

PREVENTION AND TREATMENT OF HEAT AND COLD STRESS INJURIES



NAVY ENVIRONMENTAL HEALTH CENTER

BUREAU OF MEDICINE AND SURGERY

**PREVENTION AND TREATMENT OF HEAT AND COLD
STRESS INJURIES**

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FOREWORD

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Reviewed and Approved

A handwritten signature in black ink, appearing to read 'W. R. Stover', is written over a horizontal line.

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Commanding Officer

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Chapter 1:

C1. 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. With the publication of this revised technical manual, all previous Navy documents dealing with the prevention and treatment of heat and cold stress injuries will be obsolete and replaced by this document and the P-5010.

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 Outdated, erroneous thinking about developing “dehydration tolerance” or that one’s unit can “stand” more than another because they are “tougher” or “more motivated”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. **All supervisors should be familiar with the documents in [C11. Heat Stress Injuries Prevention and Treatment](#) and [C12. Cold Stress Injuries Prevention and Treatment](#)** which briefly summarize relevant information for those not involved with patient care. The main document text gives further details.

Chapter 2:

C2. 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 (which is actually a form of convection). 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 1180844](#)],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: $M + R + C - E = S$

C2.1.2. Heat Dissipation (Removal)

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 even when surrounding temperatures are greater than body temperature.

Sweat gland function increases when skin (not air) temperature approaches 95° F (35° C) [[CHIPPM](#)].² Sweating depends on sufficient hydration, adequate blood flow to the skin, and proper operation of sweat glands, which are under neurologic [[PMID 1442139](#)]³ and hormonal control (including growth hormone [[PMID 11474638](#)],⁴ [[PMID 11106921](#)],⁵ sex hormones [[PMID 1947733](#)],⁶ and prolactin [[PMID 3780532](#)]⁷ [[PMID 3397041](#)],⁸ and subject to pharmacologic influence, such as cholinergics or anticholinergics [[PMID 2120621](#)].)⁹ Women tend to sweat less profusely than men [[PMID 10414066](#)]¹⁰ [[PMID 8504843](#)].¹¹ Sweating efficiency is not influenced by exercise intensity [[PMID 8851527](#)].¹² Sweating efficiency in

removing body heat is influenced by environmental factors, especially humidity (reflecting the partial pressure of water in the atmosphere) and wind speed.

Theoretically, water can remove up to 540 calories (kcal) per liter (heat of vaporization of water = 540 calories/gram = 0.54 kcal/g = 2260 joules/g). However, sweating is less efficient at cooling than that. Not all sweat evaporates, especially at high rates of sweating (or low rates of evaporation, as in high humidity), when sweat may run or drip off the body or be absorbed by clothing [[PMID 1193094](#)].¹³

C2.1.2.1.1. Skin Heat, Moisture, and Texture

A normal response to heat stress exposure is increased cutaneous blood flow and increased sweating [[PMID 481154](#)].¹⁴ 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. 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.

Cool dry skin is abnormal in the presence of heat stress, and may signal blood shunting away from the skin and the cessation of sweating. Cool moist (“clammy”) skin may signal that blood shunting away from the skin has occurred, with moisture on the skin remaining from previous sweat that has not evaporated due to a humid atmosphere.

C2.1.2.1.2. Blood Shunting

Blood shunting refers to diminished cutaneous blood flow, resulting in diminished ability to lose heat from the skin. During heat stress, skin blood flow can be 60% of cardiac output [[PMID 8504843](#)].¹⁵ In heat stress conditions, blood flow to the skin must be maintained so that heat loss (especially by evaporation through sweating) can occur. However, during exercise, blood flow to muscles dramatically increases. For example, calf blood flow rises 20-fold with exercise [[PMID 10747200](#)].¹⁶ Vigorous exercise appears to limit cutaneous blood flow [[PMID 591471](#)].¹⁷ If cutaneous blood flow is limited by shunting away from the skin (as may also happen in severe heat injuries), heat loss diminishes and body core temperature may rise to hazardous levels.

