NAVY AND MARINE CORPS FORCE HEALTH PROTECTION COMMAND
IMPROVING READINESS THROUGH PUBLIC HEALTH ACTION

Prevention and Treatment of Heat and Cold Stress Injuries

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Chapter 1: Introduction

Introduction

This technical manual serves to contain current Navy knowledge on heat and cold stress injuries, including their causes, prevention, treatment, and effects. It seeks to document what is scientifically sound and generally accepted medical information. Experimental procedures and potential avenues of research are avoided, although possible treatments and protocols may be mentioned in some instances. The target audience for this document is the Navy medicine community, specifically corpsmen, nurse practitioners, and physicians caring for personnel potentially suffering from the effects of heat or cold stress exposure. Hyperlinks are included as a tool for physicians and others with further interest in the subject matter. This document is not meant as a replacement for the Manual of Naval Preventive Medicine NAVMED P-5010, but rather may be considered a supplement or foundational technical manual on heat and cold stress injuries. Heat and cold stress can significantly affect military readiness and performance, and historically have had major impact on military campaigns. Leaders who push personnel in the presence of heat or cold stress exposure risk disaster. Outdated, erroneous thinking about developing "dehydration tolerance" or that one's unit can "stand" more than another because they are "tougher" or "more motivated" must be replaced by leadership decisions based on proven facts about heat and cold stress exposure. To that end, this manual is a ready repository of information for the supervisor or commander.

Notification of Changes

Changes were made to this Manual on July 25, 2024. LCDR Carey B. Vincent DO, MPH MC(FS/FMF), USN.

Changes made to the Heat section of the manual May 7th, 2024. These include an updating of medications that can affect thermoregulation for heat and cold. An update for work/rest cycles are now in accordance with army standards as of 2023. Heat acclimatization now includes recent research and data to better understand how the human body adapts to heat conditions as well as acclimatization limitations. A section on temperature measurements to include Wet Bulb Globe Temperature and human measurement modalities with their strengths and weaknesses. Heat injuries are now formatted with diagnosis and treatment along with relevant images to help assess and identify injury. Heat stroke has been updated to reflect actions to take in a field setting, with additional information for follow-up once heat victim has received care at MTF or other medical facility. Websites and other links within the document have been verified as active at the time of publishing.

Changes made to the Cold section of the Manual were completed on July 25th, 2024. The changes include additional information regarding environmental cold stress factors, individual cold stress factors, fluid shifts, and water immersion. Definitions have been updated to reflect current knowledge as defined by multiple peer reviewed publications. Additional information regarding garments and clothing has been added to the Manual. Cold weather injuries have been formatted similar to Heat injuries; Diagnosis, presentation, and treatment are broken down for each type of injury with accompanying images to help with diagnosis. An updated table has been added for quick reference for actions to take.

Chapter 2: Physiology of Heat Stress

C2.1 Heat Transfer and the Human Body

Heat is transferred to and from the human body by four mechanisms: convection, conduction, radiation, and evaporation. Convection is the transfer of heat from the source by heating the surrounding medium, which is then moved to the body (such as heated air moved by a ventilation system fan) to a living area. Conduction is the transfer of heat from the source through an object or liquid that is warmed first. An example is an electric coil heating a chair that in turn heats the person sitting in the chair. Radiation is the transfer of heat from the source without warming the intervening space. Examples include heat from the sun warming the earth, and heat from a radiator warming a person. Evaporation causes the removal of heat from an object by the vaporization of liquid, as when a person cools by sweating.

C2.1.1 Heat Equation

The net amount of heat in the human body is represented by the Heat Equation (also called the "Heat Balance Equation" [PMID: 30496710][1] as follows.

Heat production (measured by the metabolic rate)

+ radiant heat gain or loss

+ convective heat gain or loss

+ conductive heat gain or loss – evaporative cooling

= heat storage (in man)

This may be represented by the formula: S=M-WK-R-C-K-E(W)

C2.1.2. Heat Dissipation

The body constantly loses heat through breathing and convection (air moving over exposed skin, especially the head and neck). Minor amounts of heat are also lost through urinating and defecating. In heat stress conditions, the body's primary mechanism of excess heat removal is sweating (the loss of perspiration through the skin).

C2.1.2.1. Sweating (Perspiration)

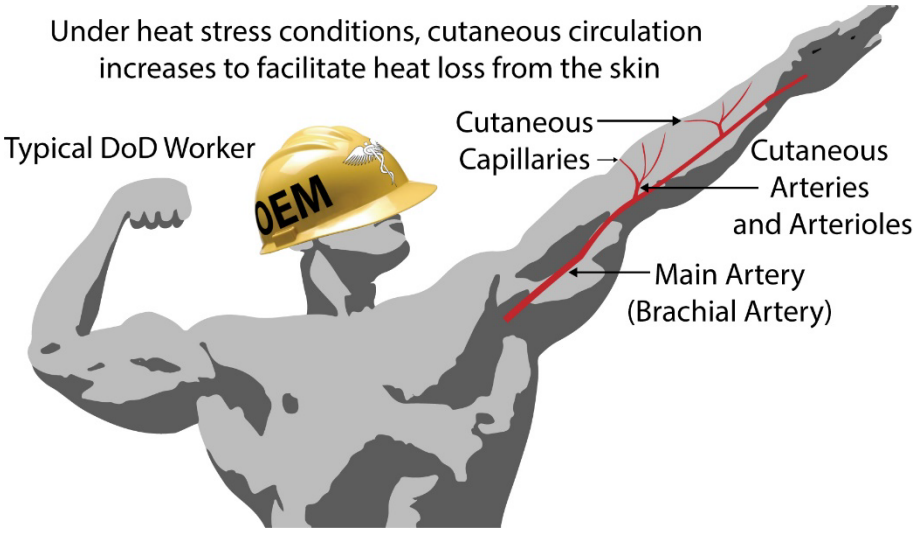
Sweating is the main mechanism the body uses to remove heat from itself in hot weather. The evaporation of sweat can cool the body due to the heat absorbed by the sweat in the heat of vaporization. Sweating depends on sufficient hydration, adequate blood flow to the skin, and proper operation of sweat glands, which are under neurologic control [PMID: 35679471].[2] Sweating efficiency in removing body heat is influenced by environmental factors, especially

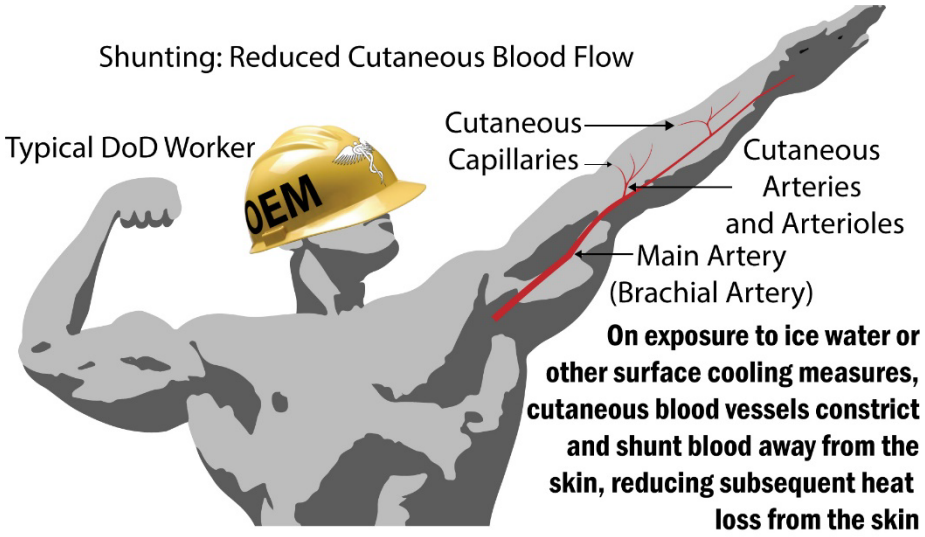
relative humidity and wind speed, and because not all sweat evaporates, especially at high rates of sweating, when sweat may run or drip off the body or be absorbed by clothing [PMID: 33440081].[3] If heat buildup continues, body temperature will become high enough to cause injury and, eventually, death. Hot dry skin may be found in conditions of heat stress when the ability to perspire has been compromised. This signals dangerous accumulation of body heat. When the core body temperature rises above 104°F (40°C), thermoregulation fails and multiorgan failure ensues [PMID: 12075060].[4]

C2.1.3 Blood Shunting

A normal response to heat stress exposure is increased cutaneous blood flow and increased sweating, resulting in increased heat loss at the skin [PMID: 481154].[5] Hot moist skin is expected in individuals perspiring normally in heat stress conditions. If there is lack or cessation of sweating (anhidrosis), heat buildup ("heat storage") will occur. The pathophysiology from heat stress is similar to that of sepsis. In heat stress conditions, blood is shunted from the splanchnic vasculature (gastric, small intestine, colonic, pancreatic, hepatic, and splenic circulations) and is redirected to the periphery (skin) in order to dissipate heat (i.e., primarily via sweating but also by convection) [PMID: 23773322].[6] During intense exercise while in heat stress conditions, blood flow to the muscles dramatically increases; instead of shunting of blood to the skin (i.e., to increase cutaneous blood flow), blood is shunted away from the skin to the muscles [PMID: 36936589].[7] If cutaneous blood flow is limited by shunting away from the skin (which also may happen in severe heat injuries), heat loss diminishes and body core temperature may rise to hazardous levels [PMID: 36936589].[8]

Figure 2-1 Normal Cutaneous Blood Flow in Heat Stress and Shunting





C2.1.4 Thirst

Thirst is a mechanism the body uses to signal the need for water replacement. In sedentary non-heat stress conditions, drinking to quench thirst adequately regulates body hydration. However, in exercise heat stress conditions, thirst sensation lags behind the actual need for water. Spontaneous drinking does not start until >2% of body weight is lost [PMID 10036337].[9] If a person only drinks to quench his or her thirst, water intake will lag behind water loss for up to several hours after heat stress conditions cease. Thus, thirst should not be the only drive for water when personnel are active in heat stress conditions. Instead, personnel should be instructed on the need to drink water replacement beyond what thirst dictates and should be encouraged to drink while in heat stress conditions.

C2.1.4.1. Hydration fluids

C2.1.4.1.1. Water

Water is an ideal rehydration fluid. It is absorbed rapidly from the upper gastrointestinal (GI) tract, and it is generally inexpensive and readily available. It is essential to make water freely available to workers, and to encourage water drinking in heat stress conditions. Sweat output can exceed fluid intake during heat stress conditions [PMID: 10919961].[10] Thirst can lag behind a water deficit by several hours. Palatability of drinks is important in stimulating intake and ensuring adequate volume replacement [PMID: 9298549].[11] Many people find plain water less palatable than flavored drinks. In severe heat stress conditions over a prolonged period (e.g., working 5 days in the desert), water alone appeared to provide adequate hydration when compared to carbohydrate-electrolyte beverages, with or without a small amount of glycerol [PMID: 8588794].[12]

C2.1.4.1.2. Water absorption

Food can delay gastric emptying and GI absorption of water. Hypotonic fluids (plain water or dilute solutions of carbohydrates) are emptied from the stomach more rapidly than fluids with higher concentrations of carbohydrates [PMID: 2733575].[13] High concentrations of sugar, complex carbohydrates, proteins, and, especially, fats, all may hinder water absorption, and thus are less desirable in water-replacement beverages. Ingestion of carbohydrate-electrolyte drinks in the post-exercise period restores exercise capacity more effectively than plain water [PMID: 9298549].[14] With increasing glucose concentration, the rate of fluid delivery to the small intestine is decreased, but the rate of glucose delivery is increased [PMID 1895359].[15] Mild exercise increases gastric emptying, while maximal exercise delays gastric emptying [PMID: 1928033].[16]

C2.1.4.2. Oral Rehydration Salts (ORS)

ORS, when mixed with the prescribed quantity of water, are sometimes also used as a rehydration liquid. However, ORS were developed as fluid replacement for GI loss and may have more salt content than is necessary. For example, ORS have about 2 grams (90 mmol) of sodium per liter [WHO].[17] Gatorade® has less than 0.5 grams of sodium per liter [Pepsico].[18] ORS are a combination of glucose, NaCl, KCl, and sodium bicarbonate. Heat stress conditions cause the body to sweat which uses the sodium chloride transport channel, transporting sodium out of the cell to the surface of the skin which causes water to follow. In conditions causing high sweat production, water loss supersedes sodium loss. Conversely, drinking ORS does not deliver enough water to counteract this problem and, furthermore, overdelivers sodium and potassium.

C2.1.4.3 Sports Drinks

Taste is a factor in fluid replacement and in that regard, drinks that taste better have an advantage over plain water. However, energy drinks generally contain a significant amount of glucose, and the caloric intake can be substantial. Five quarts of 5.8% carbohydrate solution (e.g., Gatorade®) would provide 1,120 kilocalories [Pepsico].[19] For this reason, sports drinks generally should not be used to totally replace water consumption. In one study, carbohydrate-energy drinks increased the frequency of task completion, elevated blood glucose, and reduced perceived exertion, but provided no additional benefits with regard to hydration status and physiological function during loaded walking under heat stress [PMID: 16173217].[20] The following criteria for sports drinks have been suggested by the U.S. Army: sodium 15 to 30 millimole (mmol)/L, potassium 2 to 5 mmol/L, and carbohydrate 5% to 10%; high fructose should be avoided as it may cause gastrointestinal side effects [Army].[21] Water is the preferred employer-provided hydration beverage unless workers will be expected to work long hours between meals (6 hours or more) or have unusual performance requirements (e.g., heavy labor in enclosing PPE such as chemical protective suits, certain special operations). Individuals with medical conditions affecting performance or health in heat stress conditions may also benefit from sports drinks as opposed to water; however, such decisions are the responsibility of the health care provider.

C2.1.4.3.1 Carbonated Beverages

Carbonated beverages (sodas) are prone to be acidic, cause belching, may take longer to absorb because of their high sugar content, may cause a full feeling and reduce consumption, and may be more expensive than water or sports drinks. However, because many people prefer carbonated beverages to water or sports drinks, carbonated drinks may be acceptable rehydration beverages (i.e., water is better than soda, but soda is better than nothing). For most people, carbonated beverages are not recommended as a water substitute.

C2.1.4.3.2. Alcoholic Beverages

Alcoholic beverages can cause abnormal GI absorption, vasodilation, sweating, impaired judgment, and increased urination. Thus, they may be dangerous in heat stress situations and are unacceptable as rehydration beverages.

C2.1.4.3.3. Intravenous Rehydration

Using intravenous (IV) fluids to replace fluids in heat stress conditions when no injury exists is unnecessary for healthy persons. Although plasma volume may be replaced more rapidly, research has not found IV rehydration advantageous over oral rehydration in regards to physiological strain, heat tolerance, or thermal sensations [PMID: 33829868].[22]

C2.1.5. Salt (NaCl) Tablets

Sodium (in salt) is lost through sweating. Salt tablets or salt supplements are not recommended. Normal military dietary intake is adequate to supply sufficient replacement sodium, except possibly during the first few days of heat exposure [PMID: 10919961].[23] Under certain extreme conditions, especially with protracted heat stress exposure and limited dietary sodium, supplemental salt may be required. Medical consultation should be obtained before salt supplements are used.

C2.2. Environmental Heat Stress Factors

C2.2.1. Measurement of Heat Exposure

The most obvious environmental heat stress factor is air temperature. Radiant heat (primarily from the sun but may include nearby heat sources such as furnaces) may be absorbed by the body. Relative humidity and air flow may affect heat dissipation from the body.

C2.2.1.1. Wet Bulb Globe Temperature (WBGT)

The single best measure of environmental heat stress is the WBGT, also called the WBGT Index (because it is calculated, rather than being a temperature). The WBGT takes into account air temperature, radiant heat, relative humidity, and air flow. Initially developed in the 1950's for

the military to help control and treat heat illness during training [PMID: 32490302].[24] It is calculated as follows.

WBGT = (Wet Bulb x 0.7) + (Globe Temperature x 0.2) + (Dry Bulb x 0.1)

The Wet Bulb temperature (a thermometer covered by a wet cloth or wick to account for evaporative cooling) accounts for 70% of the WBGT. Globe (or radiant) temperature (measured by a thermometer enclosed in a copper globe painted black) accounts for 20% of the WBGT. Dry Bulb temperature (a dry thermometer in the shade) accounts for 10% of the WBGT [PMID: 32490302].[25] By Navy convention, evaporation in mild airflow is considered more representative of actual work conditions, hence the name "aspirated" wet bulb (temperature is measured while air flows over the thermometer). Currently, rather than calculate the WBGT from three separate instruments, the Navy and Marine Corps use the Wet-Bulb Globe Temperature Meter, also known as the Heat Stress Meter, to measure the WBGT. The instrument displays each of these values as well as computes and displays the WBGT. Currently there are 2 models available for both ashore and at sea. The QUESTemp 48N NSN 6685-01-584-0785 (Shipboard AEL 2-870003051) and the legacy heat stress meter RSS-220 WBGT Meter, NSN 7G-6685-01-055-5298 (Shipboard AEL 2- 870003051). The RSS-220- is still operational until it can no longer be repaired or maintain calibration [NAVMED P-5010-3].[26]

C2.2.2. Wind

Wind has a cooling effect that increases with air velocity (until it is so great that it causes heating). However, in hot and dry conditions with temperature greater than 95°F (35°C), once the level of evaporative cooling is at maximum efficiency (e.g., at 0.3 m/sec air velocity), it cannot be improved with greater velocity of airflow (e.g., 2.0 m/sec) [PMID: 37372087].[27]

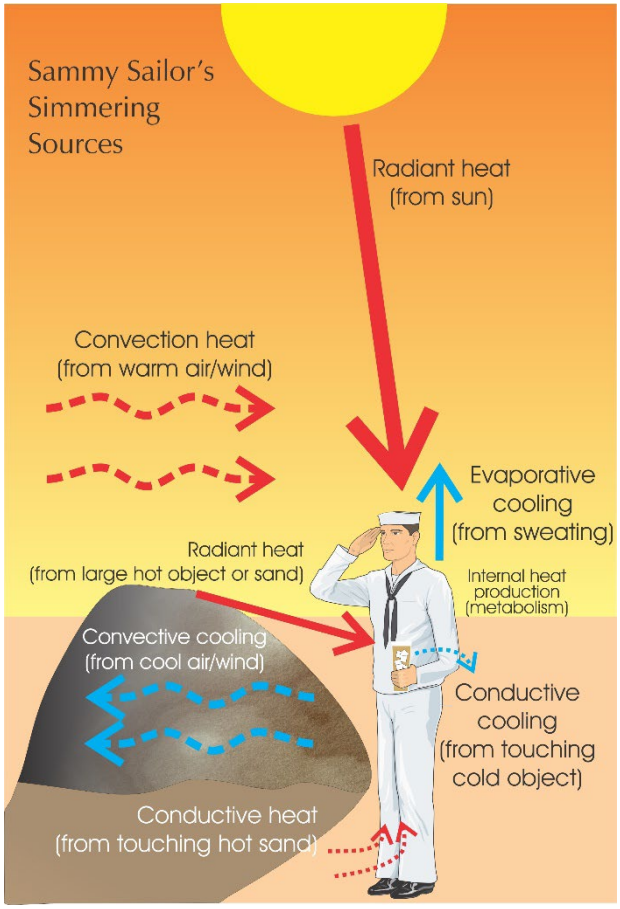
C2.2.3. Rain

Rain is generally accompanied by overcast skies (i.e., decreased direct sunlight), and raindrops are generally cooler than body temperature (and cool by conductive heat loss). However, rain is usually present with high relative humidity (decreasing evaporative cooling), and significant heat stress conditions may exist even when it is raining.

C2.2.4. Sun Position and Time of Day

The higher the position of the sun in the sky, the more radiant heat will be absorbed from surrounding terrain and from the sun. Shade afforded by trees or buildings may not only block sunlight, but on windless days may also afford relief from convective heat (hot air currents). In the winter, the sun is nearer the horizon, decreasing radiant heat absorption. (Winter sun can still present a sunburn hazard.)

Figure 2-1 Potential Heat Sources



C2.2.5. Elevation

While temperature decreases with increased elevation about 3.6°F (2°C) per 1000 feet, with decreased elevation (e.g., in deep mines) the health risk from heat stress is increased. The incidence rate ratio of heat exhaustion injuries for Australian mines operating below 1,200 meters was more than 3 times those operating above 1,200 meters [PMID: 10810098].[28]

C2.2.6 Other Radiant Heat Sources

Indoor sources of heat such as furnaces, radiators, recently cast or rolled metal (for example, rolls of sheet steel) may add significant radiant heat stress to personnel.

C2.2.7. Humidity (Relative Humidity)

High relative humidity decreases the rate of water evaporation at a given temperature. Since sweating removes body heat by evaporative cooling, conditions of high relative humidity increase heat stress because sweating becomes less effective at cooling the body. Strenuous

exercise at 85°F in 90% humidity (for example, in a temperate climate coastal area) may be more heat stressful than at 100°F in 10% humidity (for example, in an equatorial desert area) [Foster et al].[29]

C2.3 Heat Index

The "heat index" is a calculation that takes the relative humidity and the environmental temperature into account to estimate how hot conditions "feel" to people. The Heat Index is not a complete measure of heat stress conditions, it is not to be used to calculate stay times or work-rest cycles, and it is never to be substituted for the WBGT. It is mentioned here for completeness. The National Weather Service has posted a heat index equation [National Weather Service].[30] The Heat Index is an APPARENT TEMPERATURE, and may not be valid for calculated values above 135°F. Heat index tables and calculators are available on the Internet.

C2.4 Individual Heat Stress Factors

C2.4.1 Sunburn

Sunburn (a result of over-exposure to the sun, not discussed here) can decrease sweating, and thus may theoretically impair heat loss and the body's ability to tolerate heat stress (although elevated core temperature was not found in an experimental study) [PMID: 1566925].[31]

C2.4.2. Clothing and Personal Protective Equipment (PPE)

Since heat loss through sweating depends on evaporation, clothing or gear that decreases skin exposure may increase heat stress. Long sleeves and pants may decrease heat stress from radiant solar heat more than short sleeves and shorts, even though wearing long clothing may feel less comfortable [PMID: 10901990].[32] In conditions of high humidity, the heat stress added by protective clothing may be especially significant. Heat strain indices, including tolerance time, are significantly affected by extremes of humidity during both light and heavy exercise while wearing a semi-permeable nuclear, biological, and chemical protective clothing ensemble [PMID 8971493].[33] The American Conference of Governmental Industrial Hygienists (ACGIH) has suggested that WBGT values should be adjusted according to the clothing worn, although the magnitude of suggested WBGT adjustments has varied in recent decades. For example, in 2003 the suggestion was that WBGT values should be increased by 9° F (5° C) when double-cloth coveralls are worn (impermeable, water vapor resistant, and encapsulating suits excluded) [ACGIH][34]; in 2017, the suggestion was that WBGT values should be increased by 5.4°F (3.0°C) when double-cloth coveralls are worn [ACGIH 2017].[35]

C2.5 Exertion Levels, Work-Rest Cycles, And Stay Times

Administrative controls to prevent heat stress injuries include monitoring or restricting exertion levels, the duration of exertion or work before requiring a rest or cooling-off time, and the length of time personnel may remain in heat stress. Those controls, when followed carefully,

have been shown to decrease heat stress injuries. In addition, as heat stress can decrease alertness and grip strength [PMID: 36282602][36] and increase job injuries (not just heat injuries) [PMID: 23411755],[37] following those administrative controls may increase work efficiency and has been shown to decrease Navy ship refitting time.[38] (See C2.18 Activity, Rest, and Sleep.) Respirator use while working in heat stress significantly increases respiratory rate, heart rate, and, at high levels of work, systolic blood pressure. Air temperatures immediately anterior to the face of respirator wearers have been found to increase an average of 13.5°F (7.5°C). Also, as work intensity of respirator wearers increases, so does breathing resistance [PMID 1858664].[39]

C2.6. Illness

Underlying illness (infections, hypertension, diabetes, sickle cell trait [PMID: 8677839],[40] congestive heart failure [PMID: 16216975],[41] etc.) may impair the ability of the body to tolerate heat stress. Type 2 diabetics have a lower whole body heat loss compared to those without diabetes due to reduced evaporative heat loss which can increase cardiac heart rate and strain. Impaired thermoregulation and autonomic nervous system responses at high temperatures lead to increased vulnerability and ER visits [Vallianou et al].[42] Hyperthyroidism is associated with hyperthermia. Undiagnosed hyperthyroid or those who take medication that can lead to thyroid storm (see C2.15 Medications) are at increased risk for heat injury due to internal buildup of heat that is greater than heat dissipation [Walter et al].[43] Any condition associated with diarrhea, vomiting, polyuria, impaired thirst or sweating, or altered consciousness may decrease heat stress tolerance. Disabilities may hinder individuals from normal responses to heat stress (such as moving out of the sun or away from other heat sources, getting to or drinking sufficient water, etc.).

C2.7. Immunizations (Vaccinations, Inoculations)

Immunizations in progress (i.e., recent immunizations) may place additional stress on the body, possibly resulting in diminished heat stress tolerance [PMID: 10050577].[44] To date no papers have been published to show any overt link to immunizations and heat stress or illness risk.

