

erythema, edema, increased warmth, and a sensation of itching. With repeated episodes superficial blisters or ulcers may appear.

Treatment

In the initial stages of chilblains, gradually rewarm the exposed area at room temperature. If the face is involved, you may simply hold a warm hand to the area. If the hands are affected, place them in the axilla or crotch. If blisters form, gently cleanse the area, and protect it to avoid infection. Usually the duty time lost from such an injury is insignificant. An episode of chilblains should encourage everyone to conscientiously practice prescribed protective measures.

Immersion Injuries

This is a tissue injury resulting from prolonged exposure to cold water temperatures above freezing for 12 hours or longer. Cases of immersion foot can occur, however, at temperatures as high as 70 degrees F (21 degrees C). The lower water temperature reduces the duration of exposure necessary to produce the injury. It was commonly seen in lifeboat survivors in World War II. The term "trench foot" is applied to a similar pathological condition seen in troops engaged in trench warfare where mobility is limited and dry boots and socks are unobtainable. In general, the symptoms and treatment of both conditions are similar.

Symptoms

Initially, there is a stage of severe peripheral vasoconstriction caused by local and general body cooling. The resultant ischemia is a major factor in nerve and muscle injury. The feet will be cold, swollen, waxy white, and mottled with cyanotic burgundy-to-blue splotches. The feet will be resilient upon palpation in contrast to the solid frozen tissue encountered in the frostbite injuries. Walking will be difficult because the skin is anesthetized and deep musculoskeletal sensation is usually lost.

Treatment

Treatment consists of gentle drying, elevation, and exposure of the extremity in an environmental temperature of 64 to 72 degrees F (18 to 22 degrees C). Keep the rest of the body warm. Initially, hyperemia and swelling with superficial blistering can be seen. This hyperemic stage may be quite painful for the victim. Bed rest, cleanliness, and pain relief are essential. The prognosis, even with excellent treatment, depends on the extent of the original damage as shown in Table 1.

Table 1. Prognosis of Immersion Foot Cases

SEVERITY	TISSUE DAMAGE	NERVE DAMAGE	ESTIMATED HOSPITALIZATION
Minimal	Reversible None to transient	Reversible None to transient	Hours
Mild	Reversible Edema, paresthesia	Reversible	Days to weeks
Moderately	Increased Edema, paresthesia	Irreversible	Weeks to months
Severe	Irreversible Gangrene	Irreversible	6 months or more

In mild cases the average hospital stay is 6 to 8 weeks. Patients are usually fit to return to full duty within 4 months after onset.

Moderately severe cases usually show other signs of faulty circulation. The average hospital stay is 9 weeks to 6 months. Only 25% of the cases recover sufficiently to return to full duty; most of the remainder can perform limited duty only.

In severe cases, gangrene invariably develops. Extensive anesthesia is present 6 months after rescue. The average hospital stay is at least 6 months. Few cases become fit even for light military duty.

Recovered cases often present late complications of edema, deep stabbing pain, cold sensitivity, chronic infection, causalgia, hyperhidrosis, and a Raynaud's-like phenomenon.

HYPOTHERMIA

This is an abnormal reduction of the body's core temperature below its maximum efficiency level (98.6 degrees F or 37 degrees C), which results in a progressive deterioration in cerebral, musculoskeletal, and cardiac functions. Accidental hypothermia, the insidious killer that is responsible for so many deaths in cold conditions, is caused by cold water immersion or exposure to cold. Hypothermia can develop in situations where the ambient temperature is well above freezing whenever calories lost to the environment exceed those produced by metabolism. The rate and severity of the development of hypothermia is determined by the degree to which heat loss becomes greater than heat production.

Controlled hypothermia induced in a patient to increase the margin of safety for various neurosurgical and cardiovascular surgical procedures is well-recognized. It is hypothermia occurring in uncontrolled situations that will threaten combatants in a military situation. The potential for this condition will be increased in the presence of fatigue, inadequate hydration, poor nutrition, inadequate protective clothing, and cold water immersion or cold exposure. The heat is lost through four mechanisms:

- Radiation: Emission of infrared energy from the body.
- Conduction: Direct contact between the body and a colder object.
- Convection: Heat transference from the body to a moving fluid, such as water or air.
- Evaporation: Loss of energy (in the form of body heat) in the conversion of internal liquids to vapor (such as sweat).

Hypothermia is best prevented by properly dressing for the climate to be encountered. Remember, it doesn't have to be near freezing to cause hypothermia. The biggest threat aboard ship is exposure to cold water, which can cause a rapid cooling of the body and lead to death in a matter of minutes.

HYPOTHERMIA PROGRESSION CHART

SEVERITY	BODY TEMPERATURE	SYMPTOMS
Mild	95 degrees F (35 degrees C)	Intense uncontrollable shivering; impaired ability to perform complex tasks.
(Severe)	89.6 degrees F (32 degrees C)	Violent shivering, difficulty speaking, sluggishness; amnesia begins.
Moderate	89.6 degrees F	Shivering decreases; muscles become rigid; muscle coordination impaired; erratic movements.
(Severe)	80.6 degrees F (27 degrees C)	Irrational; stupor; lost contact with environment; pulse and respiration slow.
Deep	80.6 degrees F	No response to words; reflexes stop working; heartbeat is erratic; unconsciousness begins.
(Severe)	Below 77 degrees F (25 degrees C)	Failure of heart and lungs; internal bleeding; death.

Symptoms

The body's initial reaction to a decrease in the core temperature is a multifaceted attempt to simultaneously stop the heat loss and at the same time to increase the temperature in the internal metabolic furnace. These complex reflexes are controlled at the hypothalamus. Vasomotor reflexes will shut down the skin and subcutaneous vessels, shunting the blood to deeper structures and cutting down the heat loss at the skin surface. Blood pressure may temporarily rise as the increased peripheral resistance increases cardiac work. Vigorous shivering will take place as the body attempts to warm itself. This shivering will raise the body's metabolic requirements, especially oxygen, significantly. These protective hypothalamic responses may be blocked by certain drugs such as phenothiazines and sedatives, alcohol intoxication, and head injuries. Intense shivering is a clear warning that the victim is on the verge of becoming hypothermic.

If the core temperature continues to drop, effective muscular activity deteriorates progressively until it has virtually ceased at about 90 degrees F (32 degrees C). Clinically, this will be recognized in the hypothermic victim who will initially complain of being cold. The victim may then pass through the stage of violent shivering and may appear simply too tired to care. Muscular weakness, stumbling gait, disorientation, and an almost total disinterest in the surroundings are all ominous signs of progressive hypothermia. As hypothermia progresses, respiratory effort is decreased along with the oxygen demand of the body. However, a point is eventually reached where the weakened respiratory effort results in serious hypoxia. The ability to deliver oxygen to the hypothermic victim will determine whether the heart and brain will survive this thermal insult.