In addition to blood shunting from cutaneous circulation, as body temperature rises, blood flow through splanchnic circulation initially decreases significantly (enabling maintenance of blood pressure). As temperature continues to increase, however, splanchnic circulation suddenly is re-established, possibly signaling loss of that compensatory mechanism [[PMID 3403442](#)].¹⁸

C2.1.2.1.2.1. 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](#)].¹⁹ 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.

C2.1.2.1.2.2. Drinks

C2.1.2.1.2.2.1. Water

To make water freely available to workers is essential; to encourage water drinking in heat stress conditions is important, as thirst can lag behind water deficit by several hours.

Water is an ideal rehydration drink. It is absorbed rapidly from the upper gastrointestinal (GI) tract, and it is generally inexpensive and readily available. However, many people find plain water less palatable than flavored drinks. Palatability of drinks is important in stimulating intake and ensuring adequate volume replacement [[PMID 9298549](#)].²⁰ 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](#)].²¹

C2.1.2.1.2.2.2. Sports Drinks

Sports drinks (Gatorade®, Powerade®, etc.), sometimes referred to as “carbohydrate-electrolyte (CHO-E) fluid replacement,” are generally sweetened, flavored drinks that contain some or all of the electrolytes lost in sweat. They are absorbed at approximately the same rate as water, and cost more. Sports drinks are acceptable fluid replacement beverages in heat stress conditions. However, that they are superior to water is not universally accepted; also, the need to use them to replace electrolytes is not established in conditions where a normal diet is consumed, workers are heat-acclimatized (as people are acclimatized, they lose less salt in their sweat), and there is not prolonged heat stress or time between eating or an unusual performance requirement. Sports drinks do have the advantage that, to some people, they taste better than water and thus are more likely to be consumed by those that do not like drinking pure water (and thus may be more likely to maintain better hydration). Sports drinks also contain sugar, which may be advantageous in special nutrition or performance situations. In one study, CHO-E 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](#)].²²

Potential caloric intake from sports drinks can be substantial. Five quarts of 5.8% carbohydrate solution (e.g., [Gatorade®](#))²³ would provide 1,120 kilocalories, and five quarts of 8% carbohydrate solution would provide 1,545 kilocalories; for this reason, sports drinks generally should not be used to totally replace water consumption.

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 [[CHIPPM](#)].²⁴

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.

C2.1.2.1.2.2.3. Oral Rehydration Salts (ORS)

ORS, when mixed with the prescribed quantity of water, may also be used as a rehydration liquid. However, ORS were developed as fluid replacement for GI loss and may have more salt (sodium) than is necessary. For example, ORS has about 2 grams (90 mmol) of sodium per liter [[WHO](#)],²⁵ while Gatorade® has less than 0.5 grams of sodium per liter [[Stokely-Van Camp](#)].²⁶

C2.1.2.1.2.2.4. 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.

C2.1.2.1.2.2.5. Alcoholic Beverages

Alcoholic beverages may 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.

C2.1.2.1.2.3. 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 17146319](#)].²⁷

C2.1.2.1.2.4. 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](#)].²⁸ High concentrations of sugar, complex carbohydrates, proteins, and, especially, fats, all may hinder water absorption, and thus are undesirable in water-replacement beverages. However, ingestion of carbohydrate-electrolyte drinks in the post-exercise period restores exercise capacity more effectively than plain water [[PMID 9298549](#)].²⁹ 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](#)],³⁰ [[PMID 2733575](#)].³¹ Mild exercise increases gastric emptying, while maximal exercise delays gastric emptying [[PMID 1928033](#)].³²

C2.1.2.1.3. Sodium (Salt)

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](#)].³³ Under certain extreme conditions, especially with protracted heat stress exposure and limited dietary sodium, supplemental salt may be required. However, medical consultation should be obtained before salt supplements are used.

C2.2. Environmental Heat Stress Factors

C2.2.1. Measurement of Temperature

The obvious and most environmental heat stress factor is temperature. The single best measure of environmental heat stress is the Wet Bulb Globe Temperature (WBGT) Index. The WBGT takes into account several factors, including temperature, radiant heat (sun), humidity, and air flow. It is calculated as follows.

$$\text{WBGT} = (\text{Wet Bulb} \times 0.7) + (\text{Globe Temperature} \times 0.2) + (\text{Dry Bulb} \times 0.1)$$

Thus, Wet Bulb (a thermometer covered by a wet cloth or wick, to account for evaporative cooling) accounts for 70% of the WBGT. (By Navy convention, evaporation in mild airflow is considered more representative of actual work conditions; hence the name “aspirated” wet bulb.) Globe Temperature (a thermometer enclosed in a black globe in the sun) accounts for 20% of the WBGT. Dry Bulb (a dry thermometer in the shade) accounts for 10% of the WBGT.