C2.8. Prior Heat Stress Injury

A past medical history that includes heat illness or injury may limit tolerance to heat stress conditions. Such individuals may require additional time for acclimatization, and may never be able to tolerate heat stress as well as persons who have not sustained heat stress-related injury [PMID: 2406545].[45]

C2.9. Recent Heat Stress Exposure

In addition to a history of prior heat stress injury, recent exposure to heat stress conditions (e.g., the previous day) has been associated with increased risk of exertional heat illness in two studies of U.S. Marine Corps recruits [PMID: 8900989],[46] PMID: 15632673].[47] Similarly,

competition over several days in heat stress conditions has been associated with heat cramps in athletes [PMID: 8653105].[48] Whether this is limited to exercise in heat stress conditions only subsequent to some degree of tissue injury is suspected by researchers [Bergeron, 2003],[49] but is not yet established in the medical literature [PMID: 30574523].[50]

C2.10 Body Habitus

Large objects (including people) have a lower surface area-to-mass ratio (SA: mass) than small objects. Metabolic heat is generated by body tissue (which has greater mass in large individuals) and is dissipated primarily at the skin (the body surface, by skin blood flow and sweating). A lower body SA: mass ratio makes it more difficult to dissipate metabolically generated body heat. A study of Marine Corps recruits at Parris Island in the late 1980s and early 1990s demonstrated that higher body mass index (BMI) was associated with a significantly increased risk of exertional heat illness. For every unit increase in BMI there is a 3% increased risk for exertional heat stroke [Giersch et al., 2023].[51]

C2.11 Gender

Women over the last few decades have increasingly participated in endurance sports and have played roles as military combatants that were traditionally only open to men. There is no current evidence that demonstrates women are at a greater risk of heat illness when standard risk management precautions (exercise intensity, clothing, hydration) are accounted for [Yanovich et al., 2020].[52] Core body temperature of females may increase (approximately 0.5 degrees) during the luteal phase of the menstrual cycle. However, this temperature elevation has not been found to be significant in tolerating heat stress during exercise [PMID: 36936589].[53] Cardiovascular strain and orthostatic intolerance are 5 times more likely to occur in women age (18-35 years old) compared to men of similar age and health while exercising in the heat. This is due to overall smaller blood volume and lower cardiac output in addition to differences in autonomic control of blood circulation [Yanovich et al., 2020].[54] One reviewer has concluded that “aerobic capacity, surface area-to-mass ratio, and state of acclimation are more important than sex in determining physiological responses to heat stress” [PMID: 3888617].[55] The physiological adaptions from acclimatization result in increased efficiency of sweating, expansion of plasma volume, increased volume of sweating, and cutaneous vasodilation and occur in both men and women [Yanovich et al., 2020].[56]

C2.12 Pregnancy

Pregnant individuals become less tolerant of heat stress as pregnancy continues. In addition to being a source of metabolic heat and increased weight to the mother, the unborn child may also be susceptible to heat injury [PMID: 6446171].[57]

C2.13 Age

The ability for the human body to physiologically adapt to increased temperatures declines with age. Individuals over the age of 60 are especially vulnerable. Heat related illness or injuries are exacerbated with comorbidities including obesity, cardiovascular disease, diabetes, and respiratory disease [Kenny et al., 2010].[58]

C2.14 Race

No significant differences have been found regarding physiological effects of heat with regards to peripheral blood flow, resting metabolic rate, heart rate, and temperature [PMID: 6736576].[59]

C2.15 Medications

C2.15.1 Anticholinergics

Anticholinergics can disrupt thermoregulation via inhibition of the parasympathetic system that mediates sweat secretion. Inhibition of the sweating mechanism leads to reduced heat loss. Medications including atropine, found in the Mark I chemical warfare treatment injector, and scopolamine, may inhibit sweating. Pyridostigmine (an anticholinesterase pre-treatment against nerve agents) may increase rather than decrease sweating; however, the effect of pyridostigmine on heat stress tolerance is unknown. Additionally, the hypothalamic temperature set point of the temperature regulation center can be increased by the antidopaminergic activity of antipsychotics. Examples include bladder antispasmodics, tricyclic antidepressants, and neuroleptics [Lee et al].[60] These drugs block the binding of acetylcholine to the M_3 receptor, which prevents channels from opening and allowing sweat to move across the membrane to outside the body [Low et al].[61]

C2.15.2 Antidepressants

Tricyclic antidepressants impact the body's ability to sweat by the inhibition of the muscarinic sweat glands. Selective Serotonin Reuptake Inhibitors (SSRIs) can lead to Serotonin Syndrome, in which the body heats up due to decreased thermoregulation. Reuptake Inhibitors alone or in combination with other drugs, such as meperidine, fentanyl, or tramadol, enhance the availability of serotonin in the brain [Paden et al].[62]

C2.15.3 Antihypertensives

Angiotensin-Converting Enzyme Inhibitors (ACE-I) and Angiotensin Receptor Blockers (ARBs) can affect overall hydration by reducing sense of thirst, leading to dehydration via decreased fluid intake via angiotensin II activity [Cheshire, 2016].[63] Beta-blockers can reduce heat loss by convection and conduction due to being unable to increase heart rate when blood is

shunted to the peripheral circulatory system resulting in reduced blood flow to the skin [PMID: 1679517].[64]

C2.15.4. Antipsychotics

Antipsychotic drugs potentially increase the hypothalamic temperature set point through their antidopaminergic activity [Bongers et al., 2020].[65] These drugs also have a known side effect of neuroleptic malignant syndrome [UpToDate].[66]

C2.15.5 Anxiolytics

Medications such as Xanax (alprazolam), Valium (diazepam), Klonopin (clonazepam), and Ativan (lorazepam) may modify the vigilance level and alter an individual's behavior or motivation to seek out water when regulating temperature and water intake [Martin-Latry et al].[67]

C2.15.6 Antihistamines

May decrease sweating and reduce systolic blood pressure. (Also see 0C2.15.1 Anticholinergics).

C2.15.7. Diuretics

Diuretics reduce blood pressure and can cause dehydration, due to volume loss leading to consequent reductions in peripheral blood flow. Lithium may cause water loss (through diabetes insipidus) [PMID: 11246113].[68]

C2.15.8. Carbonic Anhydrase Inhibitors

Topiramate and acetazolamide inhibit sweat production at the level of the secretory coil clear cell or apex of ductal cells [De Carolis et al].[69]

C2.15.9. Psychomotor Stimulants

Psychomotor stimulants acutely raise brain metabolism by increasing the release of monoamine neurotransmitters, which can cause pathological brain hyperthermia (brain temperature > 104 °F (40 °C) that exceeds systemic hyperthermia. This effect is compounded with environmental heat stress conditions as well as with exercise [Matsumoto et al],[70] [Kiyatkin].[71] Recreational use drugs cocaine, methamphetamine, 3,4-methylenedioxy-methamphetamine (MDMA or "ecstasy"), and heroin frequently cause hyperthermia. Cocaine causes hyperthermia largely through impaired heat dissipation. Even a small dose of intranasal cocaine prior to heat stress impairs sweating and cutaneous vasodilation and heat perception [PMID: 12044126].[72] Prescribed amphetamine salts "Adderall and Ritalin" are stimulants and can lead to dehydration.

C2.15.10 Antipyretics (NSAIDs, Acetaminophen, Aspirin)

Physically active individuals who are trying to increase performance may take non-steroidal anti-inflammatory drugs (NSAIDs) for their antipyretic effects, believing that taking the drugs will keep their core temperature down thus increasing exercise duration and protecting against hyperthermia or exertional heat illness. There is no evidence that NSAIDs have any such benefit. NSAIDs can adversely affect gastrointestinal, kidney, and cardiovascular functions [Emerson et al].[73] NSAIDs may elevate the hypothalamic temperature set point, leading to less heat loss [PMID: 27768523].[74] Intoxication with drugs, such as salicylate and methyl salicylates (which may be in sports or "heat" creams, mouth wash, and oil of wintergreen), that uncouple oxidative phosphorylation can also cause hyperthermia [Clark & Lipton, 1984].[75]

C2.15.11 Miscellaneous

A recent review identified 49 medications (not generally associated with heat stress) that were associated with an increased risk of heat injury or illness [PMID: 37009927].[76]

Table 1: Medications Associated with an Increased Risk of Heat Stress Injury or Illness

Alprazolam	Ivacaftor	Risperidone
Amitriptyline	Levomepromazine	Rofecoxib
Aripiprazole	Levothyroxine	Rosuvastatin
Benzatropine	Lithium	Ruxolitinib
Canagliflozin	Losartan	Sertraline
Chlorpromazine	Mianserin	Solifenacin
Clomipramine	Naltrexone	Teriflunomide
Clonazepam	Natalizumab	Tezacaftor-ivacaftor
Clozapine	Olanzapine	Topiramate
Dimethyl fumarate	Orlistat	Trazodone
Esomeprazole	Oxybutynin	Trihexyphenidyl
Fluticasone-salmeterol	Paroxetine	Valproic acid
Gabapentin	Peginterferon Alfa-2B	Venlafaxine
Glatiramer	Pregabalin	Ziprasidone
Haloperidol	Propranolol	Zuclopenthixol
Hydrochlorothiazide	Pseudoephedrine	
Interferon beta-1A	Ramipril	

[PMID: 37009927].[77]

C2.16 Supplements

Dietary supplements are generally of no help or are detrimental to toleration of heat stress, especially heat stress associated with exercise. Caffeine is not of ergogenic benefit in endurance races during high heat stress [PMID: 8781869].[78] Other stimulants, such as pseudoephedrine, have had detrimental health effects when used in heat stress conditions (including at least one case report of use associated with heat stroke [PMID: 1943966],[79] and

should be avoided in such conditions [PMID: 10050577].[80] Ephedrine alkaloids (amphetamine like compounds derived from various species of herbs of the genus ephedra, also referred to as Ma-huang, or Ephedra equisetina) and creatine may contribute to subclinical dehydration and heatstroke in selected individuals [PMID: 12182766].[81]

C2.17 Alcohol

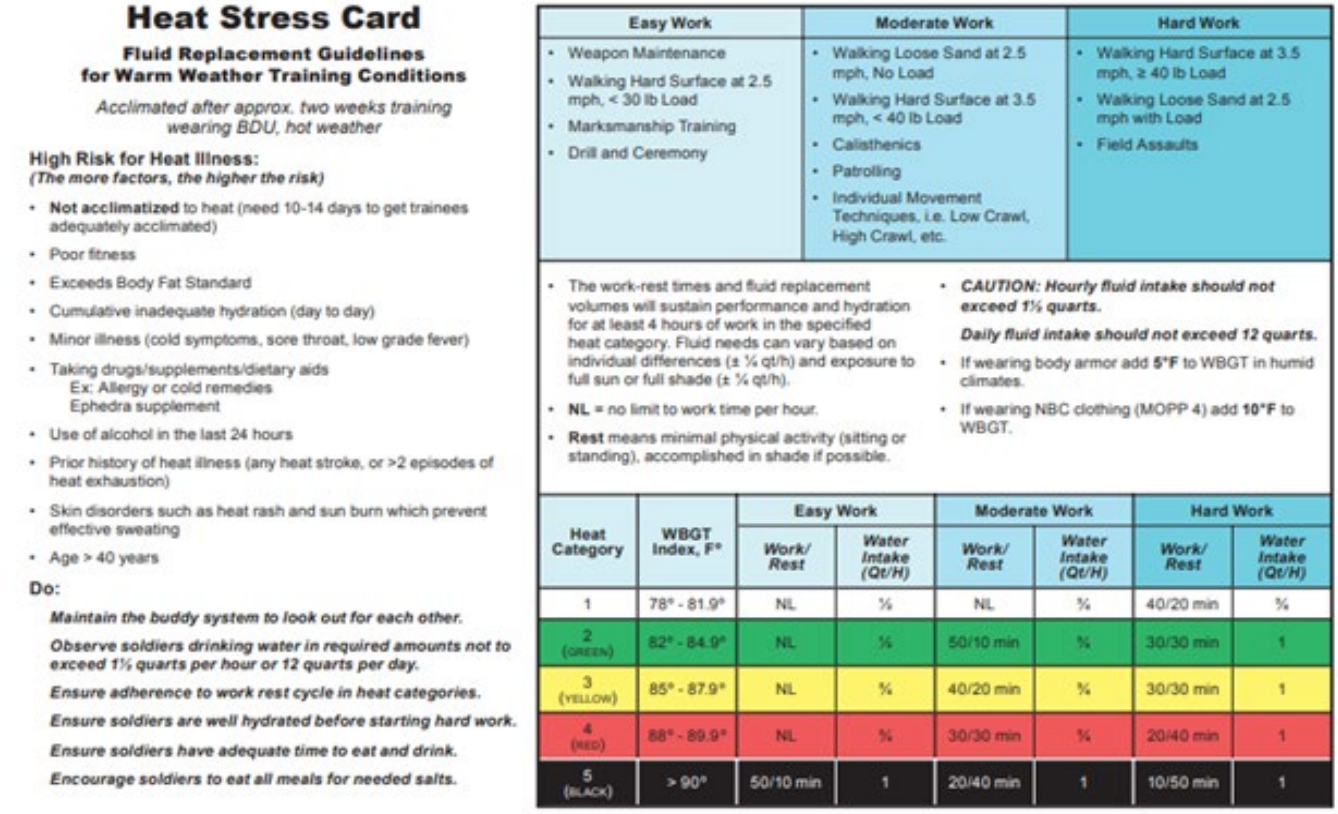
(See C2.1.4.3.2. Alcoholic Beverages.)

C2.18 Activity, Rest, and Sleep

The heat generated by metabolism can be greatly affected by a person's activity level, as muscle contraction generates a large amount of heat (oxygen metabolism increases more than 20-fold [PMID: 2583157].[82] Unfortunately, activity level is not always totally voluntary (for example, during emergency maneuvers), and the body is not sufficiently sensitive to its need to decrease activity-related heat production (the body is slow to recognize it needs to cool down). A person may run in hot weather, for example, and by the time he or she feels "too hot," he or she may have already reached a point of needing complete rest to cool sufficiently or even may suffer heat injury in spite of complete rest.

Doing work in heat stress can cause core body temperature elevations. Intermittent rest times with adequate replacement fluid available usually can prevent body core temperatures from becoming elevated. However, extremely high temperatures, heavy gear, or clothing (such as Mission Oriented Protective Posture, i.e., MOPP gear), or high work (exertion) levels may require removal from heat stress (such as resting in an air-conditioned space and/or removal of MOPP gear) for adequate core body temperature control. Rest cycles must be lengthened the higher the work levels and the greater the heat stress conditions, even requiring resting more than 2 hours after only 15 minutes of work—or working less than 15 minutes if shorter rest periods are anticipated [PMID: 12650271].[83]

Figure 2-1 Army Heat Stress Card



Heat Stress Card

Fluid Replacement Guidelines for Warm Weather Training Conditions

Acclimated after approx. two weeks training wearing BDU, hot weather

High Risk for Heat Illness:
(The more factors, the higher the risk)

- **Not acclimatized** to heat (need 10-14 days to get trainees adequately acclimated)
- Poor fitness
- Exceeds Body Fat Standard
- Cumulative inadequate hydration (day to day)
- Minor illness (cold symptoms, sore throat, low grade fever)
- Taking drugs/supplements/dietary aids
  Ex: Allergy or cold remedies
  Ephedra supplement
- Use of alcohol in the last 24 hours
- Prior history of heat illness (any heat stroke, or >2 episodes of heat exhaustion)
- Skin disorders such as heat rash and sun burn which prevent effective sweating
- Age > 40 years

Do:

Maintain the buddy system to look out for each other.

Observe soldiers drinking water in required amounts not to exceed 1½ quarts per hour or 12 quarts per day.

Ensure adherence to work rest cycle in heat categories.

Ensure soldiers are well hydrated before starting hard work.

Ensure soldiers have adequate time to eat and drink.

Encourage soldiers to eat all meals for needed salts.

Easy Work	Moderate Work	Hard Work
• Weapon Maintenance • Walking Hard Surface at 2.5 mph, < 30 lb Load • Marksmanship Training • Drill and Ceremony	• Walking Loose Sand at 2.5 mph, No Load • Walking Hard Surface at 3.5 mph, < 40 lb Load • Calisthenics • Patrolling • Individual Movement Techniques, i.e. Low Crawl, High Crawl, etc.	• Walking Hard Surface at 3.5 mph, ≥ 40 lb Load • Walking Loose Sand at 2.5 mph with Load • Field Assaults

- The work-rest times and fluid replacement volumes will sustain performance and hydration for at least 4 hours of work in the specified heat category. Fluid needs can vary based on individual differences (± ¼ qt/h) and exposure to full sun or full shade (± ¼ qt/h).
- **NL** = no limit to work time per hour.
- **Rest** means minimal physical activity (sitting or standing), accomplished in shade if possible.
- ***CAUTION: Hourly fluid intake should not exceed 1½ quarts.***
  Daily fluid intake should not exceed 12 quarts.
- If wearing body armor add **5°F** to WBGT in humid climates.
- If wearing NBC clothing (MOPP 4) add **10°F** to WBGT.

Heat Category	WBGT Index, F°	Easy Work		Moderate Work		Hard Work	
		Work/ Rest	Water Intake (Qt/H)	Work/ Rest	Water Intake (Qt/H)	Work/ Rest	Water Intake (Qt/H)
1	78° - 81.9°	NL	½	NL	½	40/20 min	¾
2 (GREEN)	82° - 84.9°	NL	½	50/10 min	¾	30/30 min	1
3 (YELLOW)	85° - 87.9°	NL	¾	40/20 min	¾	30/30 min	1
4 (RED)	88° - 89.9°	NL	¾	30/30 min	¾	20/40 min	1
5 (BLACK)	> 90°	50/10 min	1	20/40 min	1	10/50 min	1

[Army][84]

Work-rest cycle times and fluid replacement requirement charts have been developed for work in heat stress in humid and dry climates.

C2.18.1. Flag Conditions and Activity Limitations

The Navy uses a set of flags to indicate when certain heat stress hazards exist (**Error! Reference source not found.**). These are based on the Marine "Heat Condition Flag Warning System" [Enclosure 4 of Marine Corps Order 6200.1E].[85] Additional information on flag systems and their utilization procedures for ashore commands can be found in the NAVMED P-5010-3.

C2.19 Motor Vehicles

On days when temperatures exceed 86°F (30°C), the temperature inside vehicles can quickly reach 134°F to 154°F (56.7°C to 67.8°C). On clear sunny days, even with lower temperatures (72°F (22.2°C), temperatures inside vehicles can reach 117°F (47.2°C). Temperatures increase approximately 40°F (22.2°C), at an average rate of 3.2°F (1.8°C) per 5 minutes; 80% of the temperature rise occurs during the first 30 minutes. Opening ("cracking") windows 1.5 inches does not significantly decrease the rate of temperature rise in vehicles or the final maximum internal temperature [PMID: 15995010].[86]

C2.20 Hydration status

Water is essential to body cooling through the evaporation of sweat, as well as to routine physiologic processes unrelated to heat stress.

C2.20.1 Euhydration

Euhydration refers to a normal level of hydration (body water).

C2.20.2 Hypohydration

Hypohydration is having lower than normal body water (i.e., dehydration). It is sometimes used to refer only to dehydration induced prior to exercise (by limiting fluid intake, increasing urination, etc.). It is the result of inadequate fluid replacement. Hypohydration increases heat storage due to low plasma volume thereby reducing sweating rate and skin blood flow responses for a given core temperature [PMID: 11282312].[87]

C2.21 Dehydration

Properly speaking, dehydration refers to the process (rather than the state) in which total body water is decreased [USARIEM].[108] In common use, dehydration is used interchangeably with hypohydration. The term is sometimes used to connote body water deficit caused by exercise with inadequate water replacement. Dehydration increases body temperature (body temperature increases approximately 0.18°F (0.1°C) for every percent of body weight dehydration [PMID: 3569240].[88]

C2.21.1 Hyperhydration

Hyperhydration is increased total body water. It results from consuming more water than is required by the thirst mechanism (in the absence of increased sweating or other increased water loss).

C2.22 Water Intoxication

Water intoxication refers to symptomatic hyperhydration. It may occur with moderately increased fluid intake over the course of hours, or with greatly increased fluid intake (for example, more than 5 liters/hour), with hyponatremia, pulmonary edema, and cerebral edema. It has led to death in military trainees [PMID: 12053855],[89][PMID: 10091501].[90] This has led to a maximum fluid intake recommendation of 1.0 to 1.5 liter/hour. Water loss of 2.5 liters/hour during strenuous athletic competition has been documented. While replacement of such water loss is essential, it should be done with caution and, preferably, with supervision [Oh et al].[91]

C2.23 Hydration Status and Performance

Aerobic exercise tasks are likely to be adversely affected by heat stress and hypohydration [PMID: 9694412].[92] Even low levels of dehydration (2% loss of body weight) impair cardiovascular and thermoregulatory response to heat stress and reduce the capacity for exercise [PMID: 9694419].[93] Rectal temperature rise was found to be significantly greater and exercise tolerance time significantly decreased in hypohydrated subjects exercising in the heat [PMID: 9459534].[94] Hyperhydration provides no advantages over euhydration regarding thermoregulation and exercise performance in the heat [PMID: 11282312].[95]

C2.24 Acclimatization

Acclimatize (ac·cli᛫·ma·tize)**:** to adapt to a new temperature, altitude, climate, environment, or situation [Merriam-Webster].[96]

Acclimate (a᛫-kli-mate): to adapt (someone) to a new temperature, altitude, climate, environment, or situation [Merriam-Webster].[97]

When using the above terms in scientific literature there are some sources that use these words interchangeably [Stedman's].[98] Within this document when referring to acclimatization it describes adaptive physiological or behavioral changes within an organism in response to their natural climate or environment. Acclimation differs from acclimatization in that rather than adaptive processes or characteristics being changed in a natural climate or environment, the stimuli for adaptation is artificially induced and controlled in a lab or space where temperature, pressure, and oxygen can be altered and manipulated [PMID: 11252069].[99]

Table 2: Percent Optimum Heat Acclimatization

Physiologic Parameters	Day 1	Day 7	Day 14	Day 21
Rectal Temperature	6	38	72	100
Tympanic Membrane Temperature	6	37	71	100
Deep Esophageal Temperature	51	82	93	100
Mean Skin Temperature	80	93	98	100
Heart Rate	8	37	67	100
Systolic Blood Pressure	11	38	56	100
Diastolic Blood Pressure	7	36	70	100
Pulse Pressure	9	36	63	100
Mean Arterial Blood Pressure	4	35	79	100
Est. Total Vascular Resistance	8	37	70	100
Est. Cardiovascular Reserve	7	36	69	100
Sweat Rate	3	37	76	100
Urine Osmolality	3	39	82	98

Overall Percent Achievement	13	45	78	99.6

[Navy][100]

C2.24.1. Acclimatization Process Initiation

Acclimatization is accomplished by exposing individuals to heat stress over a period of days or weeks. [PMID: 5853955].[101] Studies have shown that heat acclimatization can occur in as little as 4 days and up to 21 days. It is generally accepted that taking 8 or more days to acclimatize is more effective than short-term heat acclimatization at inducing physiologic changes [PMID: 29129022].[102] At this time there are no current clear recommendations for the exact duration of how long to be in the heat in order to induce adaptations adequate for each potential heat stress environment. Multiple factors including intensity, duration, frequency, individual health status, and the number of heat exposures will change the magnitude of acclimatization [PMID: 29129022].[103] Daily heat exposure is the most effective acclimation (i.e., laboratory chamber-controlled exposures to heat) strategy, and intermittent heat exposure causes only minimal heat adaptation [PMID: 11318020].[104] Although 50% improvement in heat tolerance can be derived from 8-11 weeks of training under temperate conditions 69.8 °F (21°C), "intense training in a cool environment cannot serve as a substitute for exercise in the heat if acclimation is desired within a 2 week period" [PMID: 481157].[105] Full acclimatization may take several weeks, but two thirds or more of the adaptation is obtained within 5 days [PMID:9694427].[106] Heat acclimatization did not reduce the physiological strain and limitation of heat-exercise tolerance imposed by wearing nuclear, biological, or chemical protective clothing [PMID: 7588688].[107] The psychological strain from wearing protective clothing during vigorous exercise is not reduced by heat acclimation or by endurance training because increased sweat accumulation adds to discomfort [PMID: 9520629,[108] PMID: 8039520].[109]

C2.24.2. Water Hardening

The idea that individuals can be trained to go without water or that highly motivated individuals can tolerate more heat stress exposure is not true. People cannot be trained to require less water. Highly motivated individuals may push themselves to higher activity levels while under heat strain, and thus highly motivated individuals may be more likely to incur serious heat injury [PMID: 10063810].[110]

C2.24.3. After Acclimatization

After acclimatization, tolerance of and performance in heat stress conditions is improved. When the body is repeatedly exposed to heat stress, sweat rate increases, sweat sodium concentration decreases [PMID: 11171638],[111] plasma volume increases [PMID: 9694425],[112] and during exercise in the heat there is lowered heart rate and lowered rectal temperature [PMID: 9694427].[113] There is also a decrease in perceived exertion [PMID: 1763248].[114] Exertional heat stress was found to cause decreased cognitive performance in soldiers, but not in soldiers who had been heat-acclimated [PMID: 17357764].[115] Acclimatization improves both physical and mental performance (cognition) [PMID: 33248459].[116] Passive heat stress alone effects the kidneys by decreasing the renal plasma flow by around 30%. Exercise alone and the effect it has

on the kidneys is directly related to individual intensity of the workout. The response to exercise is inversely proportional to the fitness level of the individual. During exercise renal blood flow decreases and sodium and potassium reabsorption increases to maintain blood pressure and plasma levels. Exercise in heat stress conditions can impair the kidneys' ability to concentrate urine due to preservation of electrolyte salts. During the first few days of heat acclimatization there is a relative hypernatremia and hyperkalemia due to reabsorption of electrolytes. Over the course of the first few days the body will adapt and become more efficient with sweating and excreting salts [Chapman et al., 2021].[117]

C2.24.4. Modifiers

Heat acclimatization occurs more rapidly in persons with greater cardiopulmonary fitness due to improved efficiency and ability to accommodate increased heart rate due to increased peripheral blood flow [PMID: 1763248].[118] Heat acclimatization benefits (heart rate, core temperature) will begin to decay at a rate of approximately 2.5% for every 24 hours removed from heat conditions, with complete loss of benefit after 3 weeks [Daanen].[119] A single exercise period and/or heat exposure per week was no different from complete cessation of endurance exercise in the heat regarding loss of acclimatization-related changes in plasma volume [PMID: 36990111].[120] Heat acclimatization re-introduction, however, induces adaptations at a faster rate compared to the initial acclimatization period [Daanen].[121]

C2.24.5. Cardiovascular Performance in Heat Stress Conditions

Heat increases myocardial oxygen demand. Heat stress reduces maximum metabolic rate (VO2max) [PMID: 4039255].[122] , [PMID: 8175568].[123] Experiments have documented a lowering of cardiac stroke volume with dehydration [PMID: 10666060].[124] Acclimatization and aerobic fitness increase VO2max, but a reduction in VO2max is still caused by heat stress [PMID: 4039255].[125] Heat stress decreases cerebral blood velocity and increases cerebral vascular resistance, and physiologic responses to orthostatic challenges (e.g., increasing heart rate and blood pressure on standing up) are blunted under heat stress conditions [PMID: 16763078].[126] [PMID: 16916922].[127]

C2.24.6. Proposed Acclimatization Schedule

When feasible, heat acclimatization should be fully accomplished before strenuous exertion is required in the new environment or heat stress conditions. Two weeks of progressive heat exposure and physical work should be allowed for near complete heat acclimatization. By the end of the first week, physiologic adaptations to heat reach about 50 percent. By the end of the second week, about 80 percent of the physiologic adaptations are complete. Workers who are less fit will require additional days or weeks to fully acclimatize; very fit soldiers have been found to achieve 70 percent heat acclimatization in one week. The full effects of heat acclimatization are relative to the initial physical fitness level and the total heat stress encountered. Heat acclimatization requires a minimum exposure of two hours per day, which can be broken into two 1-hour exposures, with a physical exercise task requiring cardiovascular endurance rather than strength training. The exercise intensity should be gradually increased each day, working up to the physical intensity required for the mission. Resting in the heat, with

minimal activity, results in only partial acclimatization; physical exercise in the heat must be performed to accomplish optimal acclimatization for that work intensity in a given hot environment. A heat acclimatization strategy for military personnel has been outlined in Table 1-2. Heat acclimatization strategies by the U.S. Army Research Institute of Environmental Medicine [USARIEM].[128]

Table 3 Heat acclimatization strategies that can be considered before and after military deployment to a hot region

(1) Mimic the deployment climate.