When the victim's temperature drops to 86 degrees F (30 degrees C), cardiac irregularities occur. Initially, atrial fibrillation is seen, followed, as the temperature drops, by ventricular irritability and fibrillation. Below 80.6 degrees F (27 degrees C) the risk of ventricular fibrillation (VF) is paramount and is probably the most frequent cause of death in the hypothermic victim. The victim may show no clinical signs of life on casual observation. Pulses may be very faint and even unpalpable peripherally. Blood pressure may be non-obtainable, and the victim may be unresponsive even to painful stimuli. Pupillary and corneal reflexes may be sluggish or even absent when the core temperature goes below 82.4 degrees F (28 degrees C). Even with these clinical signs resuscitation and recovery are possible for the hypothermic victim; therefore, start treatment immediately.

Diagnosis and Treatment

Medical personnel must use a low reading temperature thermometer to diagnose hypothermia. Such a device is the subnormal thermometer (NSN 6515-00-139-4593) which reads temperatures as low as 70 degrees F. A regular mercury thermometer reads to 89 degrees F. An appropriate thermistor or thermocouple are alternatives to mercury ones.

Once hypothermia is documented or even strongly suspected on a clinical basis, start the rewarming process. The technique will depend on the facility and the equipment available. Generally, rewarming will consist of external (surface) or internal (core) methods. During external rewarming remove wet, cold clothing from the victim, and expose him or her to an external heat source. This may be done by wrapping the victim in warm blankets or a rewarming blanket, either with resistance coils or circulating fluid; applying hot water bottles; immersing the victim in water at 104 degrees F (40 degrees C); or simply exposing the victim to warm room air. Exposure in a warm room may be the best method if you know the duration of hypothermia was short and the level was not deep. Treatment of hypothermia in the field is limited to body-to-body heat.

The problems of rewarming shock may be less with slower acting external rewarming methods. Rewarming shock, a phenomenon observed following active rewarming, is characterized by cardiovascular collapse associated with vascular redistribution. This collapse is probably due to the closing of the peripheral arteriovenous shunts, peripheral vasodilation, and the subsequent return to the heart of cooled blood, which is full of the acid breakdown products of metabolism.

Internal methods of rewarming include the use of peritoneal dialysis with warmed 108 degrees F (42 degrees C) dialysate and the rapid changing of these fluids; the use of cardiopulmonary bypass with a heat exchanger in line to raise the temperature of the perfusate; and finally, the use of inhaled warm oxygen either by a mask or endotracheal tube (ET). Again, the facility, equipment, and expertise of the personnel available will determine how the hypothermic victim will be treated.

Upon reception at a definitive medical facility, the hypothermic patient will require intensive monitoring. An ECG and skull and chest X-rays should be obtained as well as CBC, BUN, blood sugar, electrolytes, calcium, amylase, arterial blood gases (ABGs), and coagulation profile tests. The patient must have continuous ECG monitoring, and there may be some bizarre patterns. Respiratory support may require intubation and even mechanical support to keep up with the increased oxygen demands as rewarming proceeds. Careful handling of the patient is essential. Any changes in body position or rough handling can precipitate VF. Premature ventricular contractions (PVCs) can be lessened by correcting hypoxia and acidosis. Lidocaine infusion is also useful for ventricular arrhythmia. Atrial arrhythmias will correct themselves as rewarming progresses. Atropine and electrical pacing will have little beneficial effect on conduction in the hypothermic heart. On the contrary, irritation of the myocardium by the pacemaker electrode itself or by its discharge can lead to VF. If the hypothermic heart is unresponsive to external countershock, extracorporeal blood rewarming must be started promptly before cardioversion can be successful.

As in any cardiac arrest, continuous closed cardiac massage and ventilation support must be provided until rewarming has restored the normal cardiac rhythm. The pH, PCO₂, and PO₂ will require frequent monitoring, and abnormalities must be corrected as the situation dictates. Central venous pressure (CVP) should be maintained between 5 to 10 cm with appropriate fluids. As peripheral vasodilation occurs in rewarming, additional warm IV fluids must be administered. If the coagulation profile test suggests a disseminated intravascular coagulation (DIC) phenomenon, heparin may be necessary. A DIC-like syndrome has been described in some cases of deep hypothermia.

DEFINITIVE CARE MANAGEMENT OUTLINE

- Determine if the patient is hypothermic using a low temperature thermometer.
- Admit patients with compromised mental status or cardiovascular irregularities to ICU.
- Evaluate patient with emphasis on identifying history of occurrence and predisposing conditions or diseases (e.g. shock, myxedema, hypoglycemia).
- Record continuous or frequent temperature readings with a low temperature recording thermistor or thermometer.
- Insert an IV line and infuse IV fluids warmed to 108 degrees F (42 degrees C) before using.
- Place arterial pressure monitoring line, if possible.
- Begin continuous cardiac monitoring.
- Insert a retention urinary catheter.
- Measure vital signs (VS), urinary output (U/O), and specific gravity (SG) at least hourly during rewarming.
- Wrap patient in rewarming blanket and set temperature at 104 to 110 degrees F (40 to 43 degrees C).
- Provide respiratory support using oxygen by mask or endotracheal tube (ET)(the tube may produce VF) with mechanical ventilation. Aim for high PO₂, normal pH, and PCO₂. Measure arterial gases and pH as frequently as necessary.
- Keep respiratory tract clear of secretions. Suction carefully; traumatic suctioning can precipitate VF.
- Perform the following blood tests: CBC, BUN, creatinine, electrolytes, glucose, amylase, calcium, fibrinogen, prothrombin time, platelet count, ABG, and pH (corrected to core temperature).
- Obtain chest and skull X-rays.
- Perform 12-lead ECG.
- Maintain CVP between 5 to 10 cm with appropriate fluids (H₂O) or expanders, calculated to gradually correct electrolyte imbalance. (Avoid those containing potassium (K) if possible, as it may cause acute renal failure or VF).
- Administer bicarbonate to correct acidosis.
- Treat PVCs with standard boluses of 1-5 mg/kg of lidocaine.
- If ventricular tachycardia, fibrillation, or standstill occur, begin closed-chest cardiac compression and assisted ventilation until extracorporeal blood rewarming, using a cardiopulmonary bypass with a heat exchanger, can be started.
- Cardioversion should occur when the heart is sufficiently warm.
- Premature atrial contractions (PACs), flutter, and fibrillation should revert to normal with rewarming.
- Give therapeutic doses of corticosteroid or thyroid hormones, if indicated.
- Give heparin for a DIC-like syndrome.
- Consider peritoneal dialysis for patients with core temperatures of 94 degrees F (35 degrees C) and below; it is definitely indicated for temperatures of 87 degrees F (31 degrees C) and less.
 - Heat all dialysis fluids to 108 degrees F (42 degrees C).
 - Infuse the first two liters as fast as the bottle elevation will allow, additional infusions should take about 20 minutes for each liter.
 - Add 1000 units of heparin to each liter to prevent clotting in the exchange system.
 - Do not add K to the dialysis fluids.
 - Culture fluids of first, fifth, and every tenth exchange.
 - Monitor U/O including amount, SG, and amount of sodium (NA) and potassium (K).

