), endocrine dysfunction and malnutrition. Sepsis and stroke must be ruled out, particularly in the elderly. Associated injuries (particularly frostbite) and complications (pneumonia, decubitus ulcer, thrombophlebitis, pancreatitis, renal failure and hyperosmolarity) should be identified and treated. Oral intake should be prohibited until rewarming is complete and cold-induced ileus has resolved

Casualties in cardiac arrest or with severe hypothermia will need more aggressive management. Ideally, medical treatment facilities will have a protocol, which addresses both the need to provide an adequate circulation and rewarming for resuscitating hypothermic patients. Ad hoc management of this profound emergency cannot be as adequate as management guided by carefully conceived and frequently reviewed treatment protocols. (Additional advice for hypothermia treatment protocols can be found in the clinical texts listed in the bibliography of this handbook.)

The following steps are suggested for the management of hypothermic cardiac arrest. Endotracheal intubation, if not already done in the field, should be done and assisted ventilation begun with heated humidified oxygen. Since hypothermic resuscitation tends to be prolonged, early institution of mechanical compression and ventilation is appropriate. In the hypothermic casualty, oxygen requirements and carbon dioxide production will be low. Ventilation needs to be guided by blood gas measurements to avoid excessive respiratory alkalosis. Blood gas measurements can be interpreted as reported by the laboratory; temperature “correction” is no longer considered appropriate.

There is usually no requirement for large amounts of parenteral fluid. One or two liters during rescue is usually sufficient to manage hypovolemia unless there is evidence of hemorrhage or a severe diuresis (e.g. diabetic hyperosmolar coma). Fluid is not retained in the vascular space in hypothermia and dramatic edema appears early in resuscitation. The edema is not of great significance in the peripheral tissues except as a possible indicator that edema is also developing in the lungs. Pulmonary compliance monitoring during ventilation can be used to provide early evidence of pulmonary edema and guide appropriate adjustment of ventilation and fluids to minimize the problem.

Hypoglycemia may be a cause of hypothermia or may be a consequence. If hypoglycemia is found, dextrose (25 cc of D50/W) should be administered immediately. 100 mg of Thiamine IV should always precede the dextrose.

As a general rule, antiarrhythmic and vasoactive drugs are not useful during resuscitation from hypothermia until core temperatures exceed 90^0F. Below that temperature, drug effects are absent or unpredictable. Also, since drug metabolism is markedly slowed be low 90^0F, applying American Heart Association Advanced Cardiac Life Support (ACLS) drug protocols in hypothermia causes the accumulation of drugs which have no manifest effect when administered, but which suddenly and dramatically express themselves as they regain activity at higher core temperatures. A possible exception is bretylium tosylate, which has been shown (Orts, A., Alcaraz, C., Delaney, K.A., Goldfrank, L.R., Turndorf, H. and Puig, M.M., 1992) to have some efficacy against ventricular fibrillation at depressed core temperatures. If drug therapy of ventricular fibrillation is required, bretylium at its usual doses would be the drug of choice.

During resuscitation, temperature should be continuously monitored either in the rectum, esophagus or central vessels. Urine output should be monitored both as an index of renal perfusion, and as a guide for replacement of renal fluid losses which can be substantial.

Coagulopathies of uncertain etiology occur in some severely hypothermic patients. Profuse bleeding may develop during rewarming which may require massive replacement with red cells, fresh frozen plasma an platelets. Coagulopathies gradually resolve after successful rewarming.

Until it is clear that the patient can maintain core temperature, core temperature monitoring is required.

After rewarming, continuing management should be directed at any associated and underlying conditions. The principal post -warming complications of accidental hypothermia are: pneumonia (including aspiration after immersion), pancreatitis, rhabdomyolysis, myoglobinuria and renal failure. Temporary left ventricular dysfunction has been seen after profound hypothermia. Late complications of hypothermia include prolonged, sometimes permanent, cold sensitivity and occasionally contractures in the hands and/or feet.

If a thorough evaluation does not reveal any underlying conditions and the circumstances of the accident account for the hypothermia, permanent disability due to the hypothermic episode is unusual.

3.4.8 Return to Duty and Physical Profiling

Upon complete recovery from hypothermia and any complications, the soldier can return to his unit with a temporary P-2 profile, permitting the use of additional cold

weather protective clothing as needed. Non-regulation clothing items are permitted under the regulation outer uniform. Unit leaders should be instructed to provide additional supervision to the soldier during cold exposure. If a recovered soldier exhibits any tendency toward cold intolerance or inability to perform in the cold, the soldier should be referred to a PEB.