(2) Ensure adequate heat stress by:

- Invoking profuse sweating.
- Using exercise and rest to modify the heat strain.
- Having 4 to 14 days of heat exposures.
- Maintaining the daily duration of at least 120 min.

(3) Start early (1 month before deployment).

- Performance benefits may take longer than physiological benefits.
- Be flexible with training.
- Build confidence.
- Pursue optimum physical fitness in the current climate.

(4) Methods.

- Pre-deployment: climate-controlled room or hot weather.
- Integrate with training by adding additional acclimatization sessions.
- Acclimatization with training; alternating acclimatization days with training days, and no detraining.
- Mimic the deployment environment by working out in a warm room wearing sweats (sic) if you are in a cool/temperate environment.

(5) On arrival.

- Start slowly at reduced training intensity and duration and limit heat exposure.
- Increase heat and training volume (intensity and duration) as tolerance permits.
- Acclimatize in heat of day.
- Physical training should be conducted in coolest part of day.
- Use work/rest cycles or interval training.
- Be especially observant of salt needs for the first week of acclimatization.
- Sleeping in cool or air-conditioned rooms will not affect heat acclimatization status and will aid recovery from heat stress.

C2.25 Heat Stress Vs. Heat Strain

C2.25.1. Heat Stress

Heat stress refers to the combination of all those factors which result in heat gain to the body (i.e., tend to increase the body core temperature). Heat stress is the force or load acting upon the body. Risk factors that contribute to heat stress include the follow [Adams and Jardine].[129]

- High temperature and humidity
- Direct sun exposure
- Indoor radiant heat sources (engines, hot metal, boilers, etc.)
- Limited air movement
- Illness or injury (diabetes, fever, burn, wound, etc.)
- Age
- Obesity
- Lack of acclimatization
- Pregnancy
- Medications
- Physical condition and health problems
- PPE and clothing (gear and equipment)
- Sleep deprivation.
- Physical exertion
- Fluid status (dehydration, diarrhea, vomiting)

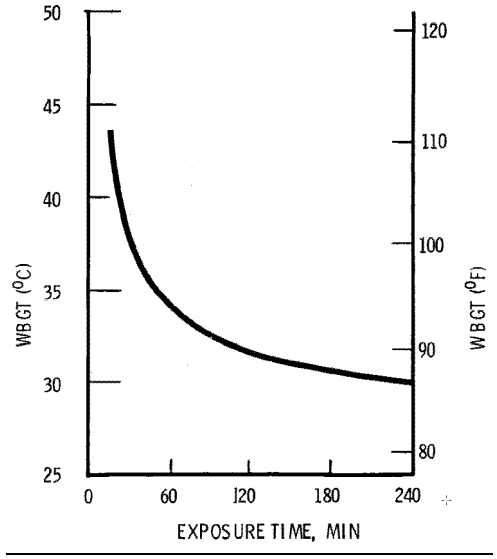
At a cellular level, cells produce heat-shock (stress) proteins, increased levels of which induce transient tolerance to a second heat stress [PMID 12075060].[130] Heat-shock protein production may play a role in acclimatization [PMID 9375300].[131] At the tissue level, there is an acute-phase response to heat stress that protects against tissue injury and promotes repair [PMID 12075060].[132] The oxidation rate of ingested carbohydrates is reduced and muscle glycogen utilization is increased during exercise in the heat compared with a cool environment [PMID 11896023,[133] PMID 11219501].[134]

Heat stress decreases cerebral blood velocity and increases cerebral vascular resistance, and physiologic responses to orthostatic challenges (e.g., increasing heart rate and blood pressure on standing up) are blunted under heat stress [PMID 16763078,[135] PMID 16916922].[136]

Severe heat stress has been noted to cause an imbalance of oxidant production and antioxidant defense (what has been called “oxidant stress”), which may lead to oxidant-mediated injury to muscle cells [PMID 10601884].[137]

Mental performance is also affected by the heat. The following graph illustrates the impact on mental performance of heat stress exposure [HEW].[138]

Figure 2-1 Upper Limits of Exposure for Unimpaired Mental Performance



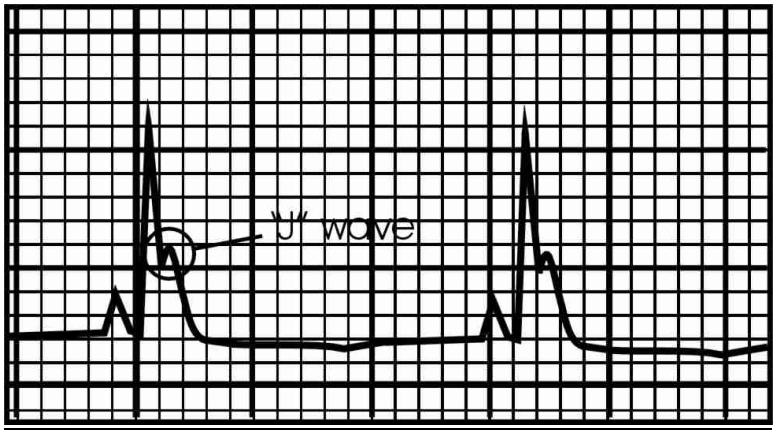
The recommended threshold WBGT value for instituting hot weather practices is 75°F or 23°C [Army].[139]

C2.26 Heat Strain

Heat strain is the resulting abnormality or "distortion" of the body's physiology when exposed to more heat stress than the body is prepared to compensate for at that time. The extreme result is the failure of the body to cool itself (thermoregulation failure), and core temperature rises (often precipitously).

Heat increases myocardial oxygen demand. Electrocardiogram (EKG) changes may include increased J-point displacement (J wave, also seen in hypothermia [Wagner],[140] S-T segment flattening (0.08 s), and prolongation of the Q-T interval with reduction in T-wave amplitude [PMID 7362568].[141]

Figure 2-1 J Wave Appearance on Electrocardiogram



C2.27. Prevention Of Heat Stress Injuries

Heat stress injuries and illnesses are preventable threats to health. Being able to identify the conditions and circumstances that lead to heat injuries, provides the opportunity to mitigate or negate these factors.

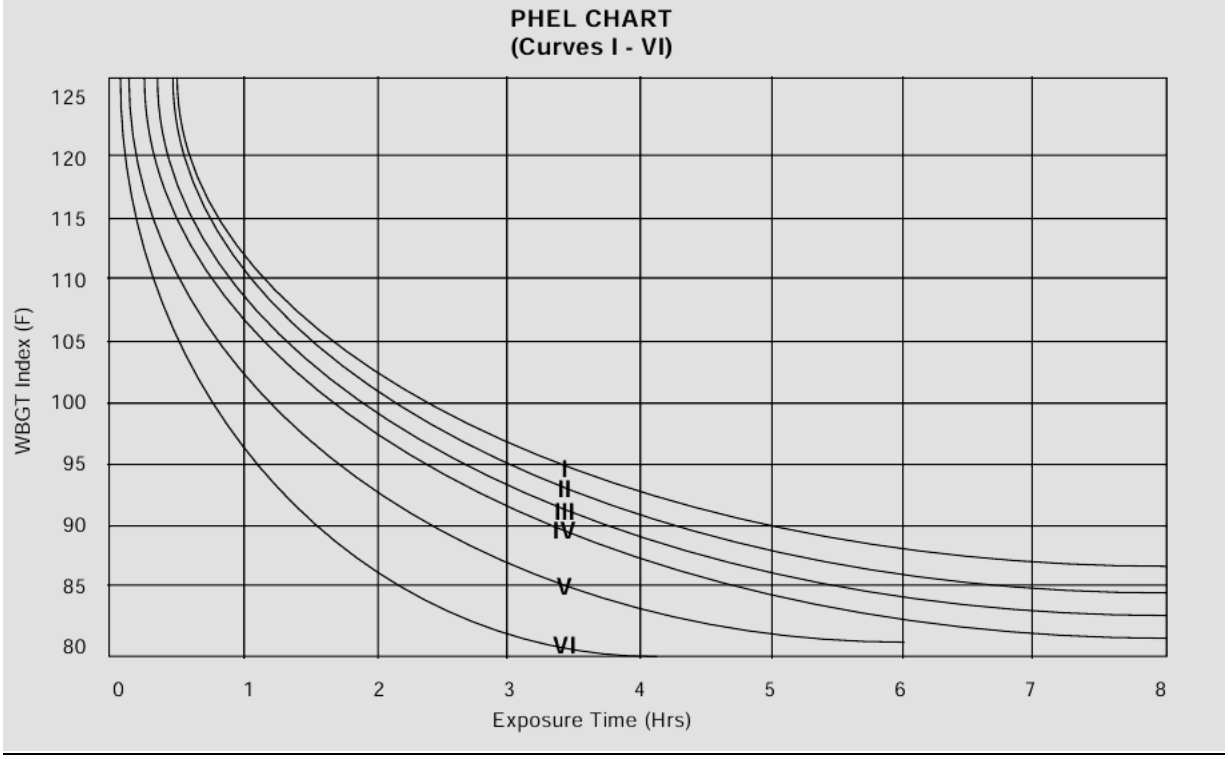
C2.28. PHEL Curves

Physiological Heat Exposure Limits graphs (PHEL curves) identify the maximal allowable exposure time or "stay time" for all U.S. Navy shipboard personnel when working in the heat [OPNAVINST 5100.19F].[142] Six categories of heat stress (PHEL curves I-VI) with different exposure times are used to protect against heat stress injuries shipboard. The correct PHEL curve is determined by considering the WBGT (rounded up to a whole number) and the work entailed by a particular job, ranging from light work (PHEL Curve I) to heavy work (PHEL Curve VI).

The PHEL curves were developed and are accurate for normal, healthy personnel who have had adequate rest (6 hours continuous sleep in the last 24 hours), adequate water intake, adequate recovery time from previous heat-stress exposure (2 hours recovery for every 1-hour exposure or 4 hours maximum), and full acclimatization to the present heat stress environment. Personnel are assumed to be wearing clothing consisting of at least 35 percent cotton fiber, not containing starch, and readily permeable to water transfer.

The limits presume that no prior heat injury or predisposing condition is present and that no cumulative heat fatigue exists prior to re-exposure. PHELs are maximum allowable standards and should be applied only in cases of short-term work exposures of up to 8 hours duration.

Figure 2-1 PHEL Curves I-VI



[NAVMED P-5010 Chapter 3][143]

Non-routine operations, such as performing operations in out-of-normal plant configurations, increases in normal watch stander work rate, and minor equipment casualties require the use of the next higher number curve. The presence of fuel vapors or combustion gases greatly reduces the safe exposure times (to approximately one-third). Stay times based on PHEL curve and work effort are shown in 4 and 5.

Table 4 Curves I-VI Exposure Times without the present of fuel vapors

Table 3-4. PHEL Time Limits for PHEL Curves I-VI Without the Presence of Fuel Combustion Gases or Fuel Vapors						
WBGT Index (F)	Total Exposure Time in Hours: Minutes					
	I	II	III	IV	V	VI
80.0	>8:00	>8:00	>8:00	8:00	6:35	4:30
81.0	>8:00	>8:00	>8:00	7:45	6:00	4:05
82.0	>8:00	>8:00	8:00	7:05	5:25	3:40
83.0	>8:00	8:00	7:45	6:25	4:55	3:20
84.0	>8:00	8:00	7:05	5:55	4:30	3:05
85.0	8:00	7:45	6:30	5:20	4:05	2:50
86.0	8:00	7:05	5:55	4:55	3:45	2:35
87.0	7:25	6:30	5:25	4:30	3:25	2:20
88.0	6:45	5:55	4:55	4:05	3:10	2:10
89.0	6:10	5:25	4:30	3:45	2:50	2:00
90.0	5:40	5:00	4:10	3:25	2:40	1:50
91.0	5:15	4:35	3:50	3:10	2:25	1:40
92.0	4:50	4:10	3:30	2:55	2:15	1:30
93.0	4:25	3:50	3:15	2:40	2:00	1:25
94.0	4:05	3:35	3:00	2:25	1:50	1:15
95.0	3:45	3:15	2:45	2:15	1:45	1:10
96.0	3:25	3:00	2:30	2:05	1:35	1:05
97.0	3:10	2:45	2:20	1:55	1:25	1:00
98.0	2:55	2:35	2:10	1:45	1:20	0:55
99.0	2:40	2:20	2:00	1:40	1:15	0:50
100.0	2:30	2:10	1:50	1:30	1:10	0:45
101.0	2:20	2:00	1:40	1:25	1:05	0:45
102.0	2:10	1:50	1:35	1:15	1:00	0:40
103.0	2:00	1:45	1:25	1:10	0:55	0:35
104.0	1:50	1:35	1:20	1:05	0:50	0:35
105.0	1:40	1:30	1:15	1:00	0:45	0:30
106.0	1:35	1:25	1:10	0:55	0:45	0:30
107.0	1:30	1:15	1:05	0:50	0:40	0:25
108.0	1:20	1:10	1:00	0:50	0:35	0:25
109.0	1:15	1:05	0:55	0:45	0:35	0:25
110.0	1:10	1:00	0:50	0:40	0:30	0:20
111.0	1:05	1:00	0:50	0:40	0:30	0:20
112.0	1:00	0:55	0:45	0:35	0:25	0:20
113.0	0:55	0:50	0:40	0:35	0:25	0:15
114.0	0:55	0:45	0:40	0:30	0:25	0:15
115.0	0:50	0:45	0:35	0:30	0:20	0:15
116.0	0:45	0:40	0:35	0:25	0:20	0:15
117.0	0:45	0:40	0:30	0:25	0:20	0:10
118.0	0:40	0:35	0:30	0:25	0:15	0:10
119.0	0:35	0:35	0:25	0:20	0:15	0:10
120.0	0:35	0:30	0:25	0:20	0:15	0:10
121.0	0:35	0:30	0:25	0:20	0:15	0:10
122.0	0:30	0:25	0:20	0:15	0:15	0:10
123.0	0:30	0:25	0:20	0:15	0:10	0:10
124.0	0:25	0:25	0:20	0:15	0:10	0:05
125.0	0.25	0.20	0.20	0.15	0.10	0.05

Table 5 PHEL Curves I-VI Exposure Times with the presence of fuel vapors

Table 3-5. PHEL Time Limits for PHEL Curves I-VI With the Presence of Fuel Combustion Gases or Fuel Vapors						
WBGT Index (F)	Total Exposure Time in Hours: Minutes					
	I	II	III	IV	V	VI
80.0	4:50	4:15	3:30	2:55	2:15	1:30
81.0	4:25	3:50	3:10	2:40	2:00	1:20
82.0	4:00	3:30	2:55	2:25	1:50	1:15
83.0	3:40	3:10	2:40	2:10	1:40	1:10
84.0	3:20	2:55	2:25	2:00	1:30	1:00
85.0	3:00	2:40	2:10	1:50	1:25	0:55
86.0	2:45	2:25	2:00	1:40	1:15	0:50
87.0	2:30	2:10	1:50	1:30	1:10	0:45
88.0	2:20	2:00	1:40	1:25	1:05	0:40
89.0	2:05	1:50	1:30	1:15	1:00	0:40
90.0	1:55	1:40	1:25	1:10	0:55	0:35
91.0	1:45	1:30	1:15	1:05	0:50	0:30
92.0	1:35	1:25	1:10	1:00	0:45	0:30
93.0	1:30	1:20	1:05	0:55	0:40	0:25
94.0	1:20	1:10	1:00	0:50	0:35	0:25
95.0	1:15	1:05	0:55	0:45	0:35	0:20
96.0	1:10	1:00	0:50	0:40	0:30	0:20
97.0	1:10	0:55	0:45	0:40	0:30	0:20
98.0	1:05	0:50	0:40	0:35	0:25	0:15
99.0	0:55	0:45	0:40	0:30	0:25	0:15
100.0	0:50	0:45	0:35	0:30	0:20	0:15
101.0	0:45	0:40	0:35	0:25	0:20	0:15
102.0	0:40	0:35	0:30	0:25	0:20	0:10
103.0	0:40	0:35	0:30	0:25	0:15	0:10
104.0	0:35	0:30	0:25	0:20	0:15	0:10
105.0	0:35	0:30	0:25	0:20	0:15	0:10
106.0	0:30	0:25	0:20	0:20	0:15	0:10
107.0	0:30	0:25	0:20	0:15	0:10	0:10
108.0	0:25	0:25	0:20	0:15	0:10	0:05
109.0	0:25	0:20	0:15	0:15	0:10	0:05
110.0	0:25	0:20	0:15	0:15	0:10	0:05
111.0	0:20	0:20	0:15	0:10	0:10	0:05
112.0	0:20	0:15	0:15	0:10	0:10	0:05
113.0	0:20	0:15	0:15	0:10	0:05	0:05
114.0	0:15	0:15	0:10	0:10	0:05	0:05
115.0	0:15	0:15	0:10	0:10	0:05	0:05
116.0	0:15	0:10	0:10	0:10	0:05	0:05
117.0	0:15	0:10	0:10	0:05	0:05	0:05

Changes in WBGT, work level or recovery time require re-calculating the remaining safe stay time. The following equation may be used:

RSST = [(1 -(Et -R/2) / Atl)] x At2

Where: RSST = remaining safe stay time (in minutes)

Et = elapsed time on station (in minutes)

R = recovery time in a cool environment (in minutes)

Atl = allowed PHEL time in first environment (in minutes)

At2 = allowed PHEL time in second environment (in minutes)

Adding intermittent rest times may extend the amount of time personnel can stay "on the job." However, exercise-rest cycles do not alter physiologic tolerance to uncompensable heat stress [PMID: 11252069].[144] PHEL curves apply only shipboard.

C2.29 Primary Prevention

Primary prevention of heat stress injuries (i.e., preventing them from occurring) is accomplished by recognizing and mitigating significant heat stress conditions and by identifying and taking steps to compensate for risk factors of heat stress injury. The main elements of primary prevention consist of adequate hydration (see C2.1.4.1. Hydration fluids), light clothing, and appropriate exercise or work limitations for the level of heat stress and PPE used.

C2.30. Secondary Prevention

Secondary prevention of heat stress injuries (i.e., treating them as early as possible) includes prompt recognition and treatment, with appropriately aggressive cooling as necessary.

C2.31. Tertiary Prevention

Tertiary prevention of heat stress injuries (limiting disability) consists of avoiding re-exposure to heat stress conditions after heat stress over-exposure has occurred. Medical clearance should be required before heat stress re-exposure if there has been a previous history of heat stress injury. After recovery, a customized gradual acclimatization process to redevelop heat tolerance may be successful. Such a process may take up to a year, and in some cases may not be possible [PMID: 2406545].[145]

Table 6 Heat Stress Injuries—Risk Factors and Predisposing Conditions

INDIVIDUAL FACTORS	ENVIRONMENTAL FACTORS
Advanced age	High temperature (>85°F)
Alcohol use	High humidity
Antihistamine use	Midday
Bromocriptine use	Hot large objects nearby (close enough to provide significant radiant heat)
Cocaine use	No air movement (wind speed = 0)
Heavy clothing, including MOPP gear.	Deep mines
High body mass index	
Insufficient sodium (salt) intake	
Insufficient water replacement	
Lack of acclimatization	
Lack of sufficient available water	
Lack of sufficient rest	
Poor physical conditioning (“out of shape”)	
Prior heat stress injury	
Respirator use	
Sleep loss.	
Stimulants	
Strenuous activity (exercise or labor)	
Sunburn	
Underlying illness (including infections, such as gastroenteritis, and chronic diseases, such as diabetes mellitus, cardiovascular disease, and congestive heart failure)	
Use of supplements/boosters such as Ephedra, Ephedrine, Ma Huang, and Guarana	

C2.32. Temperature Measurement and Thermometers

Evaluation and treatment of a heat injury requires a core temperature to assess severity and treatment progress. Being able to obtain an accurate temperature can be difficult due to environmental circumstances as well as availability of resources. There are 4 sites that are considered preferred core temperature sites: tympanic membrane, nasopharynx, esophagus, and pulmonary artery. However, these locations are limited to a clinical environment, and not practical for those operating in an austere environment, or forward deployed. Rectal thermometers must be inserted at least 6 inches and still may be unreliable [Hymczak].[146]

C2.32.1. Laser/Infrared Forehead Thermometers

This modality is relatively new and gained popularity during the COVID-19 Pandemic due to the non-contact feature as well as ease of use by non-trained medical personnel. Multiple factors can influence the result of the tests including humidity, absolute temperature, manufacturer specifications, being used in direct sunlight, barriers to skin (lotion, sweat, makeup), and the application of cold packs or other cooling methods being used [Gasim et al.].[147] False positives are also a concern for those who may be menstruating, pregnant, on hormone replacement therapy, have consumed hot beverages, or recently participating in strenuous physical activity. Infrared thermometers should not be used for the purposes of obtaining a core temperature for heat stress related applications [Aggarwal et al.].[148]

C2.32.2. Oral Thermometers

Oral thermometers are popular and are most often used to check for fever. However, they do not reflect a core temperature accurately, usually reading lower than core temperature. Recent consumption of hot (within 5 minutes [PMID: 11606822][149] or cool within 30 minutes [PMID: 11606822,[150] PMID: 3722670])[151] food or liquids, mouth breathing, improper technique, shock, and heat injury may affect either oral temperature readings or the amount they reflect true core body temperature. Oral thermometers should not be depended upon to diagnose heat stress injuries or to guide therapy of heat stress injuries.

C2.32.3. Infrared Tympanic (Ear, Aural) Electronic Thermometers

The infrared tympanic (ear) electronic thermometers have shown the highest accuracy among convenient methods for measuring core temperature under ideal conditions, [Mah et al.][152] but accuracy differs between different models and manufacturers. Furthermore, they can significantly underestimate core temperatures when affected by external conditions (such as facial fanning [PMID 9116786],[153] liquids in the ear canal (e.g., cold water used in cooling)), and improper technique. Given these confounders, tympanic thermometers do not reliably measure core body temperature under common operational conditions, thus, their use for this purpose is not recommended [PMID: 22892415].[154]

C2.32.4. Rectal Thermometers

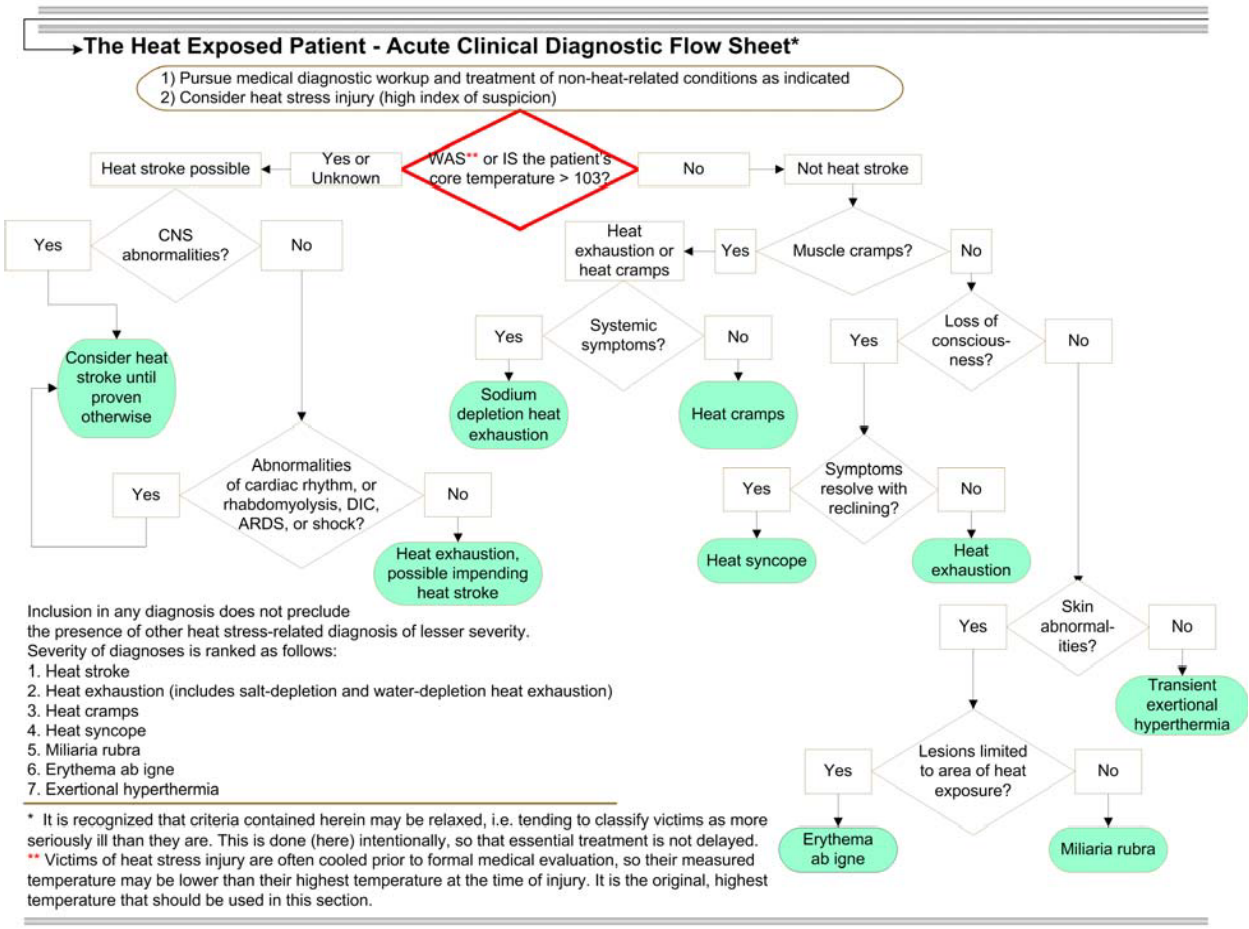
Rectal temperature measurements have historically been used to obtain a core temperature; however, research has shown rectal temperatures do not accurately reflect core temperature with current techniques. The insertion depth of approx. 2.5 cm (1 inch) used for small children does not correlate to a reliable core temperature of an adult. A study in 2021 measured bias at rectal probe depths of 4 cm, 10 cm, and 15 cm. The 15 cm depth provided the most accurate (least bias) due to the proximity to the pelvic blood supply [Hymczak,[155] Miller].[156] The type of measurement requires a thermistor that is designed for such depth. Using the standard rectal thermometer, accuracy will result in a temperature lower than true core temperature; with aggressive cooling techniques, readings may lag behind actual core body temperature [PMID: 1608386].[157] Additional considerations are rectal inflammation, hard feces that obstructs placement of thermometer, and possible perforation (especially if individual is

unconscious, resisting, or combative). If used during aggressive treatment of heat injuries, caution must be exercised not to over-treat (over-cool).