Recognizing hypothermia as a serious medical emergency is the first step to successful resuscitation. Victims often present cold, cyanotic, pale, and stiff as if in rigor with no palpable pulse, no audible heart sounds, no visible respiratory excursions, and fixed pupils. They may be in various states of duress, and if they are cooled in a crouched or huddled position, it may be impossible for them to straighten out on an examination table. Since extremely bizarre ECG readings are seen on some revivable hypothermic victims, any electrical activity on an ECG should be considered as a hopeful sign. This is true even though your initial impression of the ECG suggests artifact interference. Because patients have been successfully resuscitated at core temperature of 64 degrees F (18 degrees C) with flat ECGs, the axiom here is "no one is dead until he is warm and dead".

All patients must be kept absolutely NPO. Continuous monitoring of rectal temperature, ECG, and blood pressure is absolutely essential. Once recovered from hypothermia, the patient will require careful inpatient observation for 5 to 7 days or longer. The recovery period could be longer if complications occur.

FIELD MANAGEMENT GUIDELINES

If you treat a hypothermic victim in the field, you must be prepared to deal with the consequences. This means that in cases more severe than mild hypothermia, you must be aware of the likelihood of cardiac arrest or VF, and therefore treat this with sustained CPR. In general, moderate and severe hypothermic victims should be kept in the "metabolic icebox" and not rewarmed in the field. Although you may not wish to rewarm severe hypothermic victims in the field, you should prevent additional heat loss. If you allow his or her temperature to drop further, you increase the risk of VF when rewarming is initiated.

In summary, use the following guidelines for basic management of the three levels of hypothermia:

MILD HYPOTHERMIA: If victims can walk on their own or speak lucidly with no apparent mental changes, rewarm them by whatever means available at the battalion aid or clearing station such as hot, sweet drinks, body to body heat, sleeping bags, or other external heat sources.

MODERATE HYPOTHERMIA: If there is any question whether victims are more than mildly hypothermic, evacuate them to the clearing station. Transport them as carefully as possible to prevent VF. During evacuation do not rewarm victims, but attempt to prevent a further drop in body temperature. Rewarm them at the clearing station or evacuate them to a field hospital.

SEVERE HYPOTHERMIA: Evacuate severely hypothermic individuals from the battalion aid or clearing station to a field hospital. Prevent further heat loss and a subsequent drop in core temperature during evacuation.

COMPLICATIONS, CONSIDERATIONS, AND TREATMENT IN HYPOTHERMIA

COMPLICATION	CONSIDERATION	TREATMENT
1. Cardiac arrhythmias (greatest concern)	<ol style="list-style-type: none"> 1. Low temperature may cause intractable response in cardioversion resulting in <ol style="list-style-type: none"> a. Ineffective shocks that burn the chest b. Proper drug dosage at hypothermic levels becomes toxic with rewarming 2. PACs, flutter, and fibrillation should revert to normal with rewarming. 3. Slow, normal ECG, and cardiac response does not require CPR. 	<ol style="list-style-type: none"> 1. Bretylium - 5 to 10mg/kg over 8 to 10 minutes for ventricular ectopy with infusion rate of 2mg/min. 2. Defibrillate as necessary.
2. Psychiatric problems rewarming	<p>Patient may look exceptionally well upon rewarming but may develop problems.</p>	<p>Restrain patient on because of possible hyperactivity and disorientation.</p> <p>Observe patient over a minimum of 24 hours before releasing.</p>
3. Pneumonia	<p>Respiratory effort may be poor.</p> <p>Bronchorrhea may occur.</p>	<p>Consider prophylactic antibiotics.</p>
4. Renal failure	<p>Severe hypertension during post-rewarming period is precursor.</p>	
5. Pancreatitis Diabetic ketoacidosis	<p>Common post-rewarming sequelae.</p>	
6. DIC-like syndrome		<p>Heparin</p>
7. Myocardial infarction		
8. GI Bleeding		
9. Hypotension		
10. Peritoneal infarction Secondary to peritoneal dialysis	<p>Common post-rewarming sequelae</p>	

FREEZING INJURIES/FROSTBITE

This is a graded tissue injury, resulting from exposure to environmental temperatures below freezing. The speed of onset, depth, and severity of the injury depend on the temperature, windchill, and the duration of exposure. During the onset of frostbite, tissue freezing starts with the formation of ice crystals within the exposed tissue.

Cellular injury and death are the microscopic consequences of frostbite. Probably both direct cellular trauma from the ice crystal formation and cellular ischemic embarrassment from complex vascular reactions occurring in cold exposure contribute to the outcome. It has been postulated that this ice crystal growth mechanically compresses the surrounding cells and possibly ruptures the cell membrane, either during the freezing or later during thawing. Most of the data about tissue freezing injuries has come from animal experiments, since there is little human material available for study during the acute phase of the injury. Evidence from mammalian experiments shows that tissue freezing begins when temperatures in the deeper limb reach 50 degrees F (10 degrees C). The lowest temperature in which cells may be slowly frozen and still survive is 23 degrees F (-5 degrees C).

In freezing injuries, ice crystallization begins in the extracellular space using the available intracellular free water. This process of crystallization leads to hypertonicity in the extracellular space. Osmotic considerations then cause water to withdraw from the intracellular compartment, producing intracellular dehydration. This dehydration further endangers the cells enzymatic systems already compromised by the vasospastic ischemia, which is part of the body's initial protective response to cold exposure. The reduced blood flow, secondary to the vasoconstriction, probably causes capillary stasis of the red blood cells. Rapid thawing appears to delay the appearance of this stasis and reverses the process. Minor grades of dehydration with its hypovolemic aspects, which commonly exist before the frostbite injury, will further compound the cellular damage caused by the freezing.