Since accidental hypothermia is frequently associated with other injuries (e.g., frostbite), those other injuries may well determine the convalescent course and disposition of the soldier.

3.5 Other Medical Problems in Cold Environments.

The body surfaces directly exposed to cold (i.e., skin and respiratory mucosa), suffer a variety of conditions that, if not prevented or appropriately managed, will lead to discomfort and even acute disability. All carry a risk of progression to more serious conditions.

3.5.1 Carbon Monoxide (CO) 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 poisoning.

Carbon monoxide is extraordinarily toxic. Concentrations of 100 parts per million in sea level air will produce carboxyhemoglobin (COHb) concentrations 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. (Note also, extremities below 50^0F 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 (e g., oral mucosa).

Oxygen saturation measured by co-oximetry is a useful screening test for CO poisoning because CO reduces oxygen saturation. Oxygen saturation as reported by arterial blood gas analysis is often calculated, not measured, and should not be relied on to rule cut CO poisoning. Indeed, a difference between co-oximetric and calculated oxygen saturation is excellent evidence of CO poisoning. The definitive test is direct measurement of carboxyhemoglobin in blood.

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. General indications for hyperbaric oxygen are: COHb is greater than 25%, metabolic acidosis, angina or ECG change, or neurologic symptoms other than headache.

If air evacuation is necessary, the lowest possible altitude should be used to maintain the highest possible $P0_2$.

3.5.2 Respiratory Tract Conditions

The nasal and bronchial mucosa responds to cold air by increasing mucus production. Consequently, chronic nasal discharge and cough are a frequent accompaniment of cold weather operations. Rhinorrhea and bronchorrhea are probably protective but can cause two principal complications. First, nasal discharge causes wetting and chapping of the philtrum and nasalabial sulcus which increases the risk of cold injury and local infection. Second, 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 local hygiene is a better measure than decongestant s 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. Influenza vaccination is an essential preventive measure. Medical officers 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.

3.5.3 Cold Urticaria

Cold can precipitate a variety of local immunologic skin reactions in susceptible individuals involving a variety of immunooproteins.

Cold urticaria which is manifested as local or systemic urticaria on exposure to cold can be familial, congenital or acquired. Its onset is usually abrupt and distinctive. 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. Although not common, cold urticaria is occasionally

associated with detectable cryoproteins (cryoglobulin, cold hemolysin and cryofibrinogen). A rare “delayed” cold urticaria has been described that may depend on cellular immune mechanisms rather than circulating immune mediators. Cold urticaria is potentially lethal either from systemic anaphylaxis or laryngeal swelling upon exposure to cold liquids during drinking.

Immune mediators activated by cold can cause other reactions besides urticaria. Most cause symptoms and signs including cyanosis, livedo reticularis, pruritus, paresthesia and Raynaud’s phenomenon on in the acral regions of the body**.** Immune complexes deposited in the microcirculation can cause distal ulceration, even frank gangrene. Cold hemolysin and agglutinins can precipitate episodic hemolysis and hemoglobinuria. Individuals who develop symptoms or signs of cryoprotein-mediated disease should be referred for evaluation.

3.5.4 Chilblains (Pernio)

Chilblains 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. Ears, face and exposed shins are other commonly mentioned locations. Multiple lesions are the rule. The lesion is pruritic and painful, particularly after re-exposure 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.

The etiology of chilblains is unknown. Sane cases may be secondary to cryoprotein or localized vasculitic processes (“lupus pernio”).

Management of chilblains is by protection from cold with suitable clothing. Symptoms will remit when cold exposure is eliminated.

3.5.5 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 solar protection 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.

3.5.6 Eczema (Winter Itch, “Eczema Craquele)

This condition is extremely common on the hands in the cold and frequently will generalize to involve all the skin. It is manifested by persistent painful itch, 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 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), avoiding harsh soaps and applying emollient creams to replace neutral skin lipids.

3.5.7 Lacerations

Untreated lacerations are prone to freezing injury. The open skin allows injury to both the wound margins (perhaps due to hemostatic vasoconstriction) and to deep tissue exposed to the cold environment. If a laceration becomes frostbitten, an initially small injury can be transformed into a serious deep injury prone to infection and slow to heal. Primary closure and adequate dressing prevents freezing injury in lacerations.

3.5.8 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. Soldiers who develop this complaint may require evaluation by a MEBD and referral to a PEB if symptoms are disabling.

Section 4.
Key Points and Reminders

******* The following section a list of discrete points td be used as reminders and briefing points for training and planning.