Currently, rather than calculate the WBGT from three separate instruments, the Navy uses 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 Index value. The approved Navy Heat Stress Meter is part number 70-6685-01-055-5298.

C2.2.2. Wind

Wind has a cooling effect that increases with air velocity. Under certain conditions, at approximately 1,500 feet per minute (17 miles per hour), friction begins to generate enough heat that the rate of cooling begins to decline; also, when air temperature exceeds 95° F (35° C), air velocities greater than 300 feet per minute (3.4 miles per hour) may be undesirable [NEHC-TM92-6].³⁴ However, in some conditions workers have requested supplemental intermittent cooling air at velocities as high as 3,000 feet per minute (34 miles per hour) [Jorgensen].³⁵

C2.2.3. Humidity

High humidity decreases the rate of water evaporation at a given temperature. Since sweating removes body heat by evaporative cooling, conditions of high humidity increase heat stress because sweating becomes less effective at cooling the body. Thus, strenuous exercise at 85° in 90% humidity (for example, in a temperate climate coastal area) may be more heat stressful than at 100° in 10% humidity (for example, in a equatorial desert area).

C2.2.3.1. 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. According to the National Weather Service,³⁶ a currently used heat index equation is as follows.

Figure 1- Heat Index Equation

$$\begin{aligned}
 \text{Heat Index} = & \\
 & - 42.379 \\
 & + 2.04901523T \\
 & + 10.1433127R \\
 & - 0.22475541TR \\
 & - 6.83783 \times 10^{-3} T^2 \\
 & - 5.481717 \times 10^{-2} R^2 \\
 & + 1.22874 \times 10^{-3} T^2R \\
 & + 8.5282 \times 10^{-4} TR^2 \\
 & - 1.99 \times 10^{-6} T^2 R^2
 \end{aligned}$$

where T = ambient dry bulb temperature (in degrees Fahrenheit between 80 and 110)
and R = relative humidity between 10 and 90 percent.

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. A heat index calculator is also available [here](#) for those using Microsoft Internet Explorer®. It is important to note that the “heat index” is not a complete measure of heat stress conditions, and THE HEAT INDEX IS NOT TO BE SUBSTITUTED FOR THE WBGT INDEX IN CALCULATING HEAT STRESS OR STAY TIMES.

C2.2.3.1.1. Physiologically Equivalent Temperature

The Physiologically Equivalent Temperature (PET) is a term referring to the indoor air temperature at which body core temperature and skin temperature is the same as in given conditions outdoors. It is based on a European model of heat balance (the Munich Energy balance Model for Individuals), and seeks to present a model of human body heating and cooling indoors that is equivalent to what would be experienced outdoors. For example, on hot summer days with direct solar irradiation, the PET value may be more than 36° F higher than the air temperature, on a windy day in winter up to 27° F lower [[PMID 10552310](#)].³⁷

C2.2.4. Rain

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

C2.2.5. 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. (It is to be cautioned that winter sun can still present a sunburn hazard.)

C2.2.6. Elevation

While temperatures are decreased with increased elevation, the health risk from heat stress is increased in deep mines. The incidence rate ratio of heat exhaustion for Australian mines operating below 1,200 meters compared with those operating above 1,200 meters was 3.17 [[PMID 10810098](#)].³⁸ 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.