This is another reason for medical personnel to be continually vigilant about adequate hydration in the operating forces. Freezing injuries to the tissues are apparently a combination of direct cellular wounding and progressive tissue infarction due to multiple thrombosis of vessels supplying the injured part with clumped red cells and denatured protein sludge. Some of these changes, especially the vascular, are reversible. This fact makes aggressive measures designed to rewarm and to support tissue perfusion essential.

Classification and Pathogenesis

The lack of significant warning symptoms emphasizes the insidious nature of the frostbite injury, which is casually overlooked by many individuals temporarily subjected to cold conditions. The only symptoms may be tingling, stinging, or at most a dull aching sensation of the exposed part followed by numbness. The skin may appear red and then pale or waxy white. At this stage the affected part may feel like a block of wood. If the tissue has frozen, it appears "dead white" and is hard or even brittle, with a complete lack of sensation and movement. Medical personnel rarely see these stages of cold injury because rewarming and thawing has frequently started in the field.

The differentiation of the types of cold injury (e.g., freezing vs non-freezing) may be diagnostically difficult early after rewarming. Tissue injury is largely the result of vascular damage, therefore, it is similar in all forms of cold injury. The major variable is the severity. Cold injuries encountered are usually graded injuries, which may involve superficial and deep frostbite and conceivably adjacent non-freezing injury in less exposed parts.

SUPERFICIAL FROSTBITE

This involves only the skin or the tissue immediately beneath it. There is a certain amount of whiteness or a waxy appearance. After rewarming, the frostbitten area will first become numb, mottled blue or purple, and then will swell, sting, and burn for some time. In more severe cases blisters will occur in 24 to 36 hours beneath the outer layer of the skin. These will slowly dry up and become hard and black in about two weeks. Generally, swelling of the injured area will subside if the casualty stays in bed or at complete rest. It will last much longer if the victim refuses to cooperate. Throbbing, aching, and burning of the injured part may persist for several weeks, depending on the severity of the exposure. After the swelling finally disappears, the skin will peel and remain red, tender, and extremely sensitive to even mild cold, and it may perspire abnormally for a long time.

DEEP FROSTBITE

This is a much more serious injury and its damage not only involves the skin and subcutaneous tissue, but also affects the deep tissue beneath (including the bone). It is usually accompanied by large blisters. In marked contrast to superficial frostbite, these blisters take from 3 days to a week to develop. Swelling of the entire hand or foot will also take place and may last for a month or more. During this period of swelling there may be marked limitation of mobility of the injured fingers or toes, and blue, violet, or gray (the worst) discoloration takes place. After the first two days, aching, throbbing, and shooting pain may be experienced for as long as 2 to 8 weeks. The blisters finally dry up, blacken, and slough off, sometimes in the form of a complete cast of the finger or toe, nail and all. This leaves an exceptionally sensitive, red, thin layer of new skin, which will take many months to return to normal. Occasionally, itching and abnormal perspiration persists for more than 6 months after the initial injury, and the part will suffer lengthy or permanent sensitivity to cold.

In extreme cases of severe frostbite that have not rewarmed rapidly, permanent loss of some tissue invariably occurs. In such cases the skin does not become red and blistered after it has thawed, but it turns a lifeless gray and continues to remain cold. If blisters occur, they will probably appear along the line of demarcation between the acutely frostbitten area and the healthy remainder of the limb.

In cases of acute deep frostbite of the foot, adjacent swelling can extend as high as the knee. In a week or two after the injury, the tip of the injured area begins to turn black, dry, and shrivelled, but the rest of the damaged area may progress in one of two entirely different ways: all the tissues may become black, dry and shrivelled to almost half the normal size and mummified right up to the beginning of the healthy flesh; or it may become wet, soft, and inflamed if infection enters the picture. In the dry type, the uninjured remainder of the limb usually does not become intensely swollen or painful, and there is a clear line of demarcation between damaged and undamaged tissue. In the wet type, the whole limb tends to become painful and swollen, and originally undamaged tissue may suffer serious damage unless the infection is promptly checked.

Surgical intervention is rarely needed in less than two months. Even minor surgery on frostbitten tissue should never be performed in the field. Under normal circumstances, in an extreme case in which the loss of some tissue is inevitable, despite careful treatment, the necrotic material will simply slough off at the proper time, with maximum saving of the healthy underlying tissue. Occasionally, when unsuccessful treatment has resulted in wet gangrene, hospitalization and professional surgical intervention to stop cellulitis may be needed. However, if even this type of injury is kept scrupulously clean and sterile, the proper use is made of antibiotics, and the patient constantly stays in bed and rest throughout the illness, the chances are high that autoamputation will eventually occur.

Degrees and Pathogenesis

Early evaluation of the severity of injury is difficult even to the most experienced medical officer. Definitive classification of severity into first, second, third, and fourth degree frostbite is possible only in retrospect as definitive treatment progresses. However, since at the two extremes there can be some clinical distinction, this classification of the degrees of injury has some value.

FIRST DEGREE FROSTBITE - HYPEREMIA AND EDEMA

After rewarming, the skin becomes mottled, cyanotic, red, hot, and dry. The apparent hyperemia blanches poorly on pressure, and capillary filling is sluggish or absent. There frequently is intense itching or burning and a later deep-seated ache. The swelling begins within 3 hours and may persist for 10 or more days if the individual remains on duty. Edema usually disappears in less than 5 days if the patient is kept at bed rest. Desquamation of the superficial layers of skin may begin within 5 to 10 days after the onset of the injury and may continue as long as a month.

In milder cases, symptoms may persist for several hours causing intense discomfort, but gradually disappear without serious sequelae.

In more severe case, deep aching pain, paresthesia, cyanosis, hyperhidrosis, and coldness of the injured part may appear 2 to 3 weeks after injury and may persist for many months.

SECOND DEGREE FROSTBITE - HYPEREMIA AND VESICLE FORMATION

Hyperemia, edema, and burning pain are early manifestations after rewarming. The skin becomes deep red with mottled cyanosis and feels hot and dry to the touch. Swelling begins in 2 to 3 hours. There may be a sensation of tingling and burning of gradually increasing intensity, but light touch and position senses are frequently absent.