4.1 Useful Points to Remember.

1. Humans cannot sense core temperature. An individual becoming hypothermic will usually shiver but will not be otherwise aware of lowered core temperature. Individuals who do not shiver may have no insight into how cold they are.

2. Skin is sensitive to cold and will be painful until it cools to 50^0F (10^0C).

3. Skin accommodates to cold; exposure reduces both the sensation of cold and pain.

4. Skin is numb below 50^0F. The disappearance of pain in an extremity during cold exposure may indicate serious cold injury. Immediate visual inspection for frostbite is mandatory.

5. Cold exposure causes dieresis which aggravates the voluntary dehydration encountered routinely in the field.

6. Extremities below 50^0F (10^0C) are paralyzed. Individuals with significant hypothermia cannot help themselves.

7. The most important adaptation to cold is learning and applying skills and equipment that permit living and working in the cold in safety.

4.2 Prevention.

1. The best prevention against cold injury is a healthy, trained, equipped, well-fed and hydrated soldier.

2. In military operations and training, risk factors for freezing injuries include: 1) dehydration, 2) weight loss, 3) unplanned or unduly prolonged exposures to cold, 4) undertrained or overtired soldiers and 5) poor or insufficient equipment.

3. If one freezing injury has occurred in an operation, remember that everyone in the unit was exposed to the same conditions. Inspect everyone immediately.

4. 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.

5. Cold injuries in operations usually occur in clusters.

6. At the earliest suspicion of hypothermia, the casualty should be provided warm shelter immediately. The presence of hypothermia means that the individual is already unable to maintain body temperature. The hypothermia will only worsen if not managed. An individual with mild hypothermia will warm spontaneously and fully recover if provided warm shelter, warm liquids and supervision.

7. No one in whom hypothermia is suspected should be left alone.

8. Exercise is dangerous if significant hypothermia is present.

4.3 Rescue and Evacuation.

1. In the cold, the advantage of vehicular evacuation (ground ambulance, tracked vehicle or 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 10-12 soldiers is needed to move a casualty in the mountains or in snow. 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.

2. Fluids and medications may freeze and become useless if carried in packs or bags. For all practical purposes, 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 freezing.

3. Medical units must develop medical support plans that allow the earliest possible care and stabilization 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.

4. Whatever technique of evacuation is used, sufficient protection from cold must be available for the casualty during transportation. Since the casualty will be at rest and often depressed by i two arctic sleeping bags may be barely sufficient insulation. Airway heat loss should be prevented.

5. It is very important to remember to prevent heat loss from beneath the casualty. The down or synthetic material of sleeping bags is compressed and loses its effectiveness under the casualty' s weight. Additional incompressible, insulation (e.g., a foam mattress) is required regardless of the surface beneath.

6. Critical points in rescue and early management of accidental hypothermia include:

* To avoid sudden death, hypothermic casualties must be kept absolutely quiet. They must not participate in their own rescue. They must be kept supine or head down.

* Prevent further heat loss by removal of wet clothing, copious insulation and protection of the airway.

* Ventilate with warm, humidified air. Endotracheal intubation is safe if needed. Oxygen supplementation is not required for hypothermia alone.

* Provide volume resuscitation (1-2 liters of LR, NS or D5/0.5 NS) with warm fluid (carry next to the body during approach to keep warm)

* If evacuation requires re-exposure or interruption of resuscitation, consider rewarming in the field.

4.4 Medical Operations in the Cold.

1. Helicopter landings may raise huge opaque clouds of snow, blinding both the 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 MTF. This fluid 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.

2. Even warm liquid 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. However, once begun, supervision of

the infusion is very difficult, requiring re-exposure of the casualty.

3. Common IV solutions (LR, NS, D5/W or saline) can be thawed and used after freezing if no damage to the bag has occurred and the solutions are clear.

4. Certain medications are known to be damaged by freezing and should not be used after thawing (Weinberg, A. D., Hamlet, M.P., Paturas, J.L., McAninch, G.W. and White, R.W., 1990). These include epinephrine, NPH insulin, sodium bicarbonate, magnesium sulfate, tetanus toxoid and mannitol. These must not be used after freezing.

5. Hypothermia is a significant risk during resuscitative care of burn and trauma casualties in forward areas, particularly if surgery is required. These casualties often arrive already hypothermic due to 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.