C2.3. Individual Heat Stress Factors

C2.3.1. Sunburn

Sunburn (a result of over exposure to the sun, not discussed here) can decrease sweating, and thus may 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](#)].³⁹

C2.3.2. 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](#)]. 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](#)].⁴⁰ The American Conference of Governmental Industrial Hygienists (ACGIH) has suggested that WBGT values should be increased by 6.3° F (3.5° C) when cloth coveralls are worn, and 9° F (5° C) when double-cloth coveralls are worn (impermeable, water vapor resistant, and encapsulating suits excluded) (see [Table 5](#)) [ACGIH].⁴¹

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](#)].⁴²

C2.3.3. Illness

Underlying illness (infections, hypertension, diabetes, sickle cell trait [[PMID 8677839](#)],⁴³ congestive heart failure [[PMID 16216975](#)],⁴⁴ etc.) may impair the ability of the body to tolerate heat stress. Exposure to hot environmental conditions is not recommended for diabetic patients, although research-based support for such a recommendation, while not absent, is not robust at this time [[PMID 7698855](#)].⁴⁵ 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.), or may force persons to perform certain actions in such a way that more heat is generated than would be generated by persons without the disability.

C2.3.4. 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](#)].⁴⁶

C2.3.5. 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](#)].⁴⁷

C2.3.6. 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](#),⁴⁸ [PMID 15632673](#)].⁴⁹ Similarly, competition over several days in heat stress conditions has been associated with heat cramps in athletes [[PMID 8653105](#)].⁵⁰ Whether this is limited to exercise in heat stress conditions only subsequent to some degree of tissue injury is suspected by researchers (Sawka),⁵¹ but is not yet established in the medical literature.

C2.3.7. Size

Larger individuals generate more heat than others (simply because they have more body tissue undergoing metabolism). Additionally, the volume to surface area ratio is higher in large individuals. Thus, there is relatively less surface area (skin) through which to lose heat. The percentage of fat and the surface to mass ratio were found to have the largest effect in one investigation of the relative influence of fitness, acclimatization, gender and anthropometric measures on the physiological responses to heat stress [[PMID 2079061](#)].⁵²

Body mass index (BMI), also called Quetelet's index and used to evaluate obesity [[PMID 4030199](#)],⁵³ is used in calculations of heat stress. The BMI is calculated using body height and weight (mass). The BMI equation is $BMI = \text{weight in pounds} \div (\text{height in inches})^2 \times 703$. The metric system formula is $BMI = \text{weight in kilograms} \div (\text{height in meters})^2$. Units will be kg / M^2 [[CDC](#)].⁵⁴ The CDC also has a BMI calculator available on the Web [[CDC](#)].⁵⁵

C2.3.8. Population Characteristics

In civilian populations, there is an increased prevalence of heat injury risk factors, including older age, medication use (especially anticholinergic and psychotropic medications), obesity, previous heat injury, skin disorders [[PMID 9002705](#)],⁵⁶ and persons in whom is a greater number of chronic medical illnesses [[PMID 11434495](#)].⁵⁷

C2.3.9. Gender

Male and female body mass index values are different, due to the relative size differences. Water content varies slightly between males and females. Approximately 60% of an average 70 kg man's body weight is water. Of the 42 liters of water, 28 L is in intracellular fluid and 14 L in extracellular (mostly interstitial) fluid; female water content is somewhat lower because of the higher adipose tissue to lean body mass ratio [[PMID 10036337](#)].⁵⁸ This difference of water and fat content may be the reason that females have been found by some studies to be at a thermoregulatory disadvantage compared with males when wearing protective clothing and exercising in a hot environment [[PMID 9660153](#)].⁵⁹ (However, not all studies are in agreement with that finding [[PMID 655995](#)].)⁶⁰ A U.S. Army study ("the largest and most comprehensive epidemiological study of exertional heat injury") found that women are at increased risk of heat stroke [[PMID 16118581](#)].⁶¹

Core body temperature of females may increase (approximately 0.5 degrees) during the menstrual cycle. However, this temperature elevation has not been found to be significant in tolerating heat stress during exercise [PMID 750842],⁶² [PMID 7181811].⁶³ One study of females in the Canadian military found that exertional heat tolerance was increased during the early follicular phase in women who did not use oral contraceptives [PMID 10408316].⁶⁴ 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].⁶⁵

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].^{66, 67}

C2.3.10. Age

Young children and the elderly have decreased toleration of heat stress. For morphologic and physiologic reasons, exercising children do not adapt as effectively as adults when exposed to high climatic heat stress [PMID 10878169].⁶⁸ Aging is associated with decreased heat tolerance [PMID 3324259].⁶⁹ Whether this is due to age-related cardiovascular factors [PMID 3749652]⁷⁰ or other factors is uncertain [PMID 3324259].⁷¹ In older men, rectal temperature increases more rapidly and to greater magnitude, osmolality restoration after rehydration is slower, and thirst is less, while average total body sweat rates and chest sweat rates were not significantly different than in younger men [PMID 2589532].⁷²