In more severe cases, blisters and even huge blebs may appear within 6 to 12 hours. If the large clear blebs appear early and extend nearly to the tips of the involved digits, this is a valuable sign identifying the injury as second degree frostbite. Pain, usually a deep aching sensation associated with intense burning, increases and may require medication. Edema is usually not marked and disappears within 3 to 5 days after rewarming if the patient is not ambulatory. The vesicles are superficial to the germinative layer and frequently occur on the great toe and heel or the dorsum of one or more fingers. They dry, forming black eschars within 10 to 24 days after rewarming. There may be slight limitation of motion.

Throbbing or aching pain is usually noted 10 to 20 days after injury. Hyperhidrosis frequently occurs between the second and third week. Finally, the eschar gradually separates, revealing the intact skin, which is thin, soft, poorly keratinized, and easily traumatized.

THIRD DEGREE FROSTBITE - NECROSIS OF SKIN AND CUTANEOUS TISSUE

The injury involves whole skin thickness and extends into the subcutaneous tissue leading to ulceration. Vesicles may be present and are more commonly violaceous or hemorrhagic, smaller, and do not extend to the tips of the involved digits. Edema of the entire foot or hand usually appears in about 6 days. Most patients have burning, aching, throbbing, or shooting pains beginning on the fifth to the seventeenth day following an early period of anesthesia. The skin overlaying the area of third degree frostbite may form a black, hard, dry eschar. This finally separates, exposing underlying granulation tissue.

The healing process takes about 68 days to occur. Hyperhidrosis and cyanosis may appear between the fourth and tenth week after onset of the injury and persist for months, resulting in a prolonged, uncomfortable convalescence. Trauma and infection due to injury other than cold may complicate this degree of injury and may result in extensive tissue loss, systemic manifestations of infection, and even wet gangrene, which requires emergency amputation.

FOURTH DEGREE FROSTBITE - COMPLETE NECROSIS AND LOSS OF TISSUE

There is destruction of the entire thickness of the part, including the bone, which results in loss of the injured part. Upon rewarming, the skin may turn deep red or purple or appear mottled and cyanotic. The area involved is usually anesthetic, and although there may be rapidly developing edema proximal to the area of the fourth degree injury, reaching a maximum within 6 to 12 hours, the area itself may show no significant increase in volume. There is also the rapid appearance of dry gangrene and mummification.

In other fourth degree injuries, however, tissue injury progresses slowly and edema may be more prominent, but the eschar formation and gangrene are not evident until 2 to 3 weeks after the onset of the injury. Severe paresthesias may appear 3 to 13 days after rewarming. The line of demarcation becomes apparent in about 36 days and extends down to the bone between 60 to 80 days after onset of the injury.

MAJOR POINTS TO REMEMBER

The body parts affected in order of most common occurrence are:

- Nose
- Ears
- Cheeks
- Forehead
- Exposed wrists
- Feet, especially toes
- Fingers

The following precautions should be observed when treating frostbite:

- Don't rub or massage the injury. It will cause more damage.
- Don't use any creams or ointments.
- Don't rupture any blisters.
- Don't allow the victim to smoke or consume alcoholic beverages, even though the pain may be severe.
- Don't allow an injury to thaw and refreeze.
- Don't rub ice or snow on the injury.
- Avoid excess heat when rewarming the injury.

Basic principles for prevention of frostbite:

- Dress properly.
- Keep clothing clean and dry.
- Don't overextend either yourself or the people around you.
- Be aware of the signs of frostbite.
- Don't touch cold bare metal with bare skin.
- Always use a buddy system to look for early signs of frostbite.

Reliable Prognostic Signs

The classification of cold injury as to the degree is a retrospective diagnosis. Even to the experienced, early diagnosis of the severity of the cold injury is extremely difficult. In the early stages (first 48 to 72 hours) it may be possible to differentiate accurately only between superficial (loss of skin or less) or deep (loss of skin and other underlying structures) injuries. Such an estimation should be made since it has clinical as well as epidemiologic importance.

Certain observations noted in the early rewarming phase that can be of prognostic significance are indicated below:

GOOD PROGNOSTIC SIGNS:

- large, clear blebs developing early and extending to tips of the digits
- rapid return of sensation
- return to normal temperature in the injured area
- rapid capillary filling time after pressure blanching
- pink or mildly erythematous skin color that blanches

POOR PROGNOSTIC SIGNS:

- hard, white, cold, and insensitive tissue
- cold and cyanotic tissue without blebs or blisters
- complete absence of edema
- dark hemorrhagic blebs
- early mummification
- constitutional signs of tissue necrosis: fever, tachycardia, and prostration
- superimposed trauma
- cyanotic or dark red skin that does not blanch on pressure

Treatment

The treatment of frostbite will be considered at the field level (buddy system or first aid) and at the evacuation level (definitive medical facility care).

FIRST AID

In the field, medical personnel or the victim's buddy should first attempt to protect the individual from further injury. If the tactical situation permits, the injured person should be relieved of normal duty until the severity of the injury is medically assessed.

Remove the wet or constricting clothing, and protect the extremity from further injury with blankets or any other available dry material. Smoking, drinking alcohol, and applying local salves or ointments are strictly forbidden. When there is cold injury to the lower extremity, the victim will require litter evacuation with the injured part slightly elevated. If foot travel is the only means of evacuation, do not attempt thawing until a more definitive level of medical care is reached. From available evidence it appears that the "freeze - thaw - refreeze" injury is much more destructive to tissue than the trauma of

walking on frozen extremities.

DEFINITIVE CARE

When the victim arrives at a definitive care medical facility, life threatening conditions must be treated first. Systemic hypothermia is evaluated and treated if present; then attention is directed to the cold-injured portion. The extremity is rapidly rewarmed by immersing the part in a large volume container filled with water at a temperature between 104 and 108 degrees F (40 to 42 degrees C). The container may be whatever is available in the treatment facility. For example: a tub, large drum, or half of a "mount out" box. A thermometer must be available to ensure that the temperature in the container does not go above 108 degrees F (42 degrees C) as additional warm water is added. Temperatures above 108 degrees F may cause additional tissue injury to this precarious tissue and further compound the damage.

There is some research evidence to suggest that there is less danger to tissue from prolonged freezing than there is from slow thawing. If thawing is gradual, there will be some refreezing of water crystals at the melting interface. The new ice crystals are actually larger than the original, which enhances the possibility of further cellular disruption. This recrystallization is inhibited with rapid rewarming.