Chapter 3. Diagnosis of Heat Stress Injuries

All heat stress injuries are best recognized by having a high index of suspicion in appropriate settings. Hot climates and high humidity conditions are obvious high-risk settings. Hot workspaces, inadequate fluid replacement, or the impact of protective equipment may be less obvious. Military cases of heat stroke, although more common in summer, have occurred in the coldest part of the year [PMID: 8904496].[158] The possibility of a heat stress injury should be considered in any person with an elevated temperature not due to another cause, or that has been in a heat stress situation. An algorithm (Figure 3-1) is available to assist in the diagnosis of heat stress injury.

Figure 3-1 Acute Heat Exposed Patient Diagnostic Flow Chart



C3.1 Heat Stress Injuries

Heat stress injuries represent a continuum rather than discreet injury categories. Not all signs and symptoms are always present for each heat stress injury. Distinguishing the mildest from the most severe injuries is easy. However, heat stress injuries may include signs and symptoms of more than one injury category. For example, a case of heat exhaustion with confusion may be difficult to differentiate from heat stroke, and with inadequate treatment may in fact become a case of heat stroke.

Four major types of heat stress injuries are commonly recognized:

- Heat rash
- Heat cramps
- Heat exhaustion
- Heat stroke.

Some authors elaborate and include more heat exposure-related conditions that occur due to heat, but are not true heat injuries (as there is no actual injury to body tissue):

- Heat syncope
- Heat edema
- Heat tetany
- Erythema Ab Igne.

C3.1.1. Heat Syncope

Heat syncope (fainting) generally occurs when individuals that are not acclimatized are exposed to heat stress, most often during the first 5 days of heat exposure [PMID: 1763248].[159] A common scenario is when personnel are required to stand at attention or in formation in the heat. It is syncope from vascular insufficiency due to increased vasodilation of cutaneous blood vessels in response to the heat causing decreased central pressure (hypotension). Hypovolemia increases risk of heat syncope.

Symptoms are syncope and postural lightheadedness. Victims are tachycardic (in contrast to the bradycardia expected in vaso-vagal syncope), have normal temperatures, are sweating, and have postural hypotension.

Treatment is to place the victim in supine position, elevation of the feet, and oral or IV fluids (Normal Saline). Recovery is expected to be prompt and complete (if not recovered promptly consider heat injury).

C3.1.2. Heat Edema

Heat edema is dependent (lower extremity) edema that develops or worsens soon after heat stress exposure (usually within 48 hours). The increased cutaneous vasodilation of blood vessels in response to the heat can cause lower extremity pooling of interstitial fluid. No specific treatment is required, as the condition is expected to resolve as the acclimatization process continues [Gauer and Meyers].[160]

C3.1.3. Heat Tetany

Heat tetany is the result of hyperventilation by an individual after being exposed to heat stress. Respiratory alkalosis, resulting in decreased ionized serum calcium, may be the underlying mechanism [Schmidt,[161] PMID 2178579].[162] It generally occurs prior to acclimatization. Symptoms include muscle spasm (local or generalized) and perioral numbness and tingling. Victims are alkalotic, and blood work may show hypocarbia and high partial pressure of oxygen (pO_2). Treatment is temporary removal from heat stress. Some authors recommend that workload be decreased before resuming acclimatization [Nadel].[163]

C3.1.4. Erythema Ab Igne (Erythema Caloricum)

Erythema ab igne (erythema caloricum) is a reticulated erythematous hyperpigmented eruption that occurs after localized chronic exposure to heat [PMID: 9040977],[164] including chronic exposure to heat from fires, chairs with built-in heaters, car heaters [PMID: 9040977],[165] hot water bottles, infrared lamps, and heating pads [PMID: 7845500].[166] Central heating and not using open fires has largely reduced the incidence of erythema ab igne [PMID: 7999279],[167] [PMID: 7845500].[168] Occupational exposures causing this condition have included cooking [PMID: 1828060][169] and baking [PMID: 8772030].[170] Treatment is removal from the heat source.

Figure 3-1 Erythema Ab Igne

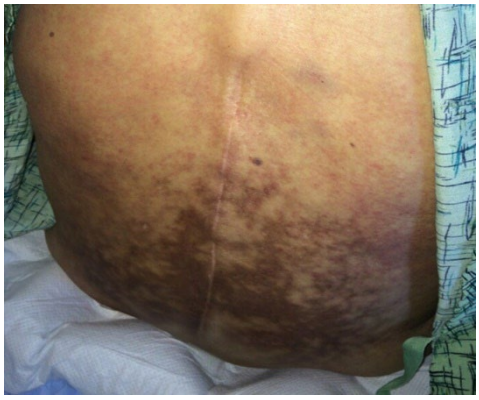


Photo used under Creative Commons license CC BY-NC 4.0 DEED.[171]

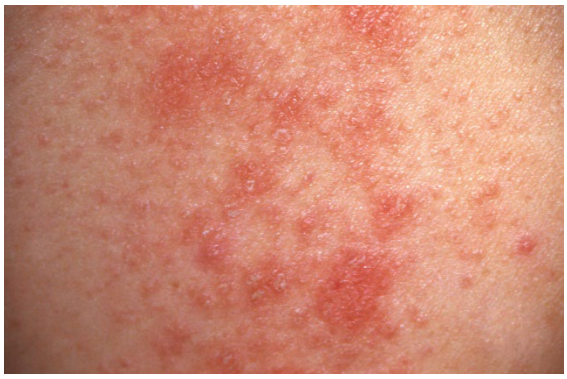
C3.2. Heat Rash (Miliaria Rubra)

Heat rash has a number of other names, including prickly heat, sweat rash, lichen infantum, lichen strophulosus, strophulus, summer rash, tropical lichen, lichen tropicus, and wildfire rash [Steadman's].[172] It is a cutaneous reaction to heat stress exposure, with redness and inflammatory skin reaction. It consists of papules and vesicles at sweat glands.

Symptoms include pruritus and a burning sensation, as well as heat intolerance [PMID: 10994246].[173] Heat rash develops when sweat pores are clogged by fluid, skin, keratin, or dirt, causing the sweat ducts to swell within the gland. This can lead to the rupture of the sweat ducts leading to a superficial vesicle on the skin that look similar to blisters. Miliaria can impair sweating and reduce heat tolerance. With continued exposure to heat, the condition can lead to heat exhaustion due to inability to produce sweat to cool down. Heat rash occurs during exercise in hot, humid environments where skin is covered or occluded by clothing [Adams and Jardine].[174] One study found that heat intolerance due to heat rash was not resolved until after 21 days [PMID 7435594].[175] If miliaria covers more than 8% of the body surface (e.g., one upper extremity, or half of a lower extremity, or one quarter of the torso, or one half of the head and neck, etc.), re-exposure to heat stress should be deferred until miliaria fully resolves.

Treatment is removal from a heat stress environment and administering antihistamines (this should be limited for cases presenting with severe itching AND the person is removed from a heat environment, in air conditioning, as antihistamines may inhibit sweating). Symptomatic relief includes application with cool wet cloths to affected areas. Cool starch baths, calamine lotion, corticosteroid lotion [Noojin,[176] Giersch],[177] sometimes with 0.25% menthol added [Merck],[178] may also be tried if necessary. This condition is considered benign, and the predominant feature is itching. However, due to scratching, heat rash can lead to skin inflammation and infections.

Figure 3-1 Miliaria



C3.3. Heat Cramps

Heat cramps occur in heat-acclimatized individuals performing vigorous physical exercise in heat stress conditions. Heat cramps in such a setting are thought to be due to hyponatremia [PMID: 8653105].[179] Continuous strenuous exercise in the heat, such as a several-day sports tournament or building project, may eventually lead to decreased electrolytes and favor development of heat cramps.

Symptoms are cramping in the affected muscles (generally those muscles that have been exercised); other than the fatigue associated with the exercise, there are generally no constitutional complaints.

Treatment is to move the victim from the heat, leg elevation, stretching, rest, and oral electrolyte fluid repletion [Gauer and Meyers].[180]

C3.4. Heat Exhaustion

Heat exhaustion has been defined as "a form of reaction to heat, marked by prostration, weakness, and collapse, resulting from severe dehydration" [Stedman's].[181] It is treated here as a distinct medical illness or injury. There are two types of heat exhaustion, distinguished by their underlying pathophysiology: sodium deficient and water deficient.

C3.4.1. Sodium-Deficient Heat Exhaustion

Sodium depletion heat exhaustion occurs when individuals exposed to excessive heat stress consume sufficient or excessive water but insufficient salt. Hyponatremia (serum sodium less than 130 mEq/L [PMID: 10530529])[182] may result from inadequate sodium in the diet (a rare occurrence in American diets), excessive sodium loss in sweat (more likely to occur prior to heat acclimatization), or water intoxication. Hyponatremia always is present, and is suggested by altered mental status or by seizures without hyperpyrexia or hypoglycemia during prolonged exercise in the heat [PMID: 10530529].[183] Treatment is water restriction and administration of hypertonic saline (or normotonic saline, if hypertonic is not available and close monitoring is not possible).

C3.4.2 Water-Deficient Heat Exhaustion

Water-deficient heat exhaustion results from excessive heat stress with inadequate water replenishment, usually due to unavailability of water. Heat exposure with increasing dehydration progressively limits the ability to tolerate heat, with virtually no ability to exercise in severe dehydration (loss of body water of 7% of body weight) [PMID: 1763248].[184]

Heat exhaustion can range from mild to severe. It is characterized by thirst, headache, dizziness, weakness, fatigue, tachycardia, ataxia, syncope, malaise, vomiting, diarrhea, and confusion [PMID: 12074531].[185] Skin is often described as "cold and clammy," although that is an inconsistent finding [Gauer and Meyers].[186]

Pathophysiology includes deficits in total body water, whole body sodium loss, and strain on the cardiovascular system. Mild forms show a slight increase in rectal temperature, nausea, vomiting and standard signs of dehydration along with circulatory insufficiency (hypotension, pale skin). Severe heat exhaustion may present with postural hypotension or syncope and altered mental status in addition to the above [Armstrong].[187] Body core temperature in heat exhaustion ranges from 101-104°F (38.3-40°C) and results in early multiorgan dysfunction [Gauer and Meyers].[188]

Treatment consists of immediate removal from the heat to a cool place and administration of water or oral sodium replacement (electrolyte) fluids (if conscious and able to swallow) or IV normal saline [Schmidt].[189] The victim should be in supine position with feet elevated, heavy clothing should be removed and victims should be allowed to rest in a shaded and ventilated

space while active cooling is initiated [Gauer and Meyers].[190] Unless more fluid is needed to maintain blood pressure, the current clinical recommendation is to limit IV saline administered in the field (i.e., pre-hospital) to 2 liters to avoid pulmonary edema [Noltkamper].[191] Heat exhaustion victims should improve rapidly with rest in the shade, cooling, and replacement of sodium. Avoidance of aspirin and other non-steroidal anti-inflammatory agents has been recommended to prevent possible paradoxical hyperthermia [Nadel].[192] Emergent transport to a hospital is appropriate if the victim is not responding to cooling, fluid, and sodium replacement or in situations where symptoms continue or worsen. Flow chart algorithms have been developed to direct emergent care of heat injuries [Sorensen].[193]

HEAT STROKE SHOULD BE SUSPECTED IN ALL PATIENTS WITH ALTERED MENTAL STATUS AND ELEVATED TEMPERATURE OR HIGH LEVELS OF EXERTION.

C3.5 Heat Stroke

Heat stroke is the most serious heat stress exposure-related illness (injury). Early or impending heat stroke may go unrecognized [PMID: 2910771].[194] It is defined as a seriously elevated temperature (> 104°F or 40°C) [PMID: 12075060][195] resulting in central nervous system (CNS) injury, caused by heat stress conditions beyond the compensatory (cooling) ability of the body. HEAT STROKE IS A LIFE-THREATENING EMERGENT MEDICAL CONDITION. Without immediate treatment, victims will sustain permanent injury or death.

C3.5.1. Non-Exertional Heat Stroke (Classic Heat Stroke)

Non-exertional heatstroke is the most common type of heat stroke that affects the elderly and those with underlying disease or other debilitating condition [Morris & Patel].[196] It is most common during summer months and tends to occur in outbreaks (e.g., associated with a “heat wave”). Excessive heat, high humidity, decreased sweating, and dehydration are mechanisms involved [Simon].[197] However, maximum daily temperature and humidity have not been found to be good predictors of the number of heat stroke injuries [PMID: 11235827].[198]

Non-exertional heatstroke risk factors for the military population include the following.

- Previous heatstroke

  Barracks or tents with restricted air movement or poor air circulation

- Medical history and medications (see C2.15 Medications)
- Alcohol consumption (which may cause dehydration)
- Physical activity (e.g., exertion in exceptionally hot environments during work or recreation—see Exertional Heat Stroke), specifically indoor activity with malfunctioning air-conditioning [PMID: 9167437].[199]

C3.5.2. Exertional Heat Stroke

Exertional heat stroke is the type most likely to be encountered in active service personnel. It tends to occur sporadically in young, active people. Increased heat production from exercising skeletal muscles is a major contributing mechanism [Simon].[200] Exercising in hot and humid conditions causes the body to store heat faster than it can release or dissipate. While hot weather or surrounding conditions increase the heat stress on an individual, exertional heat stroke can still occur at much cooler temperatures—even below 75°F due to multiple factors [PMID: 2107465].[201] When the body is heated beyond the thermal threshold, this results in cell damage which can lead to organ dysfunction and death [Navarro,[202] PMID: 2406545,[203] Simon,[204] PMID: 8990839].[205]

Table 7 Predisposing Factors for Exertional Heat Stroke

- Sleep loss.
- Generalized fatigue [PMID 2406545][206]
- Inappropriately heavy clothing
- Exposure to direct sunlight
- Skin disorders: anhidrosis, sunburn, psoriasis, etc.
- Low physical fitness
- Overweight/obesity [Navarro et al.][207]
- Dehydration
- Lack of cardiovascular conditioning
- Lack of acclimatization to heat [Simon][208]
- Dehydration or lack of access to water
- Underlying health problems (for example, sickle cell trait is associated with exertional collapse and sudden death characterized by rhabdomyolysis, heat stroke, and cardiac arrhythmia, and has a 40-fold increased risk of sudden death in affected soldiers during military basic training [PMID 8990839][209]
- Neuropsychiatric illness (one study found that a pre-existing neuropsychiatric disorder was associated with 7-fold increased odds of heat stroke) [PMID: 24981822][210]

C3.5.3. Criteria For Making the Diagnosis of Heat Stroke

Heat stroke is diagnosed when there is severely elevated body temperature that causes CNS injury. Clinically, it may be difficult to differentiate heat stroke from heat exhaustion with impending heat stroke. Altered CNS function without injury may exist, and prior emergency cooling may obscure an elevated temperature. The medical literature includes temperature criteria for the diagnosis of heat stroke ranging from 103.1°F (39.5°C) [PMID: 2406546][211] to 105.8°F (41°C) [PMID: 9694423].[212] If an alternative etiology is not apparent, it should be considered heat stroke until proven otherwise. Rapid cooling should be instituted immediately in such cases, continual monitoring of airway, breathing and circulation [Sorensen].[213]

C3.5.4. Diagnosis of Heat Stroke

Symptoms and signs of heat stroke include feeling overheated, weakness, fatigue, irritability, bizarre behavior, combativeness, hallucinations, loss of consciousness (often with little or no prodrome), and coma. Most cases are identified once the individual collapses. When any heat-related symptoms occur in the context of intense work or exercise in hot or humid conditions, exertional heatstroke should be the presumptive diagnosis until proven otherwise [DeGroot et al].[214]

The hemodynamic changes in severe heat exposure reflect a hyperdynamic circulation with tachycardia and high cardiac output [PMID: 10517377].[215] In one study, relative hypovolemia was more pronounced in patients with heatstroke compared to patients with heat exhaustion; signs of peripheral vasoconstriction were more often present in patients with heatstroke, while patients with heat exhaustion more often demonstrated peripheral vasodilatation [PMID: 10517377].[216] Arrhythmias are common in heat stroke. One study of heat stroke patients found sinus tachycardia in 43 percent of the patients [PMID: 8339628].[217]

Some severe cases of heat exhaustion have clinical and laboratory findings consistent with heat stroke but without clear evidence of CNS injury. Cases of severe exertional heat illness typically have rhabdomyolysis or other evidence of muscle, blood, kidney, liver, endocrine, or blood injury. Confusion, often found in severe heat exhaustion as well as in heat stroke, is generally transient, clearing readily and completely with treatment. The U.S. Army currently uses the designation “exertional heat illness,” although the literature has used that term to include all exertional heat stress illness. While such cases should be diagnosed as heat exhaustion, treatment is similar to that for heat stroke. Again, heat injuries often show more of a continuum of severity, rather than discreet diagnoses.

C3.5.5. Symptoms, Signs, and Findings in Heat Stroke

Heat stroke is, by definition, injury to the CNS due to heat. Symptoms, signs, and findings may be due to altered function of the CNS and any other tissue or organ that may be simultaneously affected or injured by heat stress. For example, rhabdomyolysis and paradoxical skin findings (cold, clammy skin instead of the profuse sweating that might be expected) are often seen with heat stroke.

Table 8 Heat Stroke Symptoms, Signs, and Findings

System	Symptom, Sign, or Finding
CNS	• Delirium • Coma • Euphoria • Hallucinations • Rapid eye movement • Tremors • Tonic contractions • Seizures • Cerebellar dysfunction (dysarthria, ataxia, downbeat nystagmus) [PMID 16311159][218] • Hemiplegic episodes • CNS hemorrhage • Cerebrospinal fluid normal pressure, elevated protein levels to 150 mg/dl, occasional pleocytosis
Liver	• Acute hepatic failure [PMID: 17226914][219] • Serum transaminase elevation (100x normal not uncommon) • Jaundice • Hypoglycemia
Kidneys	• Acute renal failure • Acute tubular necrosis (associated with rhabdomyolysis) • Myoglobinuria • Pyuria • Proteinuria • Microscopic hematuria • Granular casts
Muscles	• Cramps • Rigidity • Rhabdomyolysis • Elevated serum myoglobin • Hyperphosphatemia • Hyperuricemia • Elevated plasma creatinine kinase

Table 8 Heat Stroke Symptoms, Signs, and Findings

System	Symptom, Sign, or Finding
Blood	• Coagulation abnormalities • Altered clotting time and clot retraction. • Fibrinolysis • Platelet count usually low. • Factors V and VIII usually low • Purpura • Conjunctival hemorrhages • Disseminated Intravascular Coagulopathy • White blood cell count may be 30,000 - 40,000/ul
Cardiovascular	• Sinus tachycardia (as high as 150 beats per minute) • Widened pulse pressure. • Increased cardiac index or hypotension (late) • Central venous pressure normal or elevated • Moderate fluid requirement • Right heart dilation • Pericardial effusion • EKG abnormalities (ST-segment depression, T-wave abnormalities, and conduction disturbances)
GI	• Diarrhea • Vomiting • Mesenteric vascular constriction • Local areas of mucosal ulcerations • Hematemesis • Melena
Pulmonary	• Hyperventilation • Respiratory alkalosis • Hemoptysis • Pulmonary edema

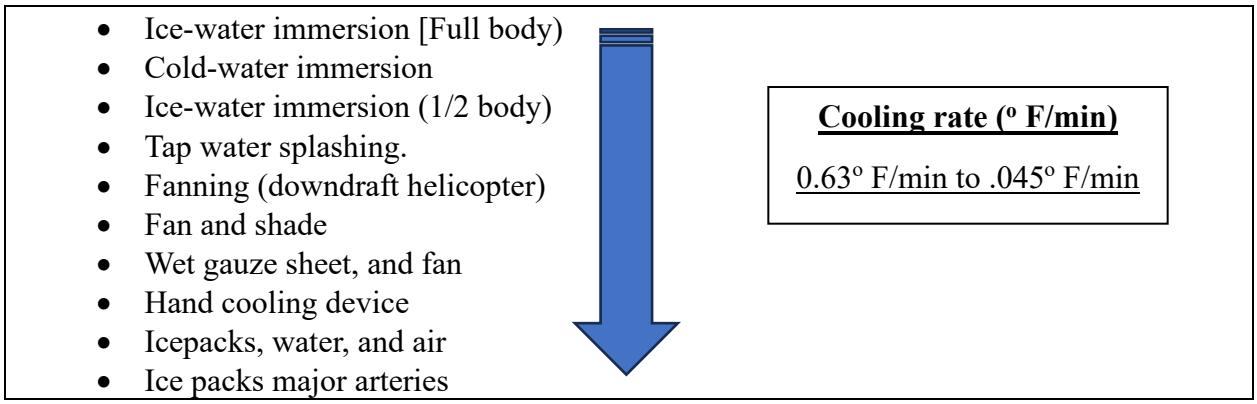
C3.6, Treatment of Heat Stroke

Comprehensive emergency management of heat stroke is beyond the scope of this manual. Specific critical issues are addressed here to give the health care provider a base of understanding from which to make clinical decisions. These patients are best treated in an intensive care setting with a multidisciplinary team.

C3.6.1 Initial Treatment of Heat Stroke

Initial treatment for heat stroke in the field is rapid cooling in addition to maintaining airway, breathing, and circulation. First aid (field) cooling should be continued while the victim is transported too Medical. Best outcomes occur when cooling is initiated within 30 minutes of presenting with the goal to reduce core body temperature to 100.4–102.2°F (38–39°C) [Sorensen].[220] The heat victim should be moved to a shaded or cooled area (preferably indoors) and gear or excessive clothing should be removed (considering CBRNE), and a rectal temperature should be obtained [Gauer and Meyers].[221] IV fluids of Normal Saline should be initiated with a maximum of 2 liters given. Emergency treatment should focus on reducing core temperature, "ABC" (airway, breathing, circulation), and IV fluids.

Figure 3-1 Optimal methods of cooling (from most to least effective)



Ice-water immersion and cold-water immersion are the most effective methods of cooling the body and bringing core temperature down [Casa,[222] McDermott].[223] With a rate of cooling (ideally 0.36 ° to 0.63°F per minute) with continual monitoring of core temperature is associated with better outcome than with a slower cooling rate [Gauer and Meyers].[224]

Where immersion is not available, pouring copious amounts of water over a heat victim while fanning can achieve a cooling rate of 0.18º F per minute [McDermott].[225] In situations where cold-water is not available or if there is airway compromise or active cardiopulmonary resuscitation, a combination of evaporative and conductive cooling methods should be employed. These include ice packs to neck, groin, axilla, fanning, and intravascular cooling [Epstein & Yanovich, 2019].[226]

Medications such as muscle relaxants, benzodiazepines, anti-seizure, dantrolene, and antipyretics are not recommended as they have not shown efficacy or improved outcomes from heatstroke [Glazer, 2005].[227]

The victim should be transported as soon as possible to a facility properly equipped to perform definitive treatment, with paramedic-level attendant or higher if available [Noltkamper].[228] During transportation, IV fluids NS should be initiated, and cooling efforts should be continued by permitting passage of air currents through the open door of the field ambulance or helicopter.

C3.6.2. Cautions While Treating Heat Stroke

As reflex hyperemia is a transient early reaction to ice water immersion, a protocol of intermittent immersion (e.g., suspending the patient over ice water and repeatedly immersing or soaking them) may be effective at cooling the victim. Immersion times can be adjusted for the victim, but example initial times may be 2 minutes in, 1 minute out.

Care must be exercised to avoid water inhalation by the patient. Vomitus, urine, blood, and fecal material may soil the water, requiring universal (isolation) precautions and making IV access and site care more difficult. Also, immersion baths must be disinfected between patients, and water in the ear canals may cause inaccurate tympanic thermometer readings.

Overcooling may result in shivering [PMID: 1608386].[229] Shivering is associated with increased involuntary muscular activity (which may accentuate tissue hypoxia and lactic acid acidosis). If simple warming measures fail to control shivering, IV benzodiazepines (such as diazepam 10 mg) may be helpful. Benzodiazepines may also be given for seizures or severe cramping [PMID: 9694424,[230] PMID: 12075060].[231]Antipyretic agents are not indicated (although aspirin may have some efficacy in treatment of platelet aggregation abnormalities in heat stroke) [PMID: 231070].[232]

C3.6.3. Electrolyte Abnormalities

In both classical and exertional heatstroke and in various animal models of human heat injury, clinical manifestations have included observations of normokalaemia, hyperkalemia, and hypokalemia [PMID: 11990141].[233] Some authors report severe hyperkalemia to be common in patients with exertional heat stroke but uncommon in those with classical heat stroke [PMID 7078400],[234] while other recent reports found hypokalemia or normokalaemia instead of hyperkalemia [PMID: 7644768].[235]

C3.7. Follow-Up Of Heat Stress Injuries

After a heat stress injury, victims are less resistant to future heat stress injuries. They are also 40% more likely to require hospitalization (although not necessarily related to heat injury) during the next four years, with that elevated rate of hospitalization decreasing over time [PMID 11528330].[236] Important aspects of post-incident care include adequate medical follow-up, careful re-exposure (or avoidance of re-exposure) to future heat stress conditions, reporting, and prevention of other heat stress injuries in the involved population [Kruijt].[237]

C3.7.1. Monitoring Health After Heat Stroke Recovery

It is recommended that all heat stroke-related abnormal studies be followed to normal after recovery. In addition, at least one careful neurological examination is recommended at 3 months after injury. Any abnormalities should be thoroughly investigated. Magnetic resonance imaging (MRI) and/or neuropsychiatric testing may be indicated.