C2.3.11. Race

Dark-colored clothing absorbs heat more readily than light-colored clothing [PMID 4068337].⁷³ However, the difference in heat tolerance in the sun by individuals of different skin colors has not been found to be significant [PMID 6736576].⁷⁴

C2.3.12. Acclimatization

Acclimatization is the process of physiologic adaptation to heat stress conditions. (Note: some sources equate this to “acclimation” [Stedman’s],⁷⁵ whereas others distinguish between “acclimation,” or short-term adaptation in laboratory conditions, and “acclimatization,” or longer-term adaptation to heat in field conditions [PMID 11252069].)⁷⁶ 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],⁷⁷ plasma volume increases [PMID 9694425],⁷⁸ and during exercise in the heat there is lowered heart rate and lowered rectal temperature [PMID 9694427];⁷⁹ there is also a decrease in perceived exertion as well as increased plasma volume [PMID 1763248].⁸⁰ Exertional heat stress was found to cause decreased cognitive performance in soldiers, but not in soldiers who had been heat-acclimated [PMID 17357764].⁸¹

Mild exercise in severe heat conditions induces significant hyperkalemia. The level of hyperkalemia is attenuated after acclimatization [PMID 880177].⁸² Men exercising in the heat have an increased ability to conserve sodium after acclimatization [PMID 880177].⁸³ Muscle glycogenolysis is unaffected by acclimation during exercise in the heat [PMID 8175568].⁸⁴ Likewise, the rate of gastric emptying does not appear to increase with heat acclimation [PMID 2920721].⁸⁵

Acclimatization is accomplished by exposing individuals to heat stress over a period of days or weeks. Exposure to heat for one hour or less, even with exercise, does not accomplish the acclimatization possible with longer daily exposures [[PMID 5853955](#)].⁸⁶ Exposure to dry heat increases sweat rate; exposure to humid heat stress results in a marked increase in sweat rate [[PMID 1763248](#)].⁸⁷ Thus, acclimatization results in increased water requirements. “TOUGHENING UP” PERSONNEL TO REQUIRE LESS WATER IN HEAT STRESS CONDITIONS IS NOT POSSIBLE. Likewise, the idea that highly motivated individuals can tolerate more heat stress exposure is also unjustified. In fact, they 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](#)].⁸⁸

Daily heat exposure is the most effective acclimation (i.e., laboratory chamber controlled exposures to heat) strategy [[PMID 11318020](#)].⁸⁹ Intermittent heat exposure causes only minimal heat adaptation [[PMID 11318020](#)].⁹⁰ Although 50% improvement in heat tolerance can be derived from 8-11 weeks of training under temperate conditions (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](#)].⁹¹ Full acclimatization may take several weeks, but two thirds or more of the adaptation is obtained within 5 days [[PMID 9694427](#)].⁹² Optimal acclimatization can be accomplished in 9 days [[PMID 5853955](#)],⁹³ although some changes occurring during acclimatization (for example, plasma volume initial expands and then contracts) continue for up to 3 weeks [[PMID 1763248](#)].⁹⁴ Even after 10 days of acclimatization, individuals may have difficulty performing tasks requiring the acquisition of new behaviors [[PMID 6626079](#)].⁹⁵ Prolonging heat acclimatization from 6 to 12 days did not reduce the physiological strain and limitation of heat-exercise tolerance imposed by wearing NBC protective clothing [[PMID 7588688](#)].⁹⁶ However, when wearing normal combat clothing, acclimation responses were greater after 12 than after 6 days of heat acclimation [[PMID 7588688](#)].⁹⁷ 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](#)],⁹⁸ [[PMID 8039520](#)].⁹⁹

Heat acclimatization occurs more rapidly in persons with greater cardiopulmonary fitness [[PMID 1763248](#)].¹⁰⁰ Active women may acclimatize to heat at a faster rate or to a greater extent than active men [[PMID 7085415](#)].¹⁰¹