Warm water immersion in the temperatures previously described is the only safe, effective method of rewarming. Since this method of thawing is quite painful, analgesics and sedatives may be necessary. Continue the immersion until the distal portion of the thawed part shows a flush and is warm to the touch. If the procedure was effective, the part should remain flushed after removing it from the water. Sometimes the color that returns to the frozen extremity is not pink, but rather a dark red or purple. That color change may indicate a more serious injury. Anesthesia after the thaw is also an ominous sign. If there is prolonged cyanosis or ischemia, the medical officer must consider performing a fasciotomy. The vesicles or blebs, if they appear, will be seen in about 2 to 5 hours after the thaw is completed.

Once thawing is completed, the injured part must be protected from trauma or additional tissue damage. If the lower extremity is involved, the patient will require complete bed rest. Attempt to avoid supervening infection. Make sure the blebs are not ruptured. Since treatment of the extremity is by the open, dry method, do not use local salves or ointments. Sterile precautions must be used when treating all deep injuries. Continue bed rest until all edema subsides and all bullae have dried. If the injury involves the lower extremity and is third degree frostbite or greater, keep the patient on bed rest until all granulations have epithelized or definitive surgery has been completed.

After the initial thawing is completed, the patient must be given 20 minute whirlpool treatments twice daily in 99 degree F (37 degree C) to which hexachlorophene or povidone iodine has been added. If a whirlpool is not available, the affected part should be gently agitated back and forth in a basin containing water and hexachlorophene at the same temperature as the whirlpool. Following this, gently dry the injured part. If the injury involves the feet or hands, place sterile cotton or lamb's wool between the digits. The injury is treated with the open, sterile technique that is so familiar in burn management. The ideal time to perform physical therapy is while the patient is receiving whirlpool treatments. This early movement of the extremities and digits, which is vitally important to lessen subsequent problems with ankylosis of the joints, should be continued at the bedside if possible.

Gentle debridement is begun by the agitating effects of the whirlpool water. Premature, active surgical debridement is strongly discouraged. Unless sepsis should occur, do not consider amputation until optimal healing has occurred, which is at least 60 to 90 days after the onset of the injury.

A booster injection of tetanus toxoid should be considered for frostbite patients; however, there is a serious question as to whether tetanus antitoxin should be given to people not previously immunized. The

decision must be based on the individual case, the extent and character of the injuries incurred, and other coincidental illness or injury.

Conservatism is the keyword in the local treatment of this situation. Blebs usually appear shortly after the initial rewarming process and should be left intact. The contents of these blisters are usually sterile and so are the underlying tissues. Debride the obviously infected blebs, using strict sterile techniques and caution. Drainage from the affected area should be cultured. You are trying not only to minimize the risk of infection but also to salvage as much viable tissue as possible. The noninfected blebs generally rupture or reabsorb in about one week; and a dry, hard, black, circumferential eschar usually develops. Whenever this eschar limits the motion of the digits, particularly the interphalangeal and metacarpal-phalangeal joints, carefully split the eschar along the dorsum or lateral borders. Do not remove the eschar because the whirlpool treatment will eliminate it at a physiologic rate in about two or three weeks. When this takes place and healing tissue is revealed underneath, you may discontinue the strict sterile precautions. The whirlpool treatment and active physical therapy should be continued.

Active surgical intervention should only be considered for an anterior tibial compartment syndrome. During this syndrome, muscle edema in the tight fascial compartments causes distal ischemia due to pressure on the blood vessels. Under these circumstances, a fasciotomy may save the limb. In general, the rule with treatment of frostbite injuries is to keep the scalpel and scissors away and simply support the healing power of nature.

Using prophylactic antibiotics in treating frostbite does not appear to be beneficial unless a specific infection has been identified. Vasodilator drugs do not appear to enhance the treatment of this injury. Additionally, corticosteroid, rutin, and antihistamines, which all have been used in hopes of controlling the post-thaw edema, have not proved beneficial.

Anticoagulant therapy should help in treating frostbite when one considers the probable role that vascular damage and thrombosis has in the genesis of the injury. Studies have shown that to be effective, the anticoagulation must be started within several hours after thawing and maintained until there is maximum tissue healing. The use of anticoagulants will depend on their availability, the medical facility's ability to monitor their use, and the presence of other combat related injuries that might contraindicate anticoagulation.

The infusion of low molecular weight dextran on a daily basis for several days following thawing decreases the tissue loss following freezing injuries in animal test models. In humans, clinical trials will be necessary to determine specific dosages and the appropriate duration of therapy. For the best results, dextran should probably be started immediately after thawing and continued for about five days. The availability of dextran and using it in large numbers of casualties may make it impractical.

In humans, sympathectomy within the first 24 to 48 hours decreases edema, lessens pain, and speeds the demarcation between viable and nonviable tissues. There is no clear evidence of improved tissue survival with any surgically affected sympathetic interruption. Peripheral nerve blocks as well as intra-arterial injections of 0.5mg of reserpine injected in single doses into the injured extremity have been reported useful. Furthermore, the availability of qualified personnel with the time and equipment to perform these procedures will determine whether they are used.

Finally, the mainstay of frostbite treatment is the initial rapid rewarming in a water bath of about 105 degrees F (41 degrees C); gentle, open, dry sterile handling of the extremity; whirlpool cleansing twice daily with progressive physical therapy; and displaying patience in generous dosages. Do not attempt premature surgical intervention if you are to achieve maximum tissue recovery and the fullest rehabilitation potential for the patient. Medical personnel may find it especially difficult to maintain good morale in the frostbite patient who may face several months of enforced bed rest while watching a black, mummified

extremity undergo autoamputation. Diversional activity, positive aspects of rehabilitation, and a hopeful attitude must be stressed to bring the patient through this ordeal.

OTHER CONSIDERATIONS

While not technically classified as cold weather injuries, certain conditions do assume significance in a cold tactical situation. Medical personnel charged with the care of troops operating in the cold must be aware of these conditions.

ACUTE MOUNTAIN SICKNESS

This is a complex hormonal and cardiorespiratory response caused by hypoxia, which results from the decreased partial pressure of oxygen in the inspired air as elevation above sea level increases. The decreased alveolar oxygen will lower the oxygen in the red cells, which is followed by decreased tissue oxygenation. The syndrome of acute mountain sickness is more than just simple hypoxia; there are also complex, and as yet only partially understood, fluid compartment shifts and hormonal changes occurring in the body.

If the tactical situation requires troop concentrations at elevations in excess of 8000 feet (2438m), then acute mountain sickness or its more serious sequela of high altitude pulmonary edema (HAPE) may occur. Like the other cold injuries, this condition can be minimized if personnel have had sufficient time to acclimatize to the new elevation. Ideally, personnel should be allowed to adapt at the 5000 foot level (1525m) for a week of light activity before increasing the workload or the elevation. Then further acclimatization will be necessary at each 2000 foot increment (610 m) of elevation.