C3.7.2. Rhabdomyolysis

Rhabdomyolysis may be recognized by discoloration of urine and should be suspected in all cases of heat stroke. It may be present in severe cases of heat exhaustion. It causes release of creatinine kinase and myoglobin into the vascular system, and may be associated with elevated uric acid, phosphate, and potassium levels (as well creatinine kinase and myoglobin). Rhabdomyolysis may lead to renal failure due to renal vasoconstriction, tubular damage caused by oxidant injury, and possibly tubular obstruction [PMID: 11430535].[238,] Urine alkalinization and increasing urine flow by osmotic diuretics such as mannitol may help minimize renal injury. Once renal injury has been sustained, hemodialysis may relieve plasma myoglobin load as well as biochemical abnormalities [PMID: 11417950].[239]

C3.7.3. Kidney Damage

Acute renal injury (acute renal failure, renal tubular necrosis) is common in exertional heat stroke, rhabdomyolysis being the major mechanism among multifactorial causes of renal failure [PMID: 7644768].[240] Uric acid may play a role in heat-related renal injury [PMID 6611841].[241] Continuous venovenous hemofiltration has been reported to be a good alternative to dialysis in hemodynamically unstable patients [PMID: 7644768].[242] Core body temperature should be monitored closely until stable.

C3.7.4. Hepatic Damage

Liver involvement is common in heat stroke, usually manifested by increased serum levels of liver enzymes, and acute liver failure has been reported [PMID: 15105986].[243] Extensive hepatocellular damage requiring liver transplant has been reported [PMID: 15838872],[244] as has spontaneous recovery of a case that initially was thought to require liver transplantation [PMID: 15105986].[245]

C3.8. Care of Residual Disability or Deficits After Heat Stroke

In one series of classic heat stroke victims, moderate to severe functional impairment was noted in 33% of patients at hospital discharge, and one year later no patient had improved functional status [PMID: 9696724].[246] One series of young heat stroke victims concluded that prominent neurological or behavioral sequelae in heat stroke victims are rare [PMID 8372119].[247] However, cases of cerebellar atrophy related to heat stroke have been reported, in which the atrophy was first noted on MRI studies 10 weeks to months after injury, and which progressed during one or more years follow-up [PMID: 9106293,[248] PMID: 7788975,[249] PMID 7575855].[250]

C3.9. Re-Exposure to Heat After Minor Heat Injuries

Most victims of minor heat stress injuries can safely be re-exposed to heat stress conditions 24 hours or less after complete recovery. Once miliaria has resolved, victims may be re-exposed to heat stress. Victims with hyperthermia (elevated core body temperature without

other apparent injury) should wait until after core body temperature has been documented to be less than 99°F (37.2°C) prior to re-exposure to heat stress conditions. Heat syncope victims should wait until core body temperature is documented to be normal prior to re-exposure to heat. Those who have experienced heat tetany may resume acclimatization after the acute condition resolves. Persons with heat edema may continue acclimatization. If edema becomes severe, they may require more gradual acclimatization. Victims with heat cramps should wait 24 hours after cramps have resolved and salt replenishment is administered (whichever is later) prior to re-exposure to heat stress.

C3.10. Re-Exposure to Heat After Major Heat Injuries

C3.10.1 Re-Exposure to Heat After Heat Exhaustion

Those who have suffered heat exhaustion should wait at least 48 hours after core body temperature, serum electrolyte values, and all heat stress-related studies have been documented as normal prior to re-exposure to heat stress. Clinical judgment by the health care provider may prolong this period on an individual basis.

C3.10.2 Re-Exposure to Heat After Heat Stroke

Recovered heat stroke victims must be recognized as having survived a life-threatening medical emergency. Re-exposure to heat must be on a case-by-case basis, according to the clinical judgment of a physician. At a minimum, it is recommended that heat stroke survivors avoid all heat stress for 2 weeks after hospital discharge and stabilization and normalization of all heat stroke-related studies. Access to air conditioning as necessary if possible is recommended. After 2 weeks, brief excursions into heat stress conditions may be allowed, as long as there is only minimal physical exertion and prompt re-entry into air conditioning is available (for example, short walks). Attempts at acclimatization after heat stroke should be delayed until at least 40 days after complete recovery (based on a series in which mean time to acclimatization was 61 days) [PMID 2406545].[251] Some victims will not successfully acclimatize until months later, if at all [PMID 2406545].[252] Some researchers have recommended testing 8 to 12 weeks after heat stroke to detect possible inability to cope with heat stress adequately [PMID: 2406544].[253]

C3.10.3 Heat Tolerance Testing

A heat tolerance test has been developed to indicate exertional heat stroke or injury victims' ability to operate under heat stress conditions. The protocol calls for 120 minutes exposure to 104°F (40°C) and 40% relative humidity in a climatic chamber while walking on a treadmill, dressed in shorts and T-shirt, at a pace of 3.1 miles per hour (5 km/h) and 2% elevation. Rectal temperature and heart rate are continuously monitored, and sweat rate is calculated [Epstein, 2016].[254] Heat tolerance testing can provide useful diagnostic utility for the prevention of a secondary exertional heat injury. The test can be done multiple times and can assist in clinical judgment and risk stratification before return to duty [Schermann].[255]

C3.10.4 Heat Re-Injury

Cardiovascular insufficiency is a major contributor to heat exhaustion and exertional heat injury, whereas heat accumulation underlies the development of heat stroke. There are no agreed upon clinical guidelines with the ability to predict who may be at a higher risk of a subsequent heat injury after exertional heat stroke [O'Connor].[256] However, most clinical guidelines recommended return to gradual activity only 7 to 21 days after clinical recovery. Additionally, no evidence has shown that clinical recovery alone represents an accurate indicator of functionality of the thermoregulatory system [Schermann].[257]

Table 9 Army activity guidelines after heat stroke [Army].[258]

Profile progression recommendations

Profile[1]	Restrictions[2]	HS without Sequelae	HS with Sequelae	Complex HS or HE/HI pending MEB
T template	Complete duty restrictions.	2 weeks	2 weeks minimum, advance when clinically resolved	2 weeks minimum, advance when clinically resolved
T template	Physical training and running, walking, swimming, or bicycling at own pace and distance not to exceed 60 minutes per day. No maximal effort; no ACFT; no wear of Interceptor Multi-Threat Body Armor (IBA); no mission oriented protective posture (MOPP) gear; no ruck marching. No airborne operations (AO).	1 month minimum	2 months minimum	Pending MEB
T template	Gradual acclimatization (see TB MED 507). No maximal effort; no ACFT; no MOPP IV gear. IBA limited to static range participation. May ruck march at own pace and distance with no more than 30 pounds. Non-tactical AO permitted.	1 month minimum	2 months minimum[3]	Not applicable
T template	Continue gradual acclimatization. May participate in unit physical training; chemical, biological, radiological, and nuclear training with MOPP gear for up to 30 minutes; IBA on static and dynamic ranges for up to 45 minutes; no record ACFT. Ruck march at own pace and distance with no more than 30 pounds up to 2 hours. Non-tactical AO permitted.	Not applicable	Pending completion of 30-day heat exposure requirement, if not accomplished during prior profile[3]	Not applicable

Notes:

[1] Temporary Profile; templates.

[2] Soldiers manifesting no heat illness symptomatology or work intolerance after completion of profile restrictions can advance and return to duty without a MEB. Any evidence/manifestation of heat illness symptomatology during the period of the profile requires a MEB referral.

[3] HS with sequelae return to full duty requires a minimum period of heat exposure during environmental stress (Heat Category 2 during the majority of included days).

C3.10.5. Reporting

All Navy heat stress-related injuries should be reported through the Naval Disease Reporting System [NMCPHC-TM 6220.12].[259] A simultaneous report to the Naval Safety Center

should be made using Risk Management Information (RMI) Streamlined Incident Reporting (SIR). Marine Corps heat injuries should be reported in accordance with MCO P5102.1A (which prescribes the mandatory use of electronic mishap reporting of all Marine Corps ground mishaps to the Marine Corps database maintained at the Naval Safety Center) and [BUMEDINST 6220.12C].[260]

C3.10.6. Prevention Of Further Heat Stress Injuries in The Population

When a heat stress injury is recognized, steps should be taken to prevent others in the involved population from heat stress injury. The victim may serve as a sentinel event, alerting health care workers, safety, and supervisors to the existence of a heat-related health risk. Training on heat stress injuries may be appropriate. A check of WBGT equipment should be done if it is possible that faulty equipment may have contributed to the heat stress injury. Adequacy of water supply, cooling facilities (HVAC system, if present), and clothing should be verified. With appropriate measures, most heat stress-related injuries can be prevented.

Chapter 4. Cold Stress Physiology

In contrast to heat stress, cold stress presents challenges to the body in retaining heat produced by metabolism.

Overall death rates from all causes increase during winter. In addition to hypothermia, cold temperature is associated with excess mortality from ischemic heart disease and cerebrovascular disease [CDC].[261] Cold temperature also can lower the immune system's resistance to respiratory infection, causing an increase in respiratory disease mortality [CDC][262] [PMID 9149695].[263] Hypothermia decreases performance [PMID 16538942],[264] increases the risk of bleeding [PMID 15211129],[265] and is an independent risk factor of mortality in trauma patients [PMID 16385283].[266]

C4.1 Heat Transfer or Loss from the Human Body

The human body is constantly producing heat by metabolism. Heat is transferred from the human body by radiation (generally to massive objects colder than the body), conduction (by touching cold surfaces or liquids), convection (by air colder than 95° F or 35° C passing over the body, and by exhaled breath that has been warmed to body temperature leaving the body), and evaporation (by sweat, water, or other liquid vaporizing from the body surface, absorbing heat as it does so). Clothing, personal protective equipment, and fat serve as insulation against heat loss from the body. Activity and certain conditions (for example, fever from infection) increase metabolism and heat production.

C4.1.2 Cold Stress, Cold Strain, and Cold Injury

When the net heat balance at a given activity level with typical clothing would result in heat loss unless the body compensates by thermoregulatory mechanisms, cold stress conditions are said to exist. When cold stress conditions are such that normal body temperature can no

longer be maintained (either generally—throughout the body—or "locally"—that is, at specific body parts, such as fingers or toes), the body may be said to be undergoing cold strain. Cold strain, if of sufficient degree or duration, may result in cold injury (either to the whole body—e.g., hypothermia, or to areas of the body—e.g., frostbite).

C4.2. Environmental Cold Stress Factors

C4.2.1 Temperature and Wind

The primary factors in thermal stress are the temperature and air movement of the immediate environment. The higher the air (wind) speed, the greater the cooling effect from convection and evaporation (termed windchill). However, as wind speed increases, friction from wind begins to generate enough heat to diminish cooling efficiency. For example, 20° F (-6.7° C) in calm or no air movement conditions ("calm temperature") becomes 13° F (–10.6° C) windchill (see **Error! Reference source not found.**) at 5 miles per hour (mph) wind speed, 4° F (–15.6° C) wind chill at 20 mph, and -4° F (–20° C) at 60 mph [NWS].[267] (The first 5 mph increase in wind speed drops the wind chill temperature 7° F, but the last 40 mph increase in wind speed drops the wind chill temperature only another 8° F.)

C4.2.2 Windchill Temperature Index

The Windchill Temperature Index is a calculation of the cooling effect on the body of cold weather conditions, taking into account temperature and wind speed.[1] The National Weather Service has recently implemented an updated equation, and future adjustments are expected to take into account sunny and cloudy sky conditions [NWS].[268] The formula used by the US and Canadian national weather services as of November 1, 2001 is:

$$\text{Windchill (°F)} = 35.74 + 0.6215T - 35.75(V^{0.16}) + 0.4275T(V^{0.16})$$

where:

V = the wind speed value in mph and

T = the temperature in °F[269]

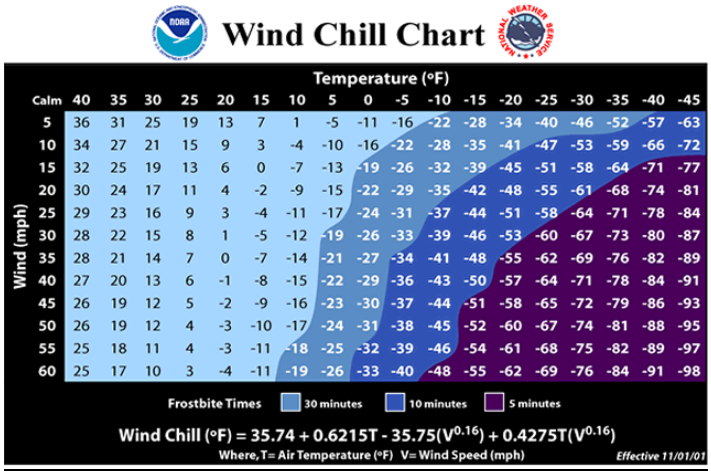
No adjustments are made for sky conditions; a clear night sky is assumed [NWS].[270]

Windchill index calculators are available on the Internet.[271]

Note: Frostbite occurs in 15 minutes or less at windchill values of -18° F (-27.8° C) or lower [NWS].[272]

[1] The term "wind chill factor" is often used to refer to the wind chill temperature index, although it may be used to refer to the significance of wind as a contributor to the effective temperature, or (perhaps more properly) to the calculation itself.

Figure 4-1 OSHA Wind Chill Chart



C4.2.3 Humidity and Moisture

Humidity affects environmental cold stress by affecting how quickly evaporation (for example, of sweat) from the skin takes place. Under dry conditions, a person with moist or wet skin who finishes exercising will lose heat rapidly. While sweat that runs or drips off does not facilitate significant heat removal in a hot environment, soaked skin or clothing may increase conductive and convective heat loss in a cold environment, especially if there is contact with cold surfaces or cold moving air.

C4.2.4 Immersion

Immersion in cold water presents special challenges to personnel. Even though seawater does not normally get below 28.6° F (-1.9° C) without freezing, water absorbs a larger amount of heat than air after penetrating clothing; i.e., water has a relatively high sensible heat, which is "the amount of heat that, when absorbed by a substance, causes a rise in temperature" [Stedman's].[273] The cold stress of immersion in 30° F (-1.1° C) seawater may be much greater than that of standing on dry land at the same temperature.

C4.2.4.1 Diving Reflex (Diving Response)

Immersion triggers the diving reflex (bradycardia response) [PMID 11816961].[274] The colder the water, the more marked the bradycardia (a 9% decrease in heart rate with facial immersion at 98.6° F or 37° C and a 29% decrease in heart rate at 37.4° F or 3° C) [York].[275]

C4.2.4.2 Fluid shifts in Cold Water

Vascular fluid shifts, body cooling, and diuresis are all greater in cold water than in cold air [PMID 3629738].[276] The percent reduction in plasma volume from cold exposure is significantly larger in cold water (-17%) than in cold air (-12%) [PMID 3629738].[277]

C4.2.4.3 Performance in Cold Water

Immersion in cold water impairs swimming performance (at 64° F or 18° C, and even more at 50° F or 10° C), which, with initial cardio-respiratory responses to immersion, probably is the major danger to cold-water immersion victims [PMID 10466663].[278] Protective effects of increased body fat against cold stress, although significant on dry land [PMID 6735815][279] and during immersion [PMID 11043627],[280] are minimal compared to the benefit of a dry suit during cold-water immersion [PMID 2803162].[281]

C4.2.4.4 Swimming Induced Pulmonary Edema (SIPE)

SIPE, also in the literature termed "cold-induced pulmonary edema," "scuba induced pulmonary edema" and "pulmonary oedema induced by strenuous swimming (SIPO)" [PMID 10854620],[282] has been associated with swimming and diving. Previously thought to be a cold-water phenomenon, recent studies (2024) now use the term Immersion Pulmonary Edema (IPE). Studies show that symptoms occur due to the level vigorous activity that takes place while diving or underwater in addition to the ventilatory constraints the breathing apparatus presents as a contributing factor. [PMID: 37979071] Symptoms include dyspnea, cough, sputum production, hypoxemia (saturation 85 to 90%), hemoptysis (in about half of victims), weakness, chest discomfort, orthopnea, wheezing, and dizziness. Physical examination may reveal rales. Chest X-ray is usually normal or with evidence of pulmonary edema, and spirometry may show a restrictive pattern. Recurrence is not uncommon (approximately 20% experience recurrence) [PMID 10854620],[283] [PMID 9068153].[284] Treatment is usually conservative, aimed primarily at resolution of pulmonary edema. Resolution of symptoms is usually within 1 or 2 days, although fatal outcomes have been reported [PMID 15796313].[285]

C4.2.4.5 Awareness of Cold Strain in Cold Water

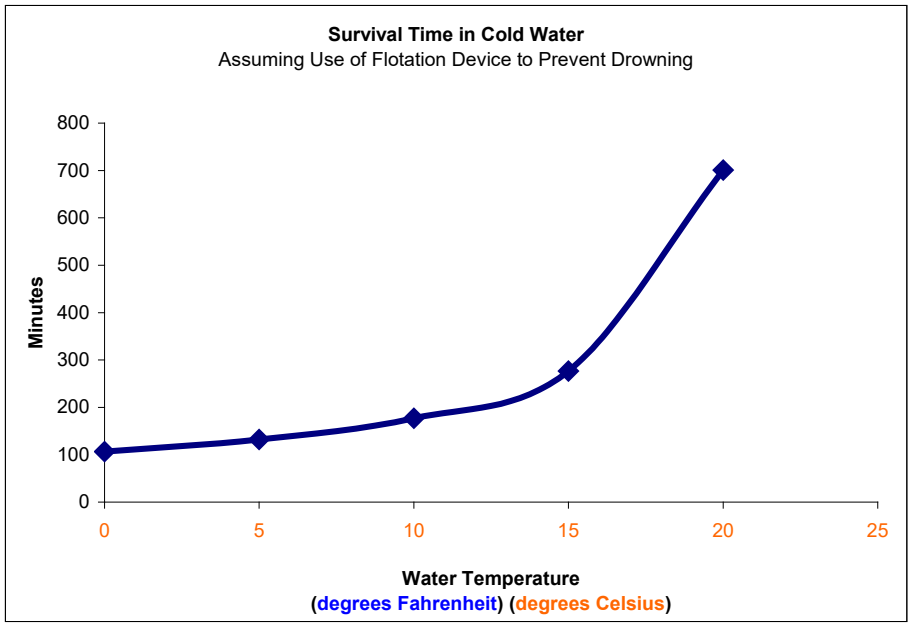
Personnel immersed in cold water cannot reliably assess how cold they are [PMID 2803163].[286] With immersion in mildly cool water, stability of the temperature of cutaneous receptors may lead to hypothermia without personnel being aware of their condition [PMID 3795105].[287]

C4.2.4.6 Cardiac Output in Cold Water

Head-out water immersion at thermo-neutral temperature increases cardiac output by increasing stroke volume; this is greater in cool (86°F or 30° C) water. Also, total peripheral resistance decreases (32% in one study) in cool water. Thus, cardiac output at a given work load is significantly higher in water than in air [PMID 10675972].[288]

C4.2.4.7 Survival Times in Cold Water

Figure 4-2 Survival Time in Cold Water



Survival times in cold water have been studied. Cold-water immersion of either men or women (at 32° F or 0° C) initially increases the ventilation rate (more than 4 times baseline for the first 2 minutes of immersion). After 10 minutes of immersion, mean skin temperature falls to 41° F (5° C), and rectal cooling rate is 10.8° F (6° C) per hour. After 20 minutes of immersion, maximum shivering metabolism peaks at nearly 4 times pre-immersion [PMID 6721816].[289] Swimming increases heat production to 2.5 times that of holding still (simply floating) in cold water (at 50.9° F or 10.5° C water temperature), but also increases the cooling rate 35%. A prediction equation for survival time of persons accidentally immersed in cold water (32° F to 71.6° F, or 0° C to 22° C) is as follows [PMID 1139445].[290]

Survival Time = 15 + 7.2/(0.0785 – (0.0034 x water temperature in °C)).

Figure 4-1 Survival Time in Cold Water (estimated)

Survival time may be shorter in very cold water (e.g., one hour at 32° F or 0° C) [PMID 6721816],[291] and in rough seas [PMID 3606516].[292] Actual survival without flotation devices may be much less (e.g., 2 to 5 minutes in seawater at 29° F or –1.67° C) [Navy].[293]

C4.2.4.8 Diving Suits

A study of cold-water exposure in diving suits showed that:

- “Dry suits” provide better protection than "wet suits".
- In rough seas, tight-fitting wet suits provide better protection than loose-fitting wet suits.
- Cold-water immersion in rough seas causes greater body core temperature to decrease than in calm seas.

- Accidental immersion in rough seas may be associated with significantly lower survival times than would be estimated from calm-water studies [PMID 3606516].[294]

C4.2.5 Contact and Handling of Cold Objects

Contact with cold objects (metal, ice, cold water, etc.) generally presents cold stress challenges only locally, unless there is a large area of skin contacting a very large object. Sensation on contact with very cold objects (-20° F or –28.9° C) includes tingling, pain, and burning (sometimes with almost no sensation of cold) [Daniels].[295] Also, once skin temperature falls below 46.4° F (8° C), tactile sensations do not function and warn (e.g., of freezing conditions and impending frostbite); hence, some investigators have chosen to use that temperature as the limit for frostbite risk [PMID 8049001].[296]

In addition to discomfort and decreased sensation on cold exposure of the skin, hand performance is substantially reduced at skin temperatures below 59° F (15° C) [PMID 8049001].[297]

C4.2.5.1 Dexterity when Handling Cold Objects

Exposure to the cold may negatively influence manual dexterity [PMID 12074025].[298] Hand skin temperature, rather than body surface temperature, is the critical factor; when hand skin temperature is 55° F (12.8° C) or lower, manual performance is impaired [PMID 5905109].[299] Manual performance (finger dexterity in knot tying) is further decreased as skin temperature falls further (e.g., to 45° F or 7.2° C), and at slower rates of cooling (generally associated with the fingers being exposed for longer times to less-severe cold) [PMID 13810475].[300]

Military researchers have noted that insulating metal equipment (e.g., with foamed plastic) in the cold is more feasible than trying to maintain manual dexterity by insulating the hand and have suggested “it is probably impossible to design a glove or mitten with adequate insulation which will permit all fine manipulations to be performed as quickly and as accurately as they can be done with the bare hand” [Daniels].[301]

C4.2.5.2 Freezing to Cold Objects

Touching very cold objects (such as touching cold metal with the tongue or fingers) is known to cause the body part to stick to the object. Freezing to cold metal has been studied and found to occur only when an “ice bridge” is formed from free water on the surface of the body part or object [Daniels].[302]

C4.2.6 Ultraviolet Light

Exposure to cold may also be accompanied by excessive ultraviolet light exposure (including ultraviolet light reflected off of snow or ice—for example, in the arctic) [PMID 2021394].[303] Thus, when protecting against cold exposure, it may be appropriate to also consider eye and skin protection often associated with warm weather (sunglasses and sunscreen lotion).

C4.3 Individual Cold Stress Factors

C4.3.0 Sunburn

Sunburn and snow blindness caused by UV radiation is not dependent on the air temperature but the sunlight itself. Light colored objects (snow, ice) reflect the sun's rays increasing chance of injury to eyes and exposed skin. Skin that is sunburnt increases heat loss during cold exposure thereby increasing the risk of hypothermia. This can be prevented by using alcohol free sunscreen that blocks UBA and UVB (alcohol: alcohol evaporates quickly leaving skin cold)

C4.3.1 Body Habitus

Individuals with higher body mass and more body fat can better tolerate cold stress exposure [PMID 984737].[304]

C4.3.2 Gender

Males tend to use carbohydrates and fats equally in their metabolic response to cold stress; females may use much more fats than carbohydrates [PMID 10198139],[305] but not all studies support that finding [PMID 3780704].[306] At least one review has concluded that individual body size, physical fitness, and state of acclimatization play far more important roles than gender in determining human thermal responses [PMID 750842].[307]

C4.3.3 Age

In a cold environment, children have lower skin temperatures, reflecting greater vasoconstriction [PMID 9587181].[308] Elderly (65 to 89 years of age) subjects exhibited less heat production, attenuated skin vasoconstrictor response, and lower core temperature after exposure to mild cold stress [PMID 17197640].[309]

C4.3.4 Race and/or Ancestral Geographic Location

As of 2022 there are no definable differences between race with regard to cold stress. A study showed that if any role in the prevention of non-freezing cold injuries with race is highly anecdotal in nature. There are no studies that clearly establish a difference in cold response with regards to race. [Haman et al., 2022][310]

C4.3.5 Medications

Beta - blockers – These medications alter the body's response to heart rate and affect the circulation of blood. This decreases transfer of heat from core to peripheral tissue. However, due to this decrease in circulation, increases the risk for peripheral cold stress (frostbite) [Blessberger et al., 2019][311].

Sedatives – Clonidine is used as a post operative medication to stop anesthesia induced shivering by disrupting the hypothalamic thermoregulation system. This mechanism causes a central thermoregulation impairment decreasing the threshold of shivering and the vasoconstrictive response [Delaunay et al., 1993][312].

Opioids - Have an effect on hypothalamic thermoregulation. They depress the sympathetic response that results in a diminished threshold when exposed to cold. The body does not activate vasoconstriction or begin shivering to produce or generate heat [Díaz & Becker, 2010][313].

Antipsychotics – These medications can cause hypothermia in the presence of predisposing factors to include exposure to cold, subclinical, or untreated hypothyroidism, and use concomitant use of benzodiazepines. It is postulated that data obtained from drug monitoring agencies suggest the number of hypothermia related cases can be at least 10 times higher than is documented in the literature [Zonnenberg et al., 2017][314].