Physical conditioning is also advantageous in the body’s response to dehydration, a heat stress-related condition. In one study, physical conditioning was associated with enhanced work performance during dehydration [[PMID 11055570](#)].¹⁰²

Inactivity results in decreased acclimatization after only a few days or weeks [[PMID 1763248](#)].¹⁰³ Exposing heat-acclimatized individuals regularly to cold temperatures (e.g., 4 hours daily for 21 days) can cause a significant loss in heat acclimatization [[PMID 4004678](#)].¹⁰⁴ A single exercise and/or heat exposure per week was not different from complete cessation of endurance exercise in the heat with regard to loss of acclimatization-related changes in plasma volume [[PMID 3699011](#)].¹⁰⁵ Acclimatization-related changes in sweat gland function may be attenuated by increases in central dopaminergic activity [[PMID 3397041](#)].¹⁰⁶

Physiologic Parameters	Percent Achievement On Acclimatization			
	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

From U.S. Navy research in 1976, contained in previous edition of [NAVMED P-5010](#). Manual of Naval Preventive Medicine: Chapter 3: Ventilation and Thermal Stress Ashore and Afloat: Section III. Physiological Principles 3-9. Effects of Heat.

Table 1 - Percent Optimum Heat Acclimatization

C2.3.13. Hydration Status

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

C2.3.13.1. Eu-, Hypo-, De-, and Hyper-hydration

C2.3.13.1.1. Euhydration

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

C2.3.13.1.2. 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 by reducing sweating rate and skin blood flow responses for a given core temperature [[PMID 11282312](#)].¹⁰⁷

C2.3.13.1.3. 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 or 0.1°C for every percent of body weight dehydration [[PMID 3569240](#)]).¹⁰⁹

C2.3.13.1.4. Hyperhydration

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

C2.3.13.2. 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](#)]¹¹⁰ [[PMID 10091501](#)].¹¹¹ This has led to a maximum fluid intake recommendation of 1.5 liters/hour. Note: 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.

C2.3.13.3. Hydration Status and Performance

Aerobic exercise tasks are likely to be adversely affected by heat stress and hypohydration [[PMID 9694412](#)].¹¹² Intermittent sprinting performance is poorer in a hypohydrated compared with a euhydrated state [[PMID10331887](#)].¹¹³ 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](#)].¹¹⁴ Rectal temperature rise was found to be significantly greater and exercise tolerance time significantly decreased in hypohydrated subjects exercising in the heat [[PMID 9459534](#)].¹¹⁵ Esophageal temperatures likewise rise more in hypohydrated subjects exercising in heat stress conditions [[PMID 2361893](#)].¹¹⁶ Experiments have documented a lowering of cardiac stroke volume with dehydration [[PMID 10666060](#)].¹¹⁷

The rate of fluid loss may exceed the capacity of the gastrointestinal tract to assimilate fluids [[PMID 11547892](#)].¹¹⁸ Gastric emptying may be below the rate of fluid loss, and training to drink during exercise has been recommended by at least one author as a way to enhance tolerance of large amounts of oral rehydration liquids during exercise in heat stress conditions [[PMID 11547892](#)].¹¹⁹ Greater severity of heat strain is associated with lower rates of gastric emptying [[PMID 2920721](#)].¹²⁰

Hyperhydration provides no advantages over euhydration regarding thermoregulation and exercise performance in the heat [[PMID 11282312](#)].¹²¹ Over-hydrating or meeting fluid needs during very long-lasting exercise in the heat with low or negligible sodium intake can result in reduced performance and, not infrequently, hyponatraemia [[PMID 11547892](#)].¹²²

C2.3.14. Medications, Including Prescription and Non-Prescription Medications, Drugs of Abuse, Folk Remedies, and Dietary 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](#)].¹²³ 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](#)])¹²⁴ and should be avoided in such conditions [[PMID 10050577](#)].¹²⁵ Ephedrine alkaloids (amphetamine-like compounds derived from various species of herbs of the genus ephedra, also referred to as Ma Huang, Ma-huang, or Ephedra equisetina) and creatine may contribute to subclinical dehydration and heatstroke in selected individuals [[PMID 12182766](#)].¹²⁶

Cocaine can cause 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](#)].¹²⁷ Cocaine has been associated with heat stroke in the U.S. Navy.¹²⁸

Bromocriptine has been found to attenuate sweat gland function responses to acclimatization [[PMID 3397041](#)].¹²⁹ Antihistamines and other anti-cholinergic drugs (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.