6. Medical staff 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.

APPENDICES

Appendix A: Wind Chill Chart
Appendix B: Cold Weather Training Guidelines
Appendix C: Clinical Manifestations of Hypothermia

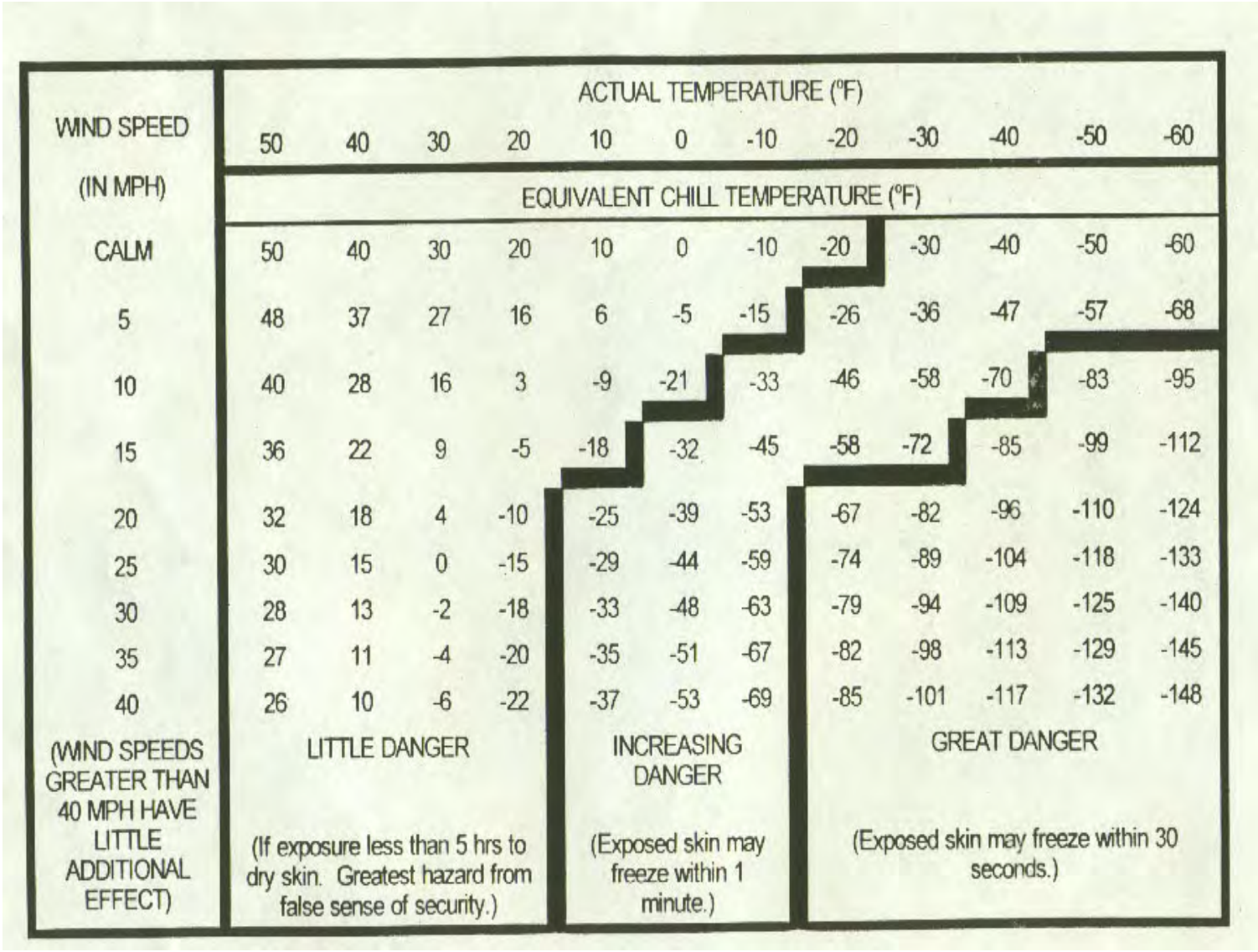
Appendix A: Wind Chill Chart

The “wind chill” temperature is an estimate of the still air temperature that causes the same skin cooling as each of the particular combinations of wind and temperature listed. For example, air at -10^0F blowing on the skin at 10 MPH has about the same cooling effect as -33^0F still air. The risk of freezing is based on the estimated cooling rate and stratified into three zones of ascending risk.

The wind-chill temperature provides a roughly quantitative index of the risk of freezing injury to exposed skin. The estimates in this table are based on t he effect of wind and cold on dry skin in healthy individuals with normal skin. Wet skin or restricted circulation will increase the susceptibility to freezing.

The table is adapted from the Wind Chill Chart at Appendix A of USARIEM Technical Note 92-2: Sustaining Health and Performance in the Cold.