4.3.6 Alcohol

Alcohol (ethanol) can cause cutaneous capillary dilation, which in turn may inappropriately increase cutaneous blood flow during cold exposure. Skin temperature fall will be blunted, while core temperature will fall more quickly. Thus, alcohol may diminish thermoregulatory responses associated with acclimatization: when rectal and skin temperatures decrease simultaneously, thermoregulation is greater than when rectal temperature alone changes [PMID 8900834].[315] A recent case report noted that severe ethanol poisoning, in the absence of any other contributing factors, may explain hypothermia [PMID 17251602].[316] However, studies have concluded that in cold exposure, moderate alcohol consumption predisposes individuals to hypothermia more by behavioral factors than via impaired thermoregulation [PMID 497899][317] [PMID 8897037].[318]

C4.3.7 Adaptations

For practical purposes it has been found that repeated cold-water immersion can cause a mild or slight increase in metabolic heat production. However, cold adaptation that results in increased metabolism and insulation cannot developed over short periods of time. Thus, we are dependent on behavioral skills to survive in cold climates. [Daanen & Van Marken Lichtenbelt, 2016][319]

C4.3.8 Hydration

Cold exposure may lead to significant dehydration (via cold diuresis, high energy expenditures, and poor access to water) [PMID 7639888].[320] Urine specific gravity is often used to monitor hydration status in field settings. However, one study of cold exposure found no significant correlation between changes in total body water and urine specific gravity or other typical urinary indicators of dehydration [PMID 7639888].[321] In addition, a study of Marines in a cold environment found that dehydration might not be readily noticeable in the field, due to maintenance of circulating volume at the expense of both intracellular and extracellular water

[PMID 3116457].[322] The same study found that inadequate drinking water availability was associated with inadequate nutritional intake (troops preferred to go hungry) [PMID 3116457].[323]

C4.4 Compensation for Cold Environments

C4.4.1 Functioning in Cold Environments

Moderate cold exposure (that does not produce core hypothermia) can impair performance of complex cognitive tasks [PMID 2818396].[324] Mechanisms to compensate with cold environmental temperatures include behavior changes (voluntary muscular activity and exercise, staying indoors, wearing warm clothing, hair growth to include facial hair) and physiological responses.

- Physiological responses to cold stress include increasing body fat, increased appetite, increased metabolism, increased activity, decreased cutaneous blood flow, shunting of blood from skin and extremities, and shivering. Additionally, an increase of heat by thermogenesis during the metabolism of brown fat [PMID: 25390014].[325]

C4.4.1.1Protective Clothing and PPE in Cold Weather

Wearing of chemical protective clothing in the cold was found to significantly impede the performance of basic medical tasks, possibly primarily due to bulkiness [PMID 18568960].[326]

C4.4.2 Metabolism

C4.4.2.1 Muscles

Lactate levels with work in cold exposure are generally higher than with work in milder conditions; the time lag between production of lactate within the muscle and its release into the venous circulation may be increased by cold exposure [PMID 1925184].[327]

C4.4.2.2 Body Fat

Significant inter-individual variation exists among persons exposed to cold stress in both body temperatures and energy expenditures (adjusted for body composition) [PMID 11934673].[328] Individuals with less body fat tend to expend more calories when exposed to cool water due to reduced insulative properties of body fat[PMID 11990094].[329]

Increased oxidation of carbohydrates and free fatty acids is a well-known phenomenon during cold stress [PMID 12079880].[330] Human studies have shown that cold exposure increases lipid oxidation [PMID 2233284].[331] Fat oxidation increases during shivering in prolonged (105 to 388 minutes) immersion in cold water; plasma glucose increases, and is lower during intense shivering than during moderate shivering [PMID 12012076].[332]

C4.4.3 Calories and Cold Exposure

C4.4.3.1 Caloric requirements

The calorie requirements of adequately clothed men living and working in a cold environment are **not increased**, except for the 2-5 percent increase in metabolic rate due to effort required by heavy clothing [PMID 844611].[333] Although a cold-induced increase in appetite may be expected, evidence for such a phenomenon is poor [PMID 10817145].[334] Water availability may affect food intake, as noted previously.

C4.4.4 Appetite

Decrease in appetite and food intake has been noted in cold exposure at high altitudes [PMID 1582718];[335] Prolonged high altitude exposure is often accompanied by considerable weight loss, thought to be due to primary anorexia, lack of comfort and palatable food, detraining, and possible direct effects of hypoxia on protein metabolism [PMID 1483750].[336] Hypoxia at altitudes >6000ft causes decreased hunger for sweet and salty foods. Acute hypoxia (acute mountain sickness) causes a rapidly suppressed appetite [Barclay et al., 2023].[337]

C4.4.5 Cardiovascular

C4.4.5.1 Shunting.

Shunting is a mechanism the body uses to direct blood flow to and from core and peripheral blood supplies. When exposed to a cold environment, cutaneous blood vessels (peripheral) constrict, redirecting blood flow to the core of the body. The vasoconstriction of the sympathetic nervous system is most pronounced in the extremities, but is minimal in the head and neck [PMID 1811574][338] [PMID 2221434].[339] This mechanism helps to maintain core body temperature at the expense of the periphery. This is the opposite mechanism that is used for Heat Stress (see above).

C4.4.5.2 Blood Pressure

Cold exposure increases blood pressure [PMID 11374119].[340]This is due to the reaction of the sympathetic nervous system (See above) causing vasoconstriction of the peripheral vasculature. Exposure to cold air increased average systolic and diastolic pressures approximately 20 millimeters of mercury each [PMID 11214769].[341] Blood pressure is noted to be higher in winter than in summer [PMID 9314429].[342]

C4.4.6 Heart Rate and Cardiac Output

When initially exposed to extreme cold weather or water, the heart rate will decrease due to combined activation of the autonomic reflex and baroreceptors [Wu et al., 2021][343]. It is recommended to allow 3-5 minutes to pass before any high intensity exercise to allow for the

physiological changes to stabilize. Cold increases the risk of arrhythmia during exercise [PMID 11505864].[344] This is important when going from a warm (building, barracks, birthing) to cold environment. Routine training can help alleviate the degree of change in heart rate change and to reduce the adaptation time [Wu et al., 2021][345].

C4.4.7 Oxygen Consumption

Exercise oxygen consumption is generally higher in the cold, but the difference between warm and cold environments becomes less as workload increases [PMID 1925184].

C4.4.8 Respiratory Tract and Ventilation Changes

Acute or chronic cold exposure can cause bronchoconstriction, airway congestion, secretions, and decreased mucociliary clearance (actively in cold-induced or exercise-induced asthma), resulting in decreased baseline ventilation and respiratory chemosensitivity. Cold exposure increases pulmonary vascular resistance. Chronic cold exposure results in increased numbers of goblet cells and mucous glands, hypertrophy of airway muscle tissue, and increased muscle layers of terminal arteries and arterioles. [PMID 7487830].[346]

Minute ventilation is substantially increased upon initial exposure to cold [PMID 1925184].[347] Acute cold stress exposure early in acclimatization causes a decrease in ventilation parameters. As acclimatization continues, there is a gradual recovery continuing up to 9 weeks [PMID 8468097].[348]

C4.4.9 Shivering

Shivering is a mechanism of thermogenesis that is triggered by skin cooling that results in increase in metabolism, ventilation, cardiac output and mean arterial pressure [Dow et al., 2019][349]Shivering is the body's reserve mechanism for dealing with extreme heat loss, as sweating is the body's mechanism for removing heat in excessive heat stress conditions.

C4.4.9.1 Metabolism of Shivering

Shivering can increase the metabolic rate to a maximum of approximately five times the resting rate [PMID 11394237].[350] Shivering can generate heat at a rate of 10 to 15 kilojoules per minute, but it impairs skilled performance, while the resultant glycogen usage hastens the onset of fatigue and mental confusion [PMID 3883460].[351]

C4.4.9.2 Peak Shivering Metabolic Rate Equation

An equation to calculate the peak shivering metabolic rate has been formulated as follows [PMID 11394237].[352]

Peak shivering metabolic rate (in milliliters of oxygen per kilogram per minute) =

30.5 + 0.348 x maximal oxygen uptake (in milliliters of oxygen per kilogram per minute) –

0.909 x body mass index (in kilograms per square meter) –
0.233age (in years).

C4.4.10 Fatigue

Vasoconstrictor responses to cold, but not shivering responses, are impaired after multiple days of severe physical exertion [PMID 11181604].[353] Fatigue induced by chronic overexertion sustained over many weeks delays the onset of shivering until body temperature is lower than in rested individuals [PMID 11282320].[354] These findings suggest that susceptibility to hypothermia is increased by exertional fatigue [PMID 11181604].[355]

C4.5 Acclimatization

C4.5.1 Acclimatization and Acclimation

Acclimatization refers to the adaptation to cold or heat stress that occurs after repeated exposure to cold or heat stress conditions. Acclimation refers to adaptation that occurs after laboratory-controlled exposure to cold or heat stress.

C4.5.2 Effects of Acclimatization

The following effects of cold acclimatization (or acclimation) have been observed:

- An increase in the delay for the onset of shivering (approximately twice as long before shivering starts) [PMID 3597234].[356]
- A slight (less than 0.5° F) decrease of core body temperature levels at the onset of shivering [PMID 3597234].[357]
- Lower core body temperature in thermoneutrality (less than half a degree F) [PMID 3597234].[358]
- A decrease of heat debt calculated from the difference between heat gains and heat losses (by about one third) [PMID 3597234].[359]
- Reduced sensitivity to the pressor effect of norepinephrine [PMID 8299617].[360]
- Reduced cold-induced muscle tenseness [PMID 8299616].[361]
- Less shivering on cold exposure [PMID 1483764].[362]
- Lower central and peripheral body temperatures at rest and during cold immersion [PMID 8765994].[363]
- Delayed metabolic response to cold [PMID 8765994].[364]
- Attenuated subjective shivering [PMID 8765994].[365]
- Lowered cold sensation [PMID 8765994].[366]
- Increased vasoconstriction, evidenced by lowered skin temperature [PMID 8765994].[367]
- Increased rate of rewarming [PMID 655993].[368]
- Lowered diastolic pressure and an increase in peripheral vasoconstriction [PMID 8891513].[369]

- Altered control of blood pressure during acute cold stress (in one study of repeated cold-water exposures, blood pressure increased significantly during the first cold-water exposure, but not during the last cold-water immersion) [PMID 3388627].[370]
- Lower oxygen consumption in cold air (but not in cold water) [PMID 3388627].[371]
- Cold acclimation attenuates the onset of metabolic heat production during cold air exposure [PMID 3388627].[372]

C4.5.3 Limitations of Acclimatization

Overall, the effects of cold acclimatization (on tolerating cold exposure) are minimal compared to the effects and advantages of heat acclimatization (on tolerating heat exposure).

- Maximal aerobic and anaerobic performances are not altered by acclimatization [PMID 8765994].[373]
- Cold acclimation does not affect the minute ventilation - carbon dioxide production relationship or the pattern of breathing in cold air or water [PMID 3388627].[374]
- Cold acclimation does not alter the magnitude of metabolic heat production [PMID 3388627].[375]
- Cold acclimation has no effect on cardiac output or arterial-venous oxygen difference [PMID 3388627].[376]
- Cold acclimation, when developed by cold-water immersion, does not influence vascular fluid responses to cold stress (increased urinary excretion rate of both sodium and potassium during cold exposure) [PMID 3629738].[377]
- Acclimatization does not induce non-shivering thermogenesis in adults [PMID 8299617].[378]

C4.5.4 Development of Acclimatization

C4.5.4.1 Altitude

Thermoregulation efficiency of man deteriorates at high altitude; general cold exposure acclimatization at high altitude may take much longer in those not native to high altitudes, or may even be unattainable [PMID 655993].[379] Temperature decreases 2C for every 1000 feet gain in altitude. The percent oxygen available also decreases. Altitude may accentuate certain responses to cold stress. The respiratory responses during acute cold exposure are similar to those of initial altitude responses [PMID 8468097].[380] Acclimatization to cold exposure at higher altitudes may appear to occur faster than it really does, as heart rate and blood pressure decreases may occur from a transient reduction in parasympathetic and sympathetic activity during initial stepwise exposure to high altitude [PMID 11903133].[381]

C4.5.4.2. Local (Rather than Whole Body) Acclimatization

Exposure to severe local cold leads to adaptive responses in which discomfort and autonomic activity are reduced [PMID 3057321].[382] Local cold acclimation induces a local cold adaptation (decreased reduction in skin temperature during cold exposure) by significantly

decreased plasma concentrations of norepinephrine [PMID 8781852].[383] Local cold acclimatization is possible regardless of what climate a person (or a person's ancestors) is from [PMID 1297856].[384]

C4.5.5. Habituation

Exposure to systemic moderate cold causes a reduction in heat production, in shivering, and in discomfort through a process known as habituation [PMID 3057321].[385] Habituation is "the method by which the nervous system reduces or inhibits responsiveness during repeated stimulation" [Stedman's].[386] Habituation to cold stress results from repeated brief exposures to cold. Rather than a series of physiologic changes, it is an enhanced tolerance to the cold due to decreased sympathetic activity, which is thought to be a diminished alarm reaction [PMID 1483764].[387]

Chapter 5: Prevention of Cold Stress Injuries

C5.1. Measurement of Cold Stress Effects on the Body

C5.1.1. Core Body Temperature

The most reliable method generally available in hospital settings for determining core body temperature is the esophageal thermometer (see section on **Error! Reference source not found.**). In field settings, rectal temperatures provide a reasonable approximation of core body temperature. Tympanic and oral temperature readings have been subject to variation due to exposure to air and/or water.

C5.1.2. Skin or Local Temperature

The need for accuracy is less in determining the temperature of the extremities exposed to cold stress. (In other words, knowing whether a toe is 25° F or 15° F, (-3.9° C to -9.4° C), is not as critical to patient management as knowing whether core body temperature is 95.4° F or 89.4° F, (35.2° C to 31.9° C, although the absolute temperature difference of the former is much greater.) Even a gross approximation, such as whether an extremity "feels frozen," may be adequate to guide appropriate treatment. Accurate documentation of skin temperature, however, requires the use of a thermometer.

C5.2. Identifying Risk Factors

C5.2.1. Ointments, Lotions, Creams, Emollients, etc.

Application of ointments to the face has been shown not to offer protection against frostbite of the head in cold climates [PMID 10086864].[388] Skin ointments may give troops a

false sense of safety against the cold, but provide no such protection and may actually be a risk factor for frostbite [PMID 10998829][389][PMID 10954213].[390]

C5.2.2 Motion Sickness

Motion sickness attenuates the vasoconstrictor response to skin and core cooling, thereby enhancing heat loss and the magnitude of the fall in deep body temperature; motion sickness may predispose individuals to hypothermia, and have significant implications for survival time in maritime accidents [PMID 11533150].[391]

C5.3. Protective Clothing

C5.3.1. Clothing

Due to wearing heavy clothing, in extremely cold conditions there may be sweating and discomfort of the torso from warmth during heavy work. Conversely, in spite of heavy clothing, cold-induced numbness and pain of the face, hands, and feet is common [PMID 3769908].[392] A review, including simulation modeling, of the military Extreme Cold Weather Clothing System during cold stress exposure reported that:

- NBC protective clothing may inadequately protect against hand and finger cooling, especially during rest following strenuous activity.
- There is no evidence substantiating suggestions that wearing NBC protective masks increases susceptibility to facial frostbite.
- Any increased risk of hypothermia associated with wearing NBC protective clothing while working in the cold is unlikely.
- Wearing NBC protective clothing during strenuous activity in cold weather may increase the risk of hyperthermia, and cause sweat accumulation in clothing which may compromise insulation and increase the risk of hypothermia during subsequent periods of inactivity [PMID 10685594].[393]

C5.3.2 Insulation and Moisture

Choosing and using clothing to keep personnel warm in cold weather should focus on two things: providing insulation and minimizing moisture in the clothing touching the body. This is best accomplished by using multiple layers of clothing and regularly changing or “rotating” clothing so that the innermost layer is dry and not soaked with sweat. Colder weather (taking wind chill into account) requires greater insulation; greater levels of exertion decrease insulation requirements and increase sweating and the need to frequently change or rotate inner garments. Personnel beginning to sweat should be quick to remove unnecessary clothing in order to prevent saturating clothes with sweat (which can subsequently pose a cooling hazard when exertion stops).

C5.3.3 Clothing Fabric Characteristics

The best insulation is provided by down, but only if the garment is kept dry. Wool, fleece, and synthetics designed to insulate also can provide excellent insulation. However, those materials can quickly become wet with sweat when worn touching the body. The best undergarments for cold weather have a "moisture-wicking" weave. Underclothing has traditionally been made of cotton, but cotton clothing should be avoided in cold environments because it readily absorbs moisture and dries slowly. Cotton has contributed to hypothermia when cotton clothing becomes saturated with sweat or water in cold environments.

The next layer (going from touching the body to outer garment) should be one that readily absorbs moisture. (Moisture wicking properties will be of no benefit if the next layer completely blocks moisture.) The outer garment should be chosen by its ability to resist penetration by wind and rain and by the ambient temperature. Wind can be expected to reduce the insulating capacity of windproof and wind-resistant garments about 20%; wind-permeable garments may lose 70% of their insulating potential [PMID: 20066643].[394]

In still or low wind conditions, thermal insulation is mostly a function of garment thickness, although reflective metal foil material provides greater thermal insulation than would be expected based on thickness [PMID: 20066643].[395] The ability to trap warm air next to the body, not garment thickness, is the key quality. For example, lightweight, non-bulky down jackets may provide an extreme degree of warmth, not because of their thickness, but because of their ability to trap warm air and keep it next to the body.

C5.3.4 Head Coverings

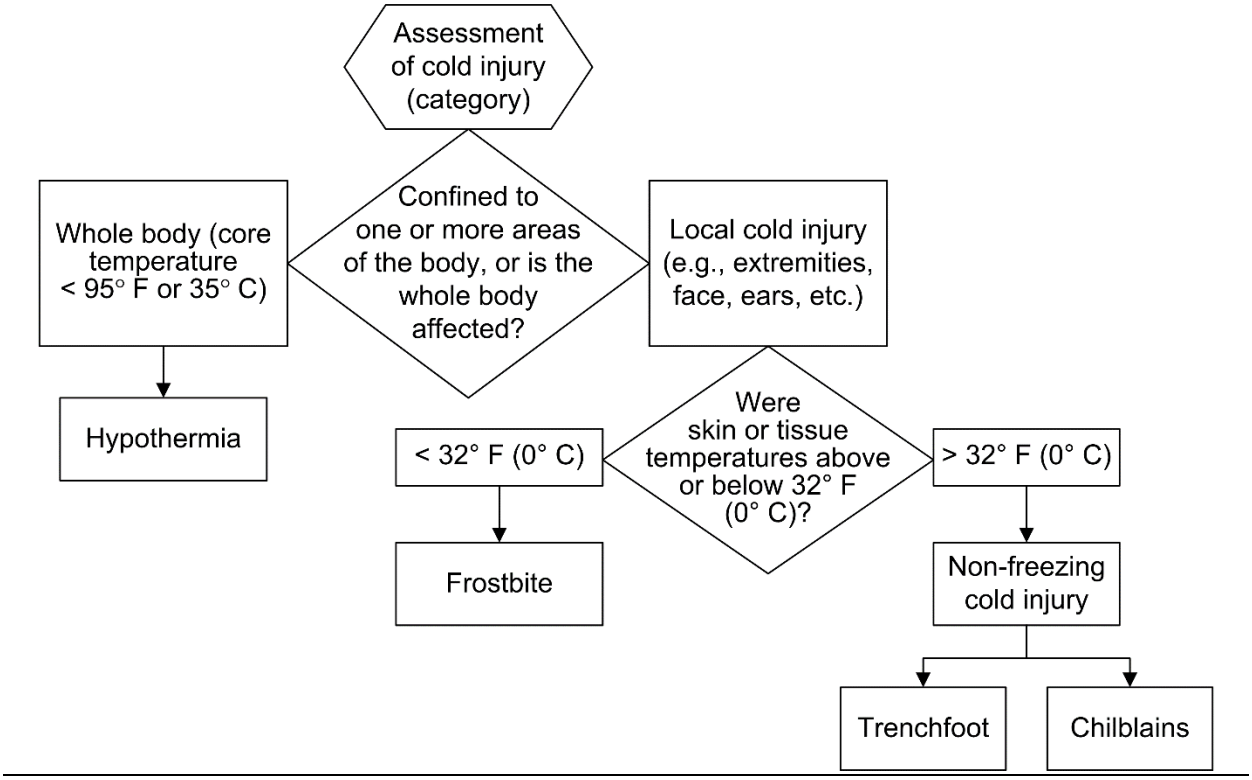
A large amount of heat can be lost from a person's head, so head covering is vital to protecting personnel during cold weather. Head gear should be chosen considering the amount of insulation needed, rain resistance, sun protection (a wide brim provides more protection against sunburn than a "beanie"-type hat, and a tighter weave provides more protection than a looser weave fabric), and ear protection. When temperatures approach freezing, facial protection should be considered. Anti-fogging goggles should be considered in temperatures below freezing, especially if there is significant wind.

C5.3.5 Hand and Foot Coverings

Hands and feet are other areas that need special protection. When temperatures go below 40° F, gloves should be worn. Mittens are preferable when it is extremely cold, but they may severely degrade performance. Footwear should be appropriate for the terrain, activity, and potential exposure to moisture or immersion. At temperatures approaching freezing, layers of socks and frequently changing socks to keep the feet dry is recommended.

Chapter 6: Diagnosis and Treatment of Cold Stress Injuries

Figure 6-1 - Assessment (Category) of Cold Injury



C6.1. Injuries

C6.1.1 Acrocyanosis

Acrocyanosis is a painless symmetrical discoloration of distal fingers (blue), and less commonly the feet, after exposure to cold. Acrocyanosis is a diagnosis of exclusion and must be differentiated from secondary causes such as Raynaud's and Chilblains.

Symptoms: local Hypothermia, increased sweatiness, and infiltration of elastin in the skin. Acrocyanosis does not have proximal pallor or distal redness with rewarming (see Raynaud's). It also isn't associated with pain (see Chilblains). Those suspected of any of these conditions should be directed to primary care for further work up. [Kent & Carr, 2021][396]

Treatment: Consists of conservative management to include:

- Smoking cessation

- Avoid cold weather.
- Insulated clothing

C6.1.2 Rosacea

Rosacea (also known as acne erythematosa and acne rosacea) is a condition of the nose and cheeks involving dilation of blood vessels and follicles. Rosacea may be exacerbated by cold weather [PMID 12182520].[397]

Treatment: Depending on severity of condition can include laser treatments and surgery. However, these are for the more severe cases of Rosacea. Deterrence of exacerbating factors should be identified and avoided if possible. These factors include:

- Hot or cold temperatures
- Wind
- Hot drinks
- Caffeine
- Exercise
- Spicy food
- Alcohol
- Emotions
- Topical products that irritate the skin and decrease the barrier.
- Medications that cause flushing [Rosacea, 2021][398]

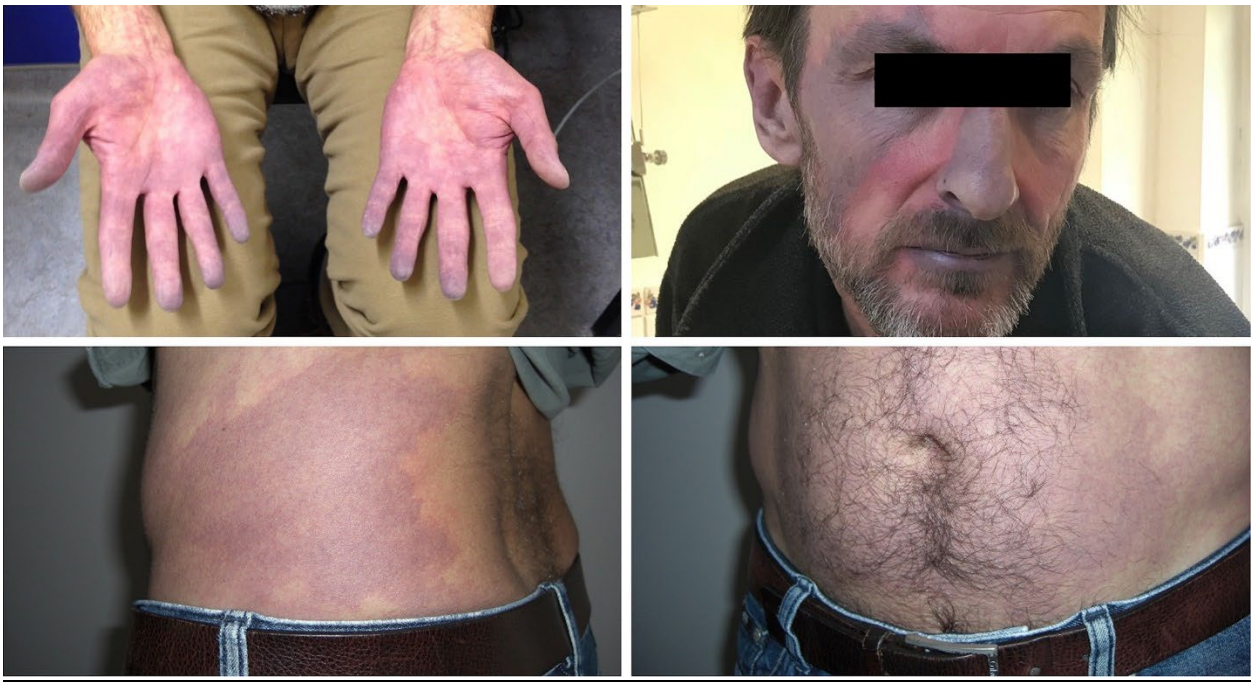
C6.1.3. Cold Agglutinin Disease

Cold agglutinin disease is a form of auto-immune hemolytic anemia. Cold-reacting antibodies attack red blood cells causing anemia. The dysfunction of auto-reactive B-cell clones may be triggered by infection of some viruses and bacteria, or by certain medications [PMID 8890588].[399] Cold agglutination may also be associated with underlying disease (lymphoma, post-mycoplasma or infectious mononucleosis infections) [Schrier].[400]

Symptoms: Presents with painful discoloration of fingers and toes with discoloration after exposure to cold. Looks similar to Acrocyanosis, and Raynaud's. Due to the loss of RBC's can often have symptoms of pallor, dyspnea, fatigue. [Berentsen, 2016][401]

Treatment: Supportive with immediate removal from cold and transported to closest hospital. Full work up should be completed with definitive diagnosis obtained. [Cold Agglutinin Disease, 2024][402]

Figure 6-2 Cold Agglutinin Disease

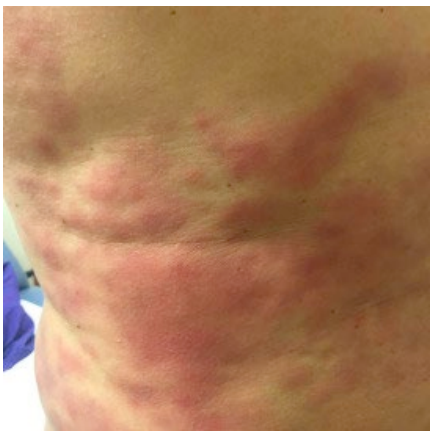


(Tomkins et al., 2020)[403]

C6.1.4. Cold Panniculitis

Cold panniculitis is a skin condition with red, cold, indurated plaques or nodules which appear one to three days after exposure to low temperatures and resolve spontaneously within several weeks without scarring [PMID 9830269].[404] Cold panniculitis can be caused by exposure of the skin to cold, including cold packs. It is the consequence of lipid crystallization within adipocytes [PMID 10667045].[405]

Figure 6-3 Cold Panniculitis

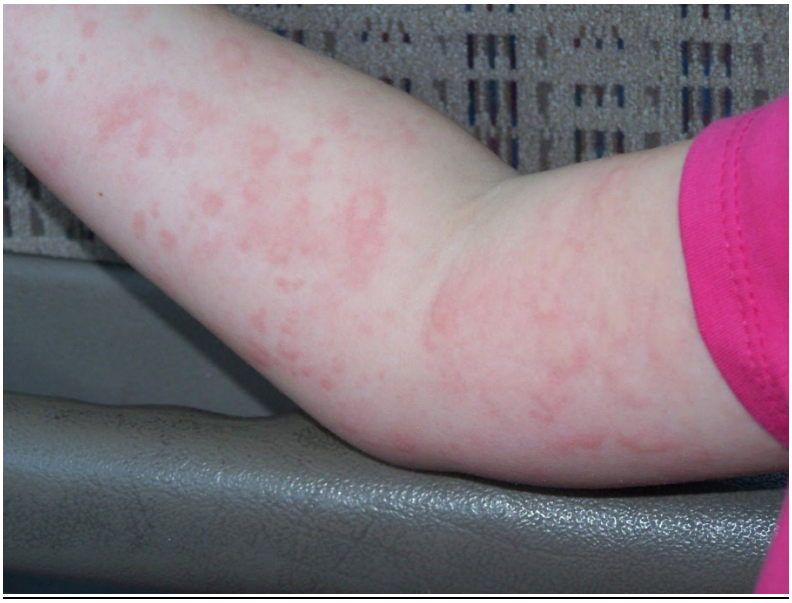


(Greenwald et al., 2018)[406]

C6.1.5. Cold-induced Urticaria

Cold-induced urticaria (hives) is a form of physical urticaria that develops on cold exposure in susceptible individuals. Most cases are of unknown etiology (primary or idiopathic), and management includes avoidance measures and antihistamines. Life-threatening symptoms necessitate carrying a self-administered injectable epinephrine [PMID 11409259].[407]

Figure 6-4 Cold Induced Urticaria



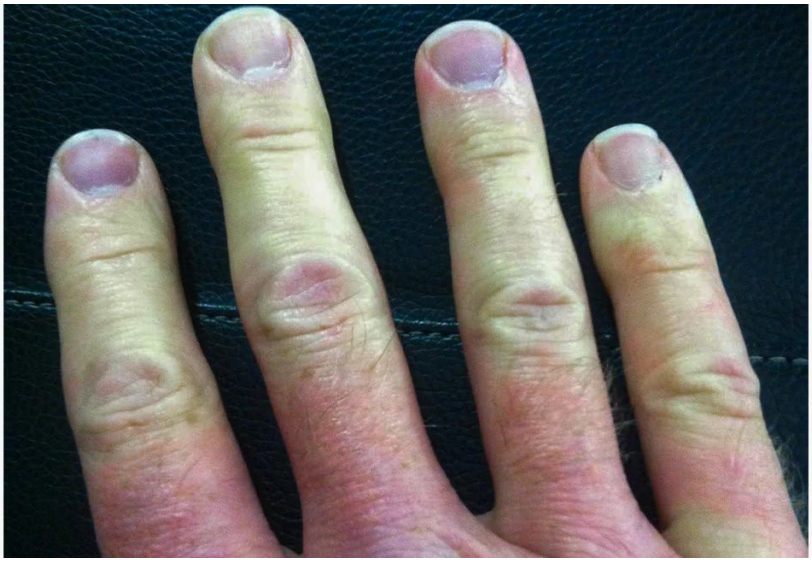
C6.1.6. Vibration White Finger

Occupational cold exposure may be a contributing factor in the development of vibration white finger [PMID 8022312].[408] Workers should have adequate hand protection when handling cold objects.