Antihypertensive medications may affect heat stress tolerance. Nonselective beta-blockers may increase predisposition to exertional hyperthermia [[PMID 1679517](#)].¹³⁰ However, one study demonstrated that although forearm blood flow was diminished by beta-blockers, rectal temperature was not significantly increased, possibly due to increased sweating from beta-blockade [[PMID 2820920](#)].¹³¹ Diuretics may cause electrolyte (including sodium) loss. Diuretics and laxatives can cause water loss, leading to dehydration.

Lithium may cause water loss (through diabetes insipidus) [[PDR.net](#)]¹³² [[PMID 11246113](#)].¹³³

C2.3.15. Alcohol

(See the section on the effect of [Alcoholic Beverages](#) on heat stress response.)

C2.3.16. 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](#)]).¹³⁴ Unfortunately, activity level is not always totally voluntary (for example, 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 [[Time](#)].¹³⁵

Doing work in heat stress can cause core body temperature elevations. Intermittent rest times with adequate replacement fluid availability usually can prevent temperatures from becoming elevated. However, extremely high temperatures, heavy gear or clothing (such as MOPP gear), or high work 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 higher 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 10414066](#)].¹³⁶ Work-rest cycle times and fluid replacement requirement charts have been developed for work in heat stress in humid and dry climates. The following is adapted from work done on laboratory acclimated subjects [[PMID 10414066](#)].¹³⁷

WBGT Index (°F)	Easy Work (250 W)		Moderate Work (425 W)		Hard Work (600 W)	
	Work-Rest (minutes)	Water Intake (quarts/hour)	Work-Rest (minutes)	Water Intake (quarts/hour)	Work-Rest (minutes)	Water Intake (quarts/hour)
78-81.9	Unlimited	0.5	Unlimited	0.75	40/20	0.75
82-84.9	Unlimited	0.5	50/10	0.75	30/30	1.0

85-87.9	Unlimited	0.75	40/20	0.75	30/30	1.0
88-89.9	Unlimited	0.75	30/30	0.75	20/40	1.0
90+	50/10	1.0	20/40	1.0	10/50	1.0

Table 2 - Work-Rest Cycles and Fluid Replacement for 4 Hour Periods

MOPP gear adds 10° F to the WBGT Index for Easy Work and adds 20° F to the WBGT Index for Moderate and Hard Work; body armor adds 5° F to the WBGT Index

Rest = sitting or standing, in shade if possible.

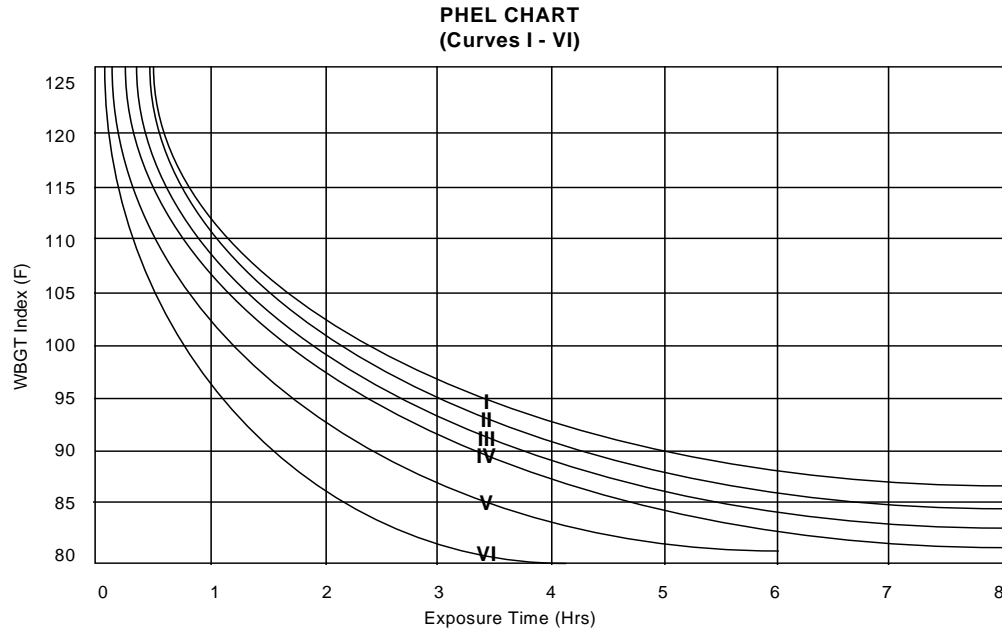
Individual water needs vary by 0.25 quarts/hour.