Additional information about the Wind Chill Index can be found in Toner, M.M. and McArdle, W.D. “Physiological Adjustments of Man to the Cold”, In: Pandolf, K.B., Sawka, M.N. and Gonzalez, R**.**R. (eds.) Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Indianapolis, IN: Benchmark Press, 1988.



WIND SPEED (IN MPH)	ACTUAL TEMPERATURE (°F)											
	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
	EQUIVALENT CHILL TEMPERATURE (°F)											
CALM	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
5	48	37	27	16	6	-5	-15	-26	-36	-47	-57	-68
10	40	28	16	3	-9	-21	-33	-46	-58	-70	-83	-95
15	36	22	9	-5	-18	-32	-45	-58	-72	-85	-99	-112
20	32	18	4	-10	-25	-39	-53	-67	-82	-96	-110	-124
25	30	15	0	-15	-29	-44	-59	-74	-89	-104	-118	-133
30	28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125	-140
35	27	11	-4	-20	-35	-51	-67	-82	-98	-113	-129	-145
40	26	10	-6	-22	-37	-53	-69	-85	-101	-117	-132	-148
(WIND SPEEDS GREATER THAN 40 MPH HAVE LITTLE ADDITIONAL EFFECT)	LITTLE DANGER				INCREASING DANGER			GREAT DANGER				
	(If exposure less than 5 hrs to dry skin. Greatest hazard from false sense of security.)				(Exposed skin may freeze within 1 minute.)			(Exposed skin may freeze within 30 seconds.)				

Appendix B: Cold-Weather Training Guidelines

The table in Appendix B was developed by CPT James Cool, MC, USA at USARIEM as a straightforward set of guidelines for use by units training in cold environments. The guidance is directed at units that do not have special cold weather experience and training.

The table is designed to be copied and distributed to unit leaders for their continued reference.

To determine the recommended cold weather protection for training, first determine the Wind Chill risk category per Appendix A and estimate the work of the soldiers. The table can then be consulted for appropriate protective measures. If the soldiers' work intensity or cold exposure is going to vary (e.g. periodically off-loading trucks), ensure appropriate protection for each level of work intensity.

Table B. Cold Weather Training Guidelines as a Function of Soldier Work Intensity and Wind Chill Risk

Wind Chill Risk Work Intensity	Little Danger	Increased Danger	Great Danger
High Digging Foxhole Running Marching with Load Making or Breaking Camp,	Surveillance by small unit leaders • Black gloves optional above 0^0F, mandatory below. • Increase hydration.	• ECWCS* equivalent gloves or mittens with liners. • No facial camouflage. • Skin covered and dry. • Warm shelter for rest periods. • Vapor barrier boots below 0^0F.	• postpone nonessential training. • Essential tasks with only <15 minute exposure • Enforce buddy system work groups no smaller than two. • No exposed skin.
LOW Walking Drill and Ceremony	Increased surveillance. • Skin covered and dry. • Mittens or gloves with liner, • No facial camouflage below 10° F. • Full head cover below 0^0F.	• Unit non-essential training. • 30.40 minute work cycles to accomplish essential tasks with careful surveillance for cold injury.	•Cancel outdoor training.
Sedentary Sentry Duty Clerical Work	• Full head cover. • No facial camouflage below 10°F. • Vapor barrier boots below 0°F. • Shorten duty cycles • Provide warning facilities.	• Postpone non-essential training. • 15-20 minute work cycles for essential tasks • Enforce buddy system work groups no smaller than two. • No exposed skin.	'Cancel outdoor training.

* **E**xtended **C**old **W**eather **C**lothing **S**ystem

Appendix C: Clinical Manifestations of Hypothermia

BODY CORE TEMPERATURE		MANIFESTATIONS
C°	F°	
	100	
37		
Normothermia		
		Increased Metabolic Rate
35	95	
Mild Hypothermia		Maximal Shivering Confused-Combative-Dysarthric
32	90	
		Obtunded
Moderate Hypothermia		Shivering Ceases. Increased Muscle Tone
30	85	Reduced Ventricular Fibrillation Threshold Atrial Fibrillation Vital Signs Often Clinically Absent
	80	
		Coma
		Survivor 16 y/o Male, 1.5 Hour Resuscitation
25		Spontaneous Ventricular Fibrillation
Severe Hypothermia	75	
		No Corneal Reflex
	70	Survivor 67 y/o Male, 3 Hour Resuscitation
20		Asystole
	65	No CNS Electrical Activity
	60	
		Survivor 23 d/o Male, 6 Hour Resuscitation
15		
	55	
10	50	
	45	Lowest Temperature of Survival from Induced Hypothermia
5		

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