Symptoms

When troops have not had adequate acclimatization time, the symptoms of acute mountain sickness will appear in 12 to 24 hours after arrival in the mountainous area. The symptoms may appear sooner if heavy physical exertion is required or if low temperatures prevail.

These acute mountain sickness symptoms are caused by large amounts of body fluids shifting from the blood into the extravascular space. This results in too little fluid in the circulatory system and too much fluid elsewhere. There is a temporary period (24 to 48 hours) of oliguria and subsequent overload in the pulmonary circulation. Coincidentally, there is increased cerebral blood flow, cerebral edema, and subsequent CNS symptoms. The observer will note tachypnea and possibly decreased peripheral perfusion. The victim may complain of fatigue, weakness, headache, nausea, anorexia, insomnia, or mental changes such as euphoria or depression. The border between acute mountain sickness and HAPE is indistinct. If the latter condition occurs, medical personnel will note the typical signs and symptoms of acute pulmonary edema with shortness of breath; dyspnea on exertion; paroxysmal nocturnal dyspnea; and a dry, irritating cough that may produce a frothy pink or frankly bloody sputum.

Treatment

This consists of immediate evacuation of the victim to a lower altitude. Bed rest should be maintained as well as oxygen administration and fluid monitoring. Experience gained by Indian medical officers operating in the Himalayan mountains suggests that 80mg of furosemide given to victims twice daily for 4 to 5 days helps to relieve the symptoms. They have also given victims 15mg of morphine parenterally and have received good results when HAPE was recognized. Steroids will help lessen cerebral edema and its attendant symptoms (e.g. dexamethesone, 10mg IV initially, then 4mg IM Q6H for 4 to 10 days).

As in the other cold injuries, prevention of acute mountain sickness or HAPE is far better than any treatment. Acute mountain sickness is aggravated by cold temperatures. Even with recovery, the affected individual is prone to recurrence of the injury with additional exposures. Because of the poor peripheral perfusion associated with this condition, the victim is also likely to develop a specific cold injury.

CARBON MONOXIDE POISONING

Carbon monoxide, a deadly gas even in low concentrations, is particularly dangerous because it is odorless and colorless. It is not the same as fumes and can be present when there are no fumes. Also, fumes can be strong when there is no carbon monoxide.

Carbon monoxide poisoning is a form of anoxia that results from inhaling carbon monoxide gas. The gas is absorbed through the lungs and combines with hemoglobin to produce hypoxia. This development of carboxyhemoglobin interferes with the oxygen-liberating function of hemoglobin, thus causing tissue anoxia. Since carbon monoxide combines with the red blood cells about 200 times more quickly than oxygen, carbon monoxide will displace any inhaled oxygen. The toxicity of carbon monoxide increases with the altitude.

The most common sources of carbon monoxide are engine exhausts and coal stoves. Although tests reveal there is less danger of carbon monoxide poisoning from gasoline stoves or lamps, do not take this for granted, and you must constantly check for adequate ventilation. Essentially, carbon monoxide is produced by a combustion process that produces carbon or uses a carbon product as fuel.

Symptoms

The symptoms include such varied conditions as intense headache, dizziness, impaired vision, mental confusion, nausea, tinnitus, palpitations, weakness, muscle pain, general collapse, unconsciousness, and death. A classic sign of carbon monoxide poisoning is the appearance of a cherry color of the lips and skin.

Treatment

Successful treatment of carbon monoxide poisoning requires immediate removal of the victim from the area of exposure and ventilation with 100% humidified oxygen either by a tight mask or ET. It will take about 45 minutes to rid the body of 50% of the carbon monoxide using the 100% oxygen. When you are using room air (21% oxygen), it would take 4 to 5 hours to reduce the carbon monoxide by the same percentage. Following successful resuscitation observe the victim for several hours to days, depending on the severity of the intoxication. Late sequelae such as pneumonia and cerebral edema must be recognized early and treated appropriately; the former with antibiotics and other support, and the latter with dexamethasone.

As in so many other cold weather medical situations, prevention is the best treatment. To lessen the risk of carbon monoxide poisoning, carefully supervise heater discipline and fire watches; check ventilation; be sure exhaust outlets of internal combustion engines are properly placed; and finally, discourage smoking in closed spaces.

SNOW BLINDNESS

This is usually a temporary visual disturbance due to injury of the conjunctiva and the superficial cells of the cornea. The injury is caused by exposing the eyes to ultraviolet rays of the sun reflected from the snow or other highly reflective surfaces. The danger of snow blindness is greatest not on a clear day, but rather on a cloudy day or when crystalline snow mist is present.

Symptoms

There is no warning until the symptoms begin to appear from 2 to 12 hours after exposure. The victim will first complain of an irritating gritty feeling in the eyes. There may be severe pain in and over the eyes due to the conjunctival irritation. The eyes will feel hot and sticky, and tears will flow excessively. Finally, the sight becomes blurred, objects develop a pinkish tinge, and the victim may develop photophobia. Once severely affected, individuals have noted remarkable photophobia and a tendency to repeated snow blindness even with lesser degrees of exposure for periods of 5 to 7 years.

Treatment

The most effective treatment is rest in a darkened room, cool eye compresses, and analgesics or sedatives. Except for topical anesthetic drops, do not instill ocular medications, since they will not accelerate the healing process and may further irritate an already injured epithelium. The recovery period usually takes from 1 to 5 days, depending on the severity of the exposure. The individual will be incapacitated for regular duty during that time.

Like all cold related injuries, snow blindness is preventable. It can be prevented by wearing sunglasses in bright daylight or yellow glasses on overcast days. If glasses are not available, make eye protectors from cardboard cut out in the shape of eyeglasses with narrow horizontal slits for vision. In addition, you can pull a muffler or stocking cap over the eyes as temporary eye covering, or blacken the lower eyelids and cheeks with carbon to reduce the amount of light getting into the eyes. The presence of a snow blinded person in the cold is a double liability.

UPPER RESPIRATORY INFECTION (URI)

Personnel engaged in cold weather operations are always susceptible to URI or the common cold. Certain conditions may act to increase the risks. The lower humidity both in the external areas as well as in heated tents and shelters may well be a factor in lowering the local resistance of the respiratory epithelium and in increasing the likelihood of subsequent URIs. You can raise the humidity by melting snow in large mouth containers on heating stoves. This will not only raise the humidity in the heated space, but will provide a ready source of water for drinking and personal hygiene.