C6.1.7. Raynaud's Phenomenon

Raynaud's Phenomenon is "sensitivity of the hands and fingers to cold, as a result of spasm of the digital arteries, with blanching and numbness or pain of the fingers" [Stedman's].[409] It manifests as "episodic vasospastic ischemia of the digits…characterized by digital blanching, cyanosis, and rubor after cold exposure and rewarming" [SAM-CD].[410] Workers with Raynaud's Phenomenon must be especially careful to use adequate gloving; under certain cold exposure conditions, some such individuals may not be able to work safely.

Figure 6-5 Raynaud's Phenomenon



CC BY-SA 4.0, via Wikimedia Commons 2012[411]

C6.1.8. Chilblain

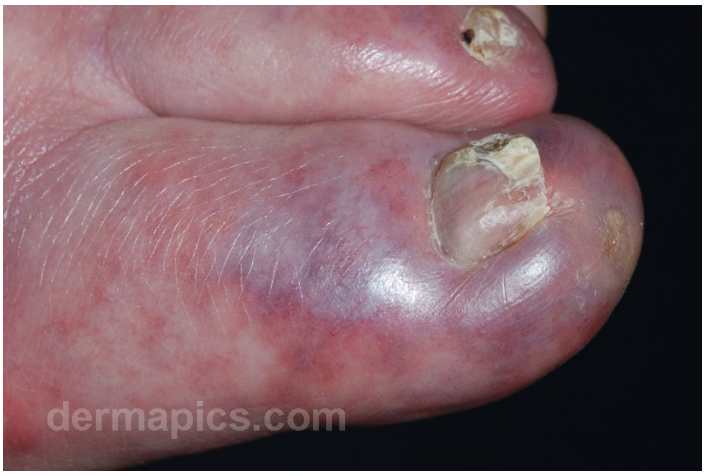
Chilblain (chilblains, Milker's chilblains, erythema pernio, perniosis) is a vascular erythrocyanotic discoloration of the distal skin on fingers and toes that occurs after exposure to the cold. Risk factors include female sex, low body mass index (BMI), and during months November – April with humid non-freezing temperatures [Prakash & Weisman, 2009][412].

Symptoms: include pruritic, painful (especially burning) red patches on the fingers and/or toes, generally bilaterally. Areas of nose, ears, lateral thighs, and buttocks have also been reported. Symptoms are painful and do not show red/white/blue discoloration (compare with acrocyanosis, and Raynaud's)

The differential diagnosis includes lupus, cold urticaria, acrocyanosis, erythromelalgia (erythermalgia, Gerhardt's disease, Mitchell's disease, red neuralgia, rodonalgia), vasculitis and the Blue Toe Syndrome (tissue ischemia secondary to cholesterol crystal or atherothrombotic embolization leading to the occlusion of small vessels [PMID 12555011])[413] [PMID 1431611].[414]

Treatment: Removal from the cold (including passive warming). Refer to Primary Care for work up and further treatment.

Figure 6-6 Chilblains



("Chilblains," n.d.)[415]

C6.2. Injuries of the Extremities Due to Cold Exposure

C6.2.1. Frostbite

Frostbite is a localized cold injury, generally of exposed or inadequately protected acral areas, resulting in tissue destruction due to freezing or sub-freezing temperatures. Victims complain of numbness, coldness, pain, or loss of use of the affected area. This can lead to gangrenous necrosis and auto amputation if not removed from the exposure [Gupta et al., 2021][416]. Cold exposure from very cold liquids (e.g., liquid nitrogen, liquid oxygen, liquid chlorine, liquid ammonia, liquid monochlorodifluoromethane [Freon®]) as well as dry ice may cause frostbite if not handled properly. [PMID 19165095,[417] PMID 11193374,[418] PMID 10751710,[419] PMID 9610968,[420] PMID 1891741].[421]

C6.2.1.1. Mechanisms (Pathophysiology) of Frostbite

Frostbite is a multistep injury that begins at the cellular level. Ice crystals begin forming in the extracellular space that leads to damage of the cell membrane. Intracellular fluids will move into the extracellular space additionally disrupting the movement of Sodium and Potassium. This leads to a change in cellular osmolarity causing proteins to denature, and release of inflammatory cytokines. [Gupta et al., 2021][422] This results in cellular injury; ensuing and concomitant ischemic anoxia and acidosis contribute to the injury.

As rewarming (thawing) of frozen tissue allows re-perfusion, vascular permeability and intravascular thrombi formation result in further anoxic damage. When rewarming is done gradually, marginal tissue ice crystal formation may recur, resulting in further damage. Similarly, freeze-thaw-refreeze (for example, when a frostbite victim is warmed, but then is exposed to

further freezing) injuries may be worse than cases left frozen until thawing can be done without re-exposure to freezing cold.

C6.2.1.2. Frostbite Risk Factors

Low air temperatures and high wind speeds are associated with an increased risk of freezing of the exposed skin; as the skin surface temperature falls from 23.4° F (-4.8° C) to 18° F (-7.8° C), the risk of frostbite increases from 5% to 95% [PMID 9018520].[423]

Factors contributing to risk of frostbite:

- African decent – due to increased vasoconstriction in periphery greater than other races.
- Inadequate clothing (hats, gloves, boots, socks, scarfs, face covering, base layer, jacket)
- Wet clothing
- Dehydration
- Inactivity
- Fatigue
- Previous cold weather injury
- Medications/Drugs (Beta-blockers, anti-psychotics, sedatives, opiates, cocaine)
- Alcohol use
- Smoking
- Diabetes
- Hand arm vibration

[PMID 9433082].[424] [PMID 11149059].[425] [Endorf & Nygaard, 2022][426][PMID 8318122].[427]

C6.2.2. Classification of Frostbite

C6.2.2.1 Frostnip

This is the mildest freezing injury of the skin and involves freezing water on the skin surface. The skin is reddened and may be swollen. Recovery is complete (similar to a mild sunburn) with removal from cold exposure.

C6.2.2.2 First Degree Frostbite

First degree frostbite is a partial thickness injury of the skin, sparing deeper structures. It is characterized by erythema, edema, and hyperemia. There is no blister formation or necrosis. Victims complain of pain (for example, a burning sensation).

Prior to thawing, in frostbite that is severe first degree or worse, skin is gray or whitish (often described as “waxy” or “waxy-white”).

Swelling occurs within 3 hours of rewarming and may last 10 days. Desquamation starts in about a week and may last up to one month. Resolution is expected to be complete and without scarring.

C6.2.2.3. Second Degree Frostbite

Second degree frostbite is characterized by blisters or blebs, which may not form until after rewarming, and by erythema and edema [PMID 10998830].[428] It is a full-thickness injury that spares subcutaneous tissue. Blisters (which usually form after rewarming) contain serous fluid. Victims may complain of numbness.

There is no permanent tissue loss. Sequelae include increased cold sensitivity, hyperhidrosis ("sweaty" feet or hands), paresthesia's, pain, and necrosis of pressure points on the feet.

The vast majority of frostbite injuries in the military are either first or second degree, according to a review of Army Experience in Alaska [PMID 9433082].[429]

C6.2.2.4. Third Degree Frostbite

Third degree frostbite includes injury to the skin and subcutaneous tissue. Hemorrhagic blisters may be present (usually after rewarming), with bluish skin and skin necrosis. Initially, involved areas are anesthetic, becoming painful on rewarming.

Skin loss by sloughing is expected, with permanent tissue loss and scarring.

C6.2.2.5. Fourth Degree Frostbite

Fourth degree frostbite involves skin, subcutaneous tissue, and deeper structures, including bone, tendon, or muscle. Affected areas are anesthetic, even after rewarming, although severe paresthesia's may develop days to weeks later [NAVEDTRA 13147-A].[430]

C6.2.3. Evaluation

Physical examination should document core body temperature, temperature of the involved area (if an exact temperature cannot be taken, then subjective descriptors such as "frozen," "cold," "cool," etc., may be used), presence of blisters and color of blister fluid, and "feel" of the affected area (e.g., waxy, hard, etc.). Peripheral pulse and capillary refilling should be documented and checked repeatedly as the injured area is rewarmed. Neurological exam (including two-point discrimination, vibration, and movement) also should be checked and followed.

The appearance of superficial tissue is often an unreliable indicator of deep-tissue viability in cases of frostbite [PMID 11822694].[431] Experienced clinicians state that 4 to 5 days may be required to ascertain whether lesions involve superficial or deep freezing; if there has been tissue necrosis, approximately one to two months may be required to define the limits of necrosis [PMID 1483773].[432] The severity of frostbite injuries can now be assessed with

triple phase bone scanning, allowing early prediction of likely subsequent tissue loss. [Imray et al., 2009][433]

Technetium (Tc)-99 bone scanning (bone scintigraphy) has become the standard imaging study employed within the first several days to assess tissue perfusion and viability [PMID 9088467].[434] Two-phase Tc-99m hydroxymethylene diphosphonate bone scans have been used in evaluation of frostbite and treatment follow-up. Correlation between absence of tracer uptake in the phalanges and later amputation was shown to have high sensitivity (0.99) and high specificity (0.96) in one study of severe frostbite [PMID 10853803],[435] but not in a study of mild to moderately severe frostbite using Tc-99m pertechnetate [PMID 11926378].[436] Successful use of Tc-99m-sestamibi scintigraphy in evaluation of frostbite has also been reported [PMID 11822694].[437] A review found that bone scintigraphy with single photon emission computed tomography (SPECT)/CT performed in the acute and subacute course of frostbite injuries led to earlier definitive management and shorter hospital stay [PMID: 30359097].[438]

MRI and magnetic resonance angiography (MRA) were felt superior to Tc-99 bone scans in a small study (two cases) of severe frostbite injury. The authors felt MRI and MRA offered the advantages of allowing direct visualization of occluded vessels, imaging of surrounding tissues, and of showing a more clear-cut line of demarcation of ischemic tissue [PMID 9088467].[439] What has been called the Helsinki frostbite management protocol includes angiography within 48 hours of severe frostbite [PMID: 28778759].[440]

C6.2.4 Treatment of Frostbite

C6.2.4.1 Rewarming

Initial treatment is immediate removal from cold exposure. If that cannot be done, further treatment (rewarming) should be delayed avoiding freeze-thaw-refreeze-thaw, which may result in worse injury than a single freeze-thaw. Rewarming of frostbitten lower extremities should not be done if the person must walk to get to medical treatment. Rewarming should never be done using an open flame [CHIPPM TN/02-2].[441]

Rapid rewarming in water (as opposed to gradual rewarming) is the definitive treatment for frostbite [PMID 10791170].[442] Water temperatures from 96.8° F (36° C) [PMID 1483773][443] to 108° F (42.2° C) [PMID 9460447][444] have been recommended. Some clinicians add a mild antiseptic to the water, and limit immersion to 20 to 30 minutes twice daily [PMID 1483773].[445] Rewarming may be quite painful and require analgesics and sedatives [PMID 9556318].[446]

Once thawing is complete, the injured part must be kept clean and dry and protected from further trauma. All patients with cold injuries of the lower extremity are litter patients. In the field, patients with MORE THAN FIRST DEGREE FROSTBITE SHOULD BE EVACUTED AS SOON AS POSSIBLE to a definitive treatment facility, since the extent of injury may not be readily apparent, and convalescence is usually prolonged.

C6.2.4.2. Rest

Rest of the affected limb or limbs should be enforced until recovery. If lower limbs are involved, bed rest is required.

C6.2.4.3. Blood Flow

"Hemodilution" (actually, optimizing hydration and circulating volume) is done to address dehydration (often present, especially in high altitude-related cold exposure injuries) and microcirculation defects [PMID 1483773].[447]

Anti-inflammatory agents (non-steroidals such as ibuprofen and aspirin) have been recommended to decrease systemic levels of thromboxane [PMID 2243830][448] (suspected to have a role in frostbite tissue damage [PMID 7204918]).[449] Several other agents targeting thromboxane and prostaglandin (to inhibit the "arachidonic acid cascade" [PMID 3631670])[450] have been promoted, including Aloe vera [PMID 2243830],[451] and methylprednisolone [PMID 3631670].[452]

Smoking is absolutely prohibited during recovery from frostbite [NAVEDTRA 13147-A].[453]

C6.2.4.4.---Hospital or Advanced Care Facility (Non-Field Therapy)

If frostbite is suspected and it is greater than level 1, immediately remove from cold and evacuate. Treatment in a non-field environment includes:

- Dextran for hemodilution
- Thrombolytics (tPA), streptokinase
- Vasodilators (nitroglycerine, reserpine, iloprost) [Gupta et al., 2021][454]

C6.2.4.5. Hyperbaric Oxygen

Hyperbaric oxygen has been used successfully on frostbite [PMID 11348755][455][PMID 20514135][456][PMID 17521112][457][PMID 11952063].[458]

C6.2.4.6. Surgery

Debridement without anesthesia may be done to help visualization of tissue [PMID 1483773].[459] Surgical amputation, if necessary, should be delayed 60 to 90 days (minimum of 3 weeks), unless sepsis occurs [PMID 1483773][460] [NAVEDTRA 13147-A].[461]

C6.2.4.7. Prevention of Infection

Tetanus toxoid booster is appropriate, if required, as tetanus is a known complication of frostbite [PMID 8323232].[462] Prophylactic antibiotics have not been found to prevent wound infection [PMID 8356126].[463]

Meticulous attention should be given to signs of infection. Mild antiseptics may be added to whirlpool baths. Blisters that may form do not require removal unless they impede joint motion, are large [PMID 6884849],[464] or show signs of infection.

C6.2.4.8. Recovery

Those who apparently have recovered from frostbite often have sequelae, including hypersensitivity to cold, numbness, declined sensitivity of touch, and decreased working ability with affected fingers. In one study, the skin temperature of frostbitten areas exposed to cold air decreased more quickly and reached lower values than in healthy control subjects [PMID 10998831].[465]

Neurosensory complications post freezing injury can include abnormal thermal and/or vibration perception. This can last up to 4 months or longer after the initial injury. Pain and or discomfort from exposure to cold, sensations of cold, and white fingers can last up to 4 years from initial injury [Carlsson et al., 2014][466]

Table 10 - Frostbite Rewarming Protocol

Pre-thaw
Prevent pressure on the injured part as much as possible. Do not rub or massage. Stay off feet if possible (if patient cannot be carried, walking is better than hypothermia) Do not try to move joints in areas already frostbitten. Remove victim from cold exposure ASAP. Do NOT thaw or warm until there is NO chance of re-freezing. Maintain adequate hydration
Thawing
Immerse in warm water (96.8° F to 108° F) Tetanus booster if needed. Ibuprofen 400 mg by mouth every 4 hours. Establish IV access, and maintain adequate hydration (orally or IV) **** Parenteral analgesics as needed. Heparin IV **** Encourage gentle motion of the affected part, but do not massage or force flexion or extension. Consider: pentoxifylline, fibrinolysin, streptokinase, hyperbaric oxygen, dextran***** Smoking is prohibited
Post-thaw
Elevate injured part and keep dry. Leave vesicles (blisters) intact unless signs of infection. Debride broken vesicles and apply topical antibiotic**** Limited debridement without anesthesia (and that does not cause victim pain!) as necessary to visualize tissue. Surgery after 2 or 3 months if necessary**** Smoking is prohibited until no further recovery is expected, and then is strongly discouraged

**** At hospital or appropriate level of care****

C6.3 Trench Foot

The Textbook of Military Medicine describes four immersion foot syndromes: trench foot, immersion foot, tropical immersion foot, and warm water immersion foot. Trench foot is distinguished from immersion foot only by whether or not the foot was actually immersed, and not just wet.[467] (The term "immersion foot" as used here is to be distinguished from the term "immersion foot" used to refer to "tropical immersion foot" and "warm weather immersion foot," which refer to water-related foot injuries not related to cold exposure, that have also been called such colorful names as "swamp foot," "jungle rot," etc. [PMID 2012466].)[468]

Trench foot (immersion foot) is a cold injury to extremities exposed to non-freezing temperatures, usually prolonged exposure involving moisture. On exposure to water from 32° F to 59° F (0° C to 15° C), clinical trench foot will develop if exposure lasts 12 to 48 hours (depending on the water temperature). Contributing factors include:

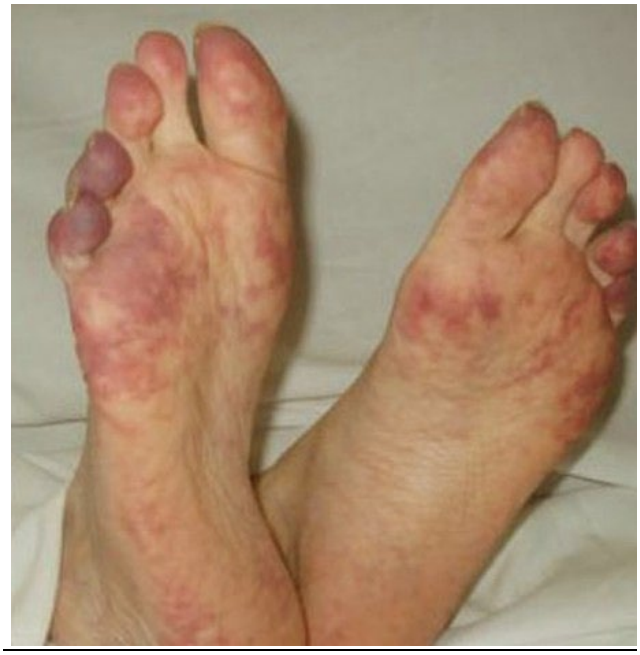
- Nutritional deficiency
- Trauma (rubbing or walking on affected feet)
- Wind
- Improper clothing type and integrity,
- Circulatory stagnation and tissue anoxia from dependency
- Inactivity
- Hemorrhage
- Shock
- Improper technique used to rewarm an injured limb.[469]

Trench foot is a very serious injury that may result in permanent nerve or tissue damage, or, untreated, may require amputation [PMID 6115374,[470] CHIPPM TN/02-2].[471]

C6.3.1 The 3 Stages of Trench Foot

Pre-hyperemic: Symptoms begin with cold, then numbness, paresthesia's, and itching. Weight bearing may be painful. With continued cold exposure, numbness progresses to anesthesia, with the classic complaint of "walking on blocks of wood." Signs include pallor, mottling or purple coloration, swelling, vesicles, bullae, and edema. There may be a "water-line" coinciding with the water level in the boot [CHIPPM TN/02-2].[472]

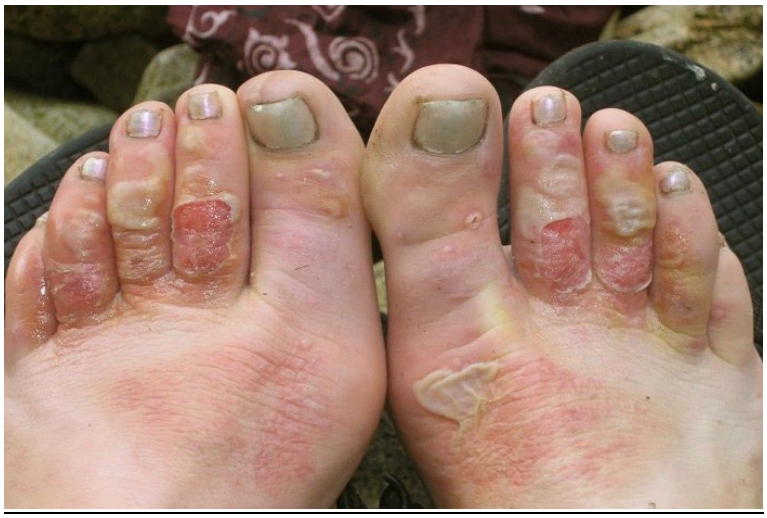
Figure 6-7 Pre-hyperemic Trench Foot



[Trench Foot – Causes, Symptoms, Treatment, n.d].[473]

Hyperemic: On or after rewarming, sensation returns proximally first, with paresthesia's and burning or throbbing pain. Heat sensitivity is increased. Hypoesthesia, further swelling, and erythema may develop. Mottling and discoloration may appear or increase. Blisters, circulatory compromise, local hemorrhage, and ecchymosis may characterize severe cases. Recovery of less severe cases may take up to 4 weeks, with exfoliation and possible scarring.

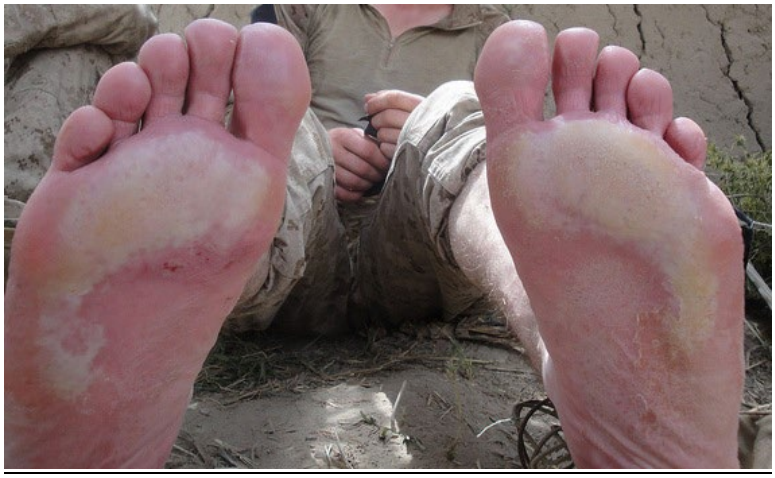
Figure 6-8 Hyperemic Trench Foot



[Trench Foot – Causes, Symptoms, Treatment, n.d.][474]

Post-hyperemic: More serious cases have a prolonged post-inflammatory phase involving compromised blood supply. Signs and symptoms include cyanosis, mottling, pain (distal and small joints), hyperesthesia (excessive physical sensitivity), paresthesia (tingling, pins and needles), anesthesia, atrophy of skin and muscles, osteoporosis, and contractures (especially clawfoot). Vascular and microvascular abnormalities related to sympathetic vasoconstriction, thrombosis, and increased vascular permeability may be involved.

Figure 6-8 Post-hyperemic Trench Foot



[Trench Foot – Causes, Symptoms, Treatment, n.d.][475]

Trench foot may result in peripheral neuropathy, and the proposed mechanisms of injury include direct axonal damage, ischemia, and ischemia-reperfusion. In mild or early cases, large, myelinated fibers are preferentially damaged, while small myelinated and unmyelinated fibers are relatively spared. Nerve damage starts proximally and extends distally with time [PMID 8712655].[476] In severe non-freezing cold injury cases, all nerve populations (myelinated and unmyelinated) may be damaged [PMID 9306996].[477] Axonal degeneration has been attributed to free radicals released during cycles of ischemia and reperfusion; however, the administration of

commonly used antioxidants has not been found to prevent cold nerve injury [PMID 12363167].[478]

C6.3.2. Treatment of Trench Foot

Treatment consists of bed rest, elevation of the legs, and air-drying at room temperature, while keeping the rest of the body warm (i.e., treating or preventing hypothermia). Rewarming of trench foot should be more gradual, passive, and at temperatures lower than recommended for frostbite. Careful foot hygiene is important. Antibiotics may be used for signs of infection (covering for Staphylococcus, Streptococcus, and Pseudomonas until culture results are available [PMID 2012466]).[479] Non-steroidal or even narcotic analgesics may be necessary. Tetanus immunization should be provided if not up to date.

Diet should have adequate protein. Smoking and other use of tobacco products (or nicotine-containing aids to smoking cessation) are contraindicated.

Treatment of post-hyperemia is directed at rehabilitation, including physical therapy, exercise, and surgical correction of deformities.

C6.3.3. Disposition

The prognosis depends upon the extent of the original tissue damage, especially nerve damage. Minimal and mild cases can resolve in hours to weeks and most eventually return to full duty. However, more severe cases can take months to heal, may require surgery, and victims may not be able return to full duty.

Military personnel who have previously suffered trench foot injury may be at increased risk for future cold injury [PMID 1969264].[480]

C6.4. Ocular (Eye) injuries Due to Cold Exposure

C6.4.1. Snow Blindness (Acute Photokeratitis, Solar Keratitis)

Snow blindness is said to be the most common acute ocular effect of environmental ultraviolet (UV) radiation [PMID 9894351].[481] It is caused by exposure of the cornea to ultra-violet radiation, often in snow conditions. It is similar in mechanism, treatment, and course to "flash burns" caused by ultraviolet light given off during welding.

Symptoms: Eye pain and photophobia, and may include tearing, conjunctival injection (redness), swollen eyelids, foreign body sensation (a "gritty" feeling in the eyes), blurred vision, and headache.

Treatment: with a short-acting cycloplegic drop (e.g., cyclopentolate 1% or 2%, or tropicamide 0.5% or 1%, or scopolamine 0.25%, to relieve painful ciliary spasm) and a topical anesthetic given by the health care provider should be adequate for immediate pain relief. (Potent topical anesthetics, such as proparacaine, should not be prescribed for, or sent home with, the

patient.) Topical antibiotic solution, suspension, or ointment (erythromycin, bacitracin, trimethoprim / polymyxin, tobramycin, or gentamicin) may help prevent infection. Nonsteroidal anti-inflammatory drugs (NSAIDs, such as ibuprofen) and small amounts of oral narcotic analgesics may be used for pain control. Topical ophthalmic NSAIDs (e.g., diclofenac, ketorolac tromethamine 0.5%) may be useful for pain relief [PMID 12514694].[482]

C6.4.2. Corneal Frostbite

Corneal frostbite is a serious ocular injury. It is generally caused only by exposure to extreme cold (e.g., liquid nitrogen splashed in the eye) or by environmental cold exposure involving loss of consciousness, defect of the eyelid, or other condition causing diminished ability to protect the eye. It may result in loss of vision.

Initial care includes treating or preventing hypothermia; rewarming must avoid water, saline, or air temperatures above 100.4° F (38° C). As the protection normally given by the eyelid may be compromised, care should be taken to maintain hydration of the cornea and to prevent trauma. Artificial tears without mercurial or benzalkonium antibacterial additives should be used every 15 minutes or more frequently; if artificial tears are unavailable, sterile normal saline may be substituted. Eye shields may be used. Do not patch (i.e., avoid anything touching the cornea). Topical antibiotic solution (not ointment) should be given (e.g., erythromycin or tobramycin) if there will be delay in ophthalmology evaluation. Definitive treatment by an ophthalmologist should be sought.

Chapter 7 Hypothermia

Hypothermia occurs when the body temperature drops below 95.0º F (35.0º C) [CDC].[483] Hypothermia is usually associated with cold or freezing temperatures but can also occur in non-freezing situations if exposed to the elements without appropriate shelter or clothing (Brown et al., 2012)[484]. As opposed to the extremities or a limited area of skin by cold stress, hypothermia refers to core or whole-body temperature at or below 95.0º F (35.0º C) [Dorland][485]

Although hypothermia is a serious cold stress injury, lowered body temperature has limited application in certain medical procedures and treatments such as cardiac surgery (Parham et al., 2009)[486] and neurosurgery [Dietrich, 2009][487].

C7.1. Categories

Hypothermia may be thought of as:

- **Primary:** due to cold exposure
  - Acute: due to cold exposure of less than 6 hours duration
  - Chronic due to cold exposure of greater than 6 hours duration
- **Secondary:** related to underlying or predisposing health-related conditions, such as illness, injury, intoxication, or extremes of age. [Currier].[488]

C7.2 Symptoms and Signs of Hypothermia

Symptoms of hypothermia begin subtly with fatigue and loss of concentration [PMID 1901977].[489] Ataxia, impaired judgment, oliguria, and slight confusion may be subtle symptoms [PMID 12092964],[490] but may progress to stupor, apathy, poor decision making, somnolence, and eventually coma.[PMID 1901977].[491] Individuals may not reliably assess whether they are experiencing hypothermia, especially under certain conditions (e.g., during immersion) [PMID 2803163,[492] PMID 3795105].[493] A useful mnemonic for recognizing hypothermia in the field is to watch for the "umbles": grumbling, mumbling, stumbling or fumbling [PMID: 31740369].[494]

Hypothermia shifts the oxyhemoglobin-dissociation curve to the left, resulting in decreased oxygen delivery to tissue [PMID 16730,[495] PMID 9239580,[496] PMID 7984198].[497] Additionally a decrease in ventilation response leads to increased carbon dioxide levels and respiratory acidosis (Dow et al., 2019).[498] A review article estimated a hematocrit increase of 2 percent for every 1.8° F (1° C) decline in temperature [PMID 7984198].[499]

C7.3. Classification of Hypothermia

Hypothermia may be classified as mild (90.0° F to 95.0° F, or 32.2° C to 35.0° C), moderate (82.5° F to <90.0° F, or 28.0° C to <32.2° C), or severe (<82.5° F, or <28.0° C) [CDC].[500] The cut-off temperatures for the various categories are not universally agreed upon in the literature, but they are within what would be expected with different (Fahrenheit and Celsius) systems in use.

Table 103 - Classification of Hypothermia

Severity	Symptom	Fahrenheit Core Temp	Celsius Core Temp
Mild	Conscious and Shivering	90.0° to 95.0°	32.2° to 35.0°
Moderate	Altered Mental Status (not shivering)	82.5° to < 90.0°	28.0° to < 32.2°
Severe	Unconscious (vital signs present)	< 82.5°	< 28.0°

C7.3.1 Temperature Measurement.

The most accurate standard for measuring core temperature in a hospital setting is the esophageal temperature. However, this requires that the airway be protected, and the placement is approx. 24cm below the larynx in adults. [Dow et al., 2019][501] This is not practical for use in the field or out of hospital situations.

Tympanic thermometer has low accuracy and is affected by ambient temperature and positioning. Additionally, will not read correctly if snow/ice/water is in ear or if there is an abundance of wax. [Niven et al., 2015][502]

The Rectal thermometer is not recommended due to difficulties with access requiring removing clothing which can cause further exposure to the victim. [Niven et al., 2015][503]

Oral thermometer. Low accuracy and is influenced by multiple factors. Not reliable in hot or cold environments. Nonelectric versions do not measure temperatures below 35.6°C (used to rule out hypothermia) [Niven et al., 2015].[504]

There is no single best option for temperature measurement in an out of hospital (field) situation. Systemic signs and symptoms are more useful for diagnosing hypothermia. Treatment should not be delayed for temperature assessment.

C7.4 Predisposing Factors for Hypothermia

Table 11 - Predisposing Factors in Hypothermia

Environmental Factors	Metabolic factors	Treatment related
Inadequate clothing Lack of Shelter Windchill High humidity Perspiration Wet Clothing	Drugs Alcohol Hypoglycemia/malnutrition Sepsis Shock Burns Traumatic injury Acidosis/Anoxia	Lotions or exfoliative skin disease Large volumes of IV fluids Removing clothing. Overly aggressive treatment of hypothermia

Hypothalamic dysfunction and decreased ability to seek shelter from the cold also may contribute. Sepsis, endocrine dysfunction (diabetic ketoacidosis, hypoglycemia, hypothyroidism, hypopituitarism, hypoadrenalism), CNS disorders (stroke, brain tumors, spinal cord injury), and skin conditions (burns, erythroderma, psoriasis, ichthyosis) are additional predisposing factors [Currier].[505]

Underlying predisposing causes of hypothermia are diabetic ketoacidosis, cerebrovascular disease, mental retardation, hypothyroidism, pituitary and adrenal insufficiency, malnutrition, acute alcoholism, liver damage, hypoglycemia, sepsis, hypothalamic dysfunction, poly-pharmacy, and the use of sedative and narcotic drugs [PMID 11759373].[506] History of orthostatic hypotension is a risk factor for developing accidental hypothermia [PMID 4065579].[507]

C7.5. Assessing The Cold Patient

C7.5.1 Cold Stressed

When evaluating a cold stress victim, they may be cold and shivering but not necessarily hypothermic. If the victim is alert and/or shivering and can function and care for themselves are considered cold, but not hypothermic.

C7.5.2 Mild Hypothermia

Need to differentiate between mild and moderate hypothermia. Shivering can be seen with both. The difference is in mental status. Mild hypothermic patients are alert, shivering, but will have abnormal cognitive function.

C7.5.3 Moderate Hypothermia

Due to the low core temperature, shivering may cease but some patients have continued to shiver below 31°C. Cognitive dysfunction is pronounced at this stage to include ataxia, hallucinations, dysarthria. Loss of consciousness is not uncommon with core temperatures between 28°C and 30°C. The risk for cardiovascular injury is also higher with findings of bradycardia, atrial fibrillation, ventricular fibrillation, AV block, and J-waves. [Vogel & Hulsopple, 2022].[508]

C7.5.4 Severe Hypothermia

At core temperature of 28°C and below, the patients are not shivering and likely are unconscious. The risk of ventricular fibrillation or asystole is extremely high. [Paal et al., 2022].[509]

****If hypothermia is suspected or a possibility – immediately remove from cold stress and transfer to closest hospital with ICU****

****Attempts to rewarm should not delay transport****

C7.6 Treatment of Hypothermia

C7.6.1 Initial care (Out of Hospital Treatment)

Initial treatment involves removing the victim from cold stress. Further actions are based on location, situation, and estimated arrival time of transport to nearest hospital or emergency care facility.

Rapid core rewarming, airway control, and prolonged cardiopulmonary resuscitation have been noted to be key factors in managing the hypothermic patient [PMID 8121213].[510] Treatment should take into account not only the degree of hypothermia, but also exposure time, state of consciousness, and complicating factors such as trauma, drugs, or alcohol [PMID 8236180].[511]

Before any treatment begins, if possible, move the patient out of the weather and or exposure into a protected area (tent, cabin, building, etc.) to prevent further cold exposure.

C7.6.2 Cold stressed and/or mild hypothermia

The most effective method of rewarming is shivering for a cold stressed to a mild hypothermic patient. Shivering can increase heat production up to 5-6 times the resting metabolic rate. However, shivering is uncomfortable and can stress the cardiovascular system, active rewarming methods are preferred. High carbohydrate drinks and warmed food assist with the rewarming but ensure drinks are not hot enough to cause burns. [Zafren, 2017][512]

If a hypothermic patient is found sitting or lying down, they should remain in a lying or sitting position. Standing them up or having them walk or stomp their feet will cause an increase of blood flow from cold legs with a return to the heart with cold blood further decreasing core temperature (After-drop). The patient should be observed for at least 30 minutes, insulated, and given high calorie food or drinks. If the patient can tolerate standing without issues can slowly walk, and only if tolerated can begin to gradually increase speed. [Zafren, 2017][513]

C7.6.2.1 After-drop

After-drop is a phenomenon of conductive heat loss. After-drop refers to a continued decrease in measured body temperature after removal from cold stress exposure. During rewarming, the peripheral tissue is colder than the heart, so any action that increases blood flow to the periphery will increase the cold blood returning to the heart (core). This is important to consider for those who are at the threshold of moderate to severe hypothermia. [Dow et al., 2019].[514] After-drop can lower core temperatures as much as 5° to 6°C during pre-hospital care [Baumgartner et al., 1992][515]

C7.6.3 Moderate to Sever Hypothermia

Moderate to severe hypothermia treatments include active external warming and active internal warming. However, the techniques and modalities for internal warming are not available outside of an established care facility. If it is possible to warm isotonic saline (40°C to 42°C) then that should be considered. IV access will also be more difficult due to systemic vasoconstriction and diminished heart rate/pulse, therefore, consider intraosseous access for resuscitation [Vogel & Hulsopple, 2022].[516]

C7.6.4 Potential Complications

Serious complications of hypothermia include hemorrhagic pancreatitis, lung edema, and myxomatous skin edema [PMID 10998830].[517] Hypothermia increases coagulation time and clot formation time; investigators found that hypothermia-induced coagulation changes are worsened by acidosis (the same degree of acidosis without hypothermia had no significant effect on coagulation) [PMID 18499589].[518]

C7.6.5 Rewarming Methods

C7.6.5.1 Rewarming Methods Passive

The goal is to stop the loss of heat by wrapping the victim to create a vapor barrier (tarp, plastic sheet, sleeping bag, emergency blanket, etc.). Place the victim off the ground or on an insulated surface to stop the heat from leaching out of the body. Ensure that the outer layer is as robust and windproof as possible. Hand coverings (gloves, mittens), head, neck, and face coverings should be applied.

If clothing is damp or dry leave the clothing on and wrap the victim.

If the victim is wet AND transportation is less than 30 minutes away, keep clothes on and wrap the victim.

However, if transport is greater than 30 minutes away, remove clothing and wrap the patient and continue to assess patient. [Dow et al., 2019].[519]

In a field situation, mild hypothermia is enough to cause impaired manual dexterity (by more than 50%). For this degree of hypothermia (mild),recovery can be achieved using passive rewarming. Additionally, promoting active movements and/or eating a high calorie meal is usually sufficient [Niven et al., 2015].[520]

The following have shown efficacy for treating cold to mild hypothermia indicating field rewarming is beneficial for the warfighter [PMID: 32682705].[521]

- A sleeping bag alone.
- A sleeping bag plus warm liquid(coffee, tea).
- Exercise. (slow walking, basic movement)

C7.6.5.2 Rewarming methods Active.

Active rewarming includes all the passive steps and includes the addition of external heat sources such as chemical heat packs, heated blankets, heat pads, or forced warm air devices. The focus should be on the chest/trunk to help prevent after-drop **(see after-drop above)** from returning cold blood from the periphery.

Caution: Using external heat methods will necessitate the need for skin checks to prevent burns from occurring.

C7.8 Medical Considerations

The risk of cardiac arrhythmias increases with the degree of hypothermia from least (mild) to greatest (severe). If the patient is not in cardiac arrest, priorities are to avoid causing cardiovascular collapse.

C7.8.1. Cardiopulmonary resuscitation (CPR)

In a rescue scenario with a hypothermic patient, continuous CPR may not be possible due to location (unsafe), injuries, or medical personnel. Delay to CPR and or interruptions should be as short as possible. In order to move a patient to a safer location. [Paal et al., 2022].[522]

Hypothermic patients in cardiac arrest should receive high quality CPR, but detecting signs of life in a cold patient can be difficult due to:

- Pulses can be faint and slow (less than 50 bmp)
- Pulses can be hard to find (cold fingers of rescuers)
- Slow and shallow breathing

Carotid pulse should be assessed for 1 minute before starting CPR with ventilations. CPR should be discontinued if there are signs of life. [Zafren, 2017].[523]

The brain is protected from hypoxia by hypothermia by reducing brain activity. [Michenfelder & Milde, 1991].[524] It has been reported that full neurologic recovery after cardiac arrest from hypothermia has been seen with delays of CPR 15 minutes [Oberhammer et al., 2008][525] and 70 minutes [Althaus et al., 1982].[526] In another case the full neurologic recovery was achieved after total circulatory arrest for over 30 minutes [Truhlář et al., 2015].[527]

C7.8.2 Cardiac monitoring/defibrillation

Defibrillation pads should be used as they decrease artifact due to shivering. This also will allow for quick detection of shockable rhythms. [Paal et al., 2022].[528] Defibrillation should on be performed for a shockable rhythm (pulseless VT or VF). Current guidelines recommend single shock at maximum power if the patient's core temp is below 30°C. If unsuccessful, wait until core temperature is greater than 30°C as defibrillation is not as effective at temperatures below 30°C. Once core temperature reaches 30°C follow standard protocol [Dow et al., 2019].[529]

C7.8.3 Ventricular Fibrillation

To prevent VF gentle handling of the patient is required. Hypothermia lowers the threshold for VF and the greatest risk is at 28°C or below. Keep patient horizontal and do not have the patient move due to having additional cold blood from the periphery further cool the heart and increase the load on the already ineffective pump. [Paal et al., 2022].[530]

C7.8.4 Atrial Fibrillation

Due to hypothermic irritation of the heart, atrial dysrhythmias can occur during the rewarming. These should be watched but not treated in otherwise hemodynamically stable patients. This will resolve during or after rewarming. [Zafren, 2017].[531]

C7.8.5 Respiration

Ventilations for a hypothermic patient are less effective but should be given at the same rate as a normothermic patient. Advanced airways will provide more effective ventilations, but there is difficulty in having one placed. Ideally endotracheal intubation should be deferred until the patient is in a warm environment with weight-based settings not relying on $ETCO_2$ [Brown et al., 2012].[532]

If patient is above sea level (greater than 2500m), consider supplemental oxygen due to the decreased percentage in the air. [Zafren, 2017].[533]

C7.8.6 Medications

Cardiovascular medications including epinephrine and amiodarone should not be given if the core temperature is <30 °C. There is insufficient human data to justify recommend medications for core temperatures less than 30 °C. The metabolism for the drugs are decreased due to reduced blood flow and protein binding is increased. When the patient begins rewarming, the drugs will become bioavailable all at once at toxic levels. [Dow et al., 2019].[534]

Epinephrine alone can be given but time is doubled to every 6-10 minutes to account for the slowing of metabolism. Once core has achieved normothermia (≥ 95 °F), standard protocols can resume. [Zafren, 2017][535]

C7.8.7 IV access/fluids

Due to the reflex vasoconstriction from cold stress, peripheral vascular access will be difficult to access. Therefore, intraosseous access should be used to provide warmed IV fluids (100.4–107.6 °F).[Paal et al., 2022].[536] Due to low temperatures, fluids (if given) should only be isotonic (normal saline) and done in a rapid bolus to prevent cooling or freezing of lines from IV bag to the patient [Zafren, 2017].[537]

C7.9 Circumrescue

Circumrescue is a phenomenon that occurs immediately before or during the rescue of a cold immersion and or hypothermic patient. This refers to light-headedness, collapse, syncope, or sudden death that can be caused by mental relaxation and decreased catecholamine output causing life-threatening hypotension or by sudden onset of cardiac dysrhythmia, likely VF [Giesbrecht, 2000].[538]

When removing a victim from water decreases hydrostatic pressure which is normally greatest around the legs. Cold water immersion (see above) increases blood pressure by vasoconstriction. When removed from water, loss of hydrostatic pressure allows blood to pool in dependent areas, causing decreased blood return with resultant hypotension or cardiovascular collapse. [Giesbrecht, 2000].[539]

Additionally, a heart that is cold from immersion and or cold may not be able to compensate for decreasing blood pressure by increasing cardiac output. To compound this, the blood returning from periphery will be cool further contributing to decrease in core temp in the form of after drop (see above). Any movement that the patient has to perform will contribute to after-drop (having to climb a ladder onto a boat [Giesbrecht, 2000].[540]

C7.10 In Hospital Care

While beyond the scope of this manual, some of the treatments that can occur for hypothermia in a hospital setting include all of the above as well as:

- Sternotomy
- Peritoneal re-warming
- Cardiopulmonary Bypass
- Intravenous Rewarming

C7.11 Prognosis in Hypothermia

The prognosis is excellent in patients in whom no hypoxic event precedes hypothermia and no serious underlying disease exists [PMID 8719198].[541]

Out of a group of 32 victims, age 15 to 36 years, of severe hypothermia (core temperature less than 82.4° F (28° C), with circulatory arrest) that received rewarming with cardiopulmonary bypass, 15 survived. Follow-up for over 5 years of survivors showed neurological and neuropsychological deficits observed in the early period after rewarming had fully or almost completely disappeared. Investigators concluded that that young, otherwise healthy people can survive accidental deep hypothermia with no or minimal cerebral impairment, even with prolonged circulatory arrest, and that cardiopulmonary bypass appears to be an efficacious rewarming technique [PMID 9366581].[542]

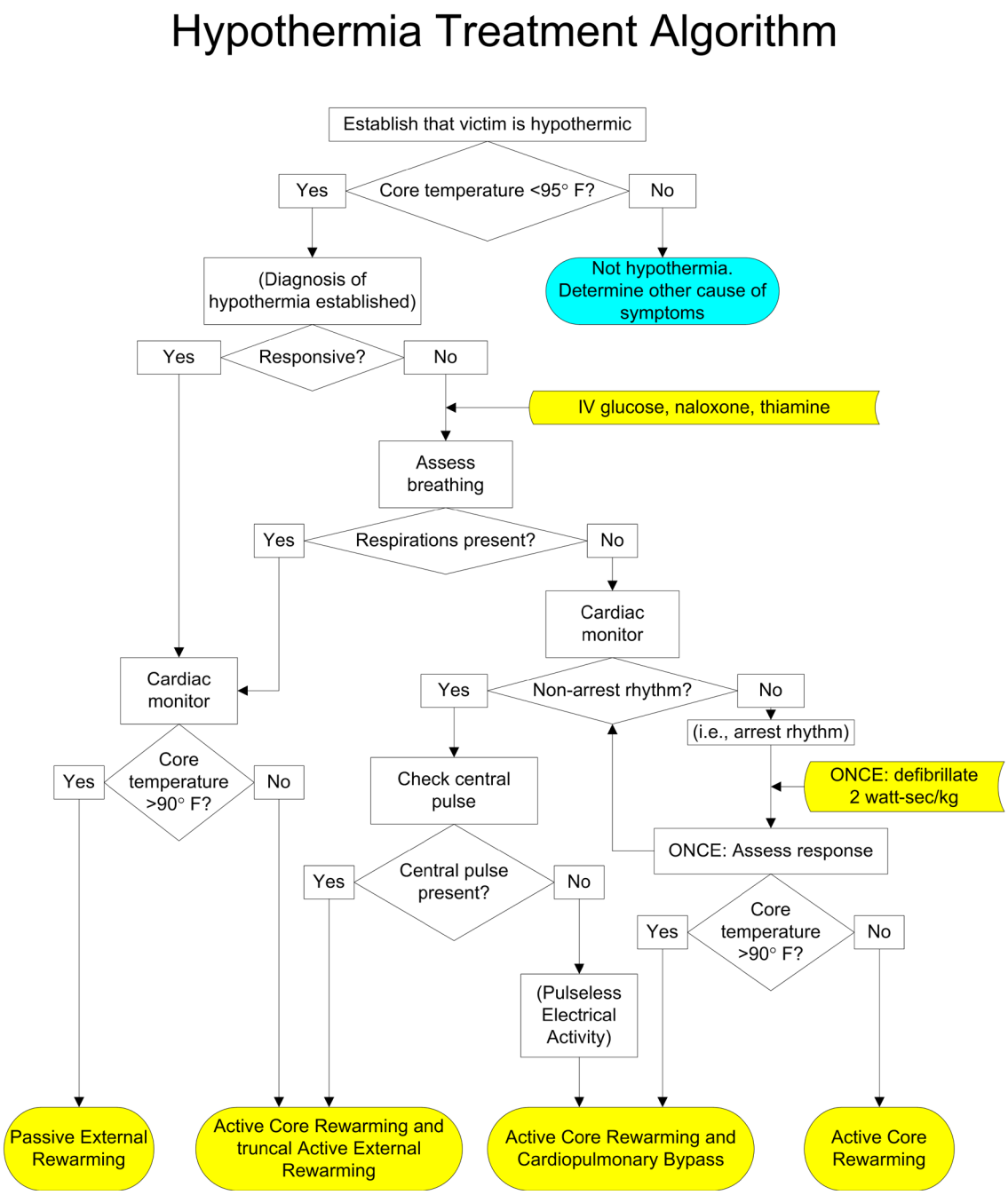
C7.12 Public Health Impact of Hypothermia

Hypothermia is not limited to individuals outdoors during excessively cold conditions. Only approximately half of deaths from hypothermia were attributed to extremely cold weather [CDC].[543] The CDC states that hypothermic mortality is underreported, because its physical signs resemble other conditions and may not be recognized, hospitals may not use low-temperature thermometers, medical personnel may be unaware of hypothermia's significance, and an autopsy cannot prove hypothermia as an underlying cause of death [MMWR].[544]

C7.13 Prevention of Further Cold Stress injuries in the Population

When a cold stress injury is recognized, steps should be taken to prevent others in the involved population from cold stress injury. The victim may serve as a sentinel event, alerting health care workers, safety, and supervisors to the existence of a cold-related health risk. Training (or retraining) on cold stress injurie s may be appropriate (see OPNAV M-5100.23).[545] A check of environmental thermometers or WBGT equipment should be done, if it is possible that faulty equipment may have contributed to the cold stress injury. Adequacy of clothing and heating facilities (HVAC system, if present) should be verified. With appropriate measures, most cold stress-related injuries can be prevented.

Figure 7-1 - Hypothermia Treatment Algorithm



Acronyms

ACGIH	American Conference of Government Industrial Hygienists
ARDS	Adult Respiratory Distress Syndrome
BMI	Body Mass Index
CNS	Central Nervous System
EKG	Electrocardiogram
GI	Gastrointestinal
IV	Intravenous
Mmol	Millimole
MRA	Magnetic Resonance Angiography
MRI	Magnetic Resonance Imaging
MOPP	Mission Oriented Protective Posture
NBC	Nuclear, Biological, and Chemical
NSAID	Nonsteroidal Anti-Inflammatory Drug
OR	Odds Ratio
ORS	Oral Rehydration Salts
PHEL	Physiological Heat Exposure Limits
PPE	Personal Protective Equipment
SIPE	Swimming Induced Pulmonary Edema
SIPO	Swimming Induced Pulmonary Oedma
Tc	Technetium
UV	Ultraviolet
WBGT	Wet Bulb Globe Temperature Index

Chapter 8: References

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