Medical personnel should always have an adequate supply of solid medications (liquids freeze and are heavier to transport) to provide symptomatic relief of upper respiratory problems. These would include antipyretics, analgesics, antitussives, and possibly some drying agents. You must consider the risk-benefit ratio before dispensing medications that have sedative side effects. Maintaining good hydration, adequate nutrition, and good personal hygiene helps keep the URIs at a manageable level. Complications such as pneumonia or severe sinusitis should be recognized and treated appropriately.

DENTAL PROBLEMS

The thermal effect of cooling teeth by breathing in cold air through the mouth causes them to contract slightly. If teeth are rapidly warmed by drinking a hot drink, they will expand slightly. This expansion and contraction cause cracks in fillings and weak teeth. Bacteria can then enter the cracks and tooth decay begins. Slow cooling and rewarming also allows teeth to crack. It is important to have your teeth in "Class 1" condition. To minimize thermal stresses on teeth, allow teeth to adjust to warm air first or slowly warm them with warm liquids immediately after exposure to extreme cold. Breathing through the nose instead of the mouth will also help to warm the teeth.

CONSTIPATION

The persistent problems of cold weather dehydration have already been mentioned. One of the most troublesome gastrointestinal by-products of this condition is constipation. In addition to dehydration, this condition is caused by limited diets that do not supply the necessary fruits, vegetables, and roughage. It is also caused by the troops' refusal to relieve themselves at the undesirable facilities in the cold weather environment. Again, personnel must be thoroughly instructed on the importance of hydration, a balanced diet, and the need to evacuate the bowels regularly.

Constipation, once established, can be disabling. Abdominal discomfort, headache, and hemorrhoids can result from prolonged constipation. Here again, prevention is paramount. Once the victim complains of constipation, laxatives (tablets again, liquids freeze and are more difficult to transport), enemas, and even digital disimpaction may be necessary. Hemorrhoids will require symptomatic treatment. They require incision and drainage if they are external and thrombosed. If internal, they will require soothing local analgesics such as ointments and suppositories. Sitz baths are beneficial, but the logistics probably will not make them possible.

DEHYDRATION

Most people associate dehydration with hot weather, perspiration, and inadequate consumption of fluids. Few people, however, recognize that dehydration can also occur in cold weather. This fact alone contributes significantly to the danger of cold weather dehydration. It is a definite problem and deliberate action must be taken to overcome it.

Dehydration, simply stated, is a lack of sufficient fluid intake to make up for fluids lost. Several factors contribute to dehydration in the cold. Increased water loss occurs from respiration, urination, and perspiration. Respiration is water lost when cold, dry air enters the lungs, heats up, picks up moisture and is breathed out. The body reduces its amount of fluid through urination. When rewarmed, the body will need additional fluids. Perspiration is not as noticeable in cold climates. Perspiring may increase when a person is overdressed. The thirst mechanism is apparently not as strong in cold weather, perhaps because people associate being hot with being thirsty, and they don't notice their thirst. Basal and periodic body weights with urine a specific gravity may be easily monitored in extended operations to identify dehydrated individuals. If no conscious effort is made to drink extra fluids, dehydration will occur.

Symptoms of dehydration are: (1) dark urine and urine stains on underwear; (2) lethargy, sunken eyes, headaches, constipation, nausea; (3) lack of saliva; and, (4) loss of appetite.

CONCLUSION

Medical personnel assigned to support a cold weather operation will find it a challenging, but not impossible, undertaking. The entire cold weather operation will function more smoothly if you plan ahead and carefully select your supplies. Be sure to include appropriate medications in solid form, if possible. When this is not possible, you must develop a method that will keep liquid medication from freezing. Your preparation must also include a plan for replenishing supplies and evacuating casualties. Finally, and most importantly, you must train everyone in basic cold survival and cold injury prevention by making them all believers in the philosophy that cold injuries are preventable. Remember that cold weather can make tasks harder to perform and they may take longer to do, but it does not make them impossible. With knowledge, equipment, and training, you can beat the cold.

APPENDIX D

CLOTHING DESCRIPTION AND STOCK NUMBERS

GOVERNMENT ISSUE CLOTHING

BOOTS

Navy Standard Boots, Cold Weather. Blucher type. Insulated vulcanized rubber boot. Black with buff colored sole and heel. Flexible non-slip outer sole and molded heel. Eyelets and laces for ankle and leg adjustment. Used where moisture and cold are critical factors.

Approximate Cost: \$72

8430-00-913-3409	6	Regular
8430-00-913-3410	6	Wide
8430-00-913-3411	7	Regular
8430-00-913-3412	7	Wide
8430-00-913-3413	8	Regular
8430-00-913-3414	8	Wide
8430-00-913-3415	9	Regular
8430-00-913-3416	9	Wide
8430-00-913-3417	10	Regular
8430-00-914-0341	10	Wide
8430-00-913-3418	11	Regular
8430-00-913-3419	11	Wide
8430-00-913-3420	12	Regular
8430-00-913-3421	12	Wide
8430-00-913-3422	13	Regular
8430-00-913-3423	13	Wide



SOURCE:

U.S. Navy Cold Weather Handbook for Surface Ships
Chief of Naval Operations
Surface Ship Survivability Office

AD-1

GLOVES

Navy Standard (new style) Mitten Set, Extreme Cold Weather. Supported chloroprene dipped outer shell. Flame bonded, polyurethane film laminate, nylon tricot fabric and polyurethane foam liner. Water impermeable. Improved dexterity. Three fingers plus thumb. Suited for wet environment.

Approximate Cost: \$28

8415-01-150-6198	Small
8415-01-150-6199	Medium
8415-01-150-6200	Large
8415-01-150-6201	X-Large



Navy Standard (old style) Mittens, Extreme Cold Weather. Double coated nylon twill outer shell. Knitted nylon fleece inner shell. Leather reinforced patches on palm and upper thumb. For cold and wet/cold. Worn with liner. Inferior to and replaced by mitten set described above.

Approximate Cost: \$36

8415-00-965-1752	Small
8415-00-965-1753	Medium
8415-00-965-1754	Large
8415-00-965-1755	X-Large



Mitten Set, Extreme Cold Weather. Wind-resistant sateen cotton/nylon shell; full grain deerskin leather palm, thumb and sidewalls; wool pile backing; and quilted polyester batting liner. Intended for use in cold, dry conditions.

Approximate Cost: \$24

8415-00-782-6715	Small
8415-00-782-6716	Medium
8415-00-782-6717	Large