Fluid intake should not exceed 1.5 quarts per hour; daily fluid intake generally should not exceed 12 quarts (note: this is not to suggest limiting fluid intake by highly conditioned persons, who may require greater than 12 quarts daily).

C2.3.16.1. PHEL Curves

Physiological Heat Exposure Limits (PHEL curves) identify the maximal allowable exposure time or “stay time” for all U.S. Navy shipboard personnel when working in the heat ([OPNAVINST 5100.19 series Appendix B2-A](#)).¹³⁸ Six categories of heat stress (PHEL curves I-VI, shown in [Figure 2](#)) with different exposure times are used to protect against heat stress injuries. The correct PHEL curve is determined by considering the WBGT index (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 - PHEL Curves



Non-routine operations, such as performing operations in out-of-normal plant configurations, increases in normal watchstander 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 [Table 3](#).

WBGT Index (F)	PHEL Curves											
	Total Exposure Time in Hours:Minutes											
	Without the presence of fuel vapors or combustion gases						With the presence of fuel vapors or combustion gases					
	I	II	III	IV	V	VI	I	II	III	IV	V	VI
80.0	>8:00	>8:00	>8:00	8:00	6:35	4:30	4:50	4:15	3:30	2:55	2:15	1:30
81.0	>8:00	>8:00	>8:00	7:45	6:00	4:05	4:25	3:50	3:10	2:40	2:00	1:20
82.0	>8:00	>8:00	8:00	7:05	5:25	3:40	4:00	3:30	2:55	2:25	1:50	1:15
83.0	>8:00	8:00	7:45	6:25	4:55	3:20	3:40	3:10	2:40	2:10	1:40	1:10
84.0	>8:00	8:00	7:05	5:55	4:30	3:05	3:20	2:55	2:25	2:00	1:30	1:00
85.0	8:00	7:45	6:30	5:20	4:05	2:50	3:00	2:40	2:10	1:50	1:25	0:55
86.0	8:00	7:05	5:55	4:55	3:45	2:35	2:45	2:25	2:00	1:40	1:15	0:50
87.0	7:25	6:30	5:25	4:30	3:25	2:20	2:30	2:10	1:50	1:30	1:10	0:45
88.0	6:45	5:55	4:55	4:05	3:10	2:10	2:20	2:00	1:40	1:25	1:05	0:40
89.0	6:10	5:25	4:30	3:45	2:50	2:00	2:05	1:50	1:30	1:15	1:00	0:40
90.0	5:40	5:00	4:10	3:25	2:40	1:50	1:55	1:40	1:25	1:10	0:55	0:35
91.0	5:15	4:35	3:50	3:10	2:25	1:40	1:45	1:30	1:15	1:05	0:50	0:30
92.0	4:50	4:10	3:30	2:55	2:15	1:30	1:35	1:25	1:10	1:00	0:45	0:30
93.0	4:25	3:50	3:15	2:40	2:00	1:25	1:30	1:20	1:05	0:55	0:40	0:25
94.0	4:05	3:35	3:00	2:25	1:50	1:15	1:20	1:10	1:00	0:50	0:35	0:25

WBGT Index (F)	PHEL Curves											
	Total Exposure Time in Hours:Minutes											
	Without the presence of fuel vapors or combustion gases						With the presence of fuel vapors or combustion gases					
	I	II	III	IV	V	VI	I	II	III	IV	V	VI
95.0	3:45	3:15	2:45	2:15	1:45	1:10	1:15	1:05	0:55	0:45	0:35	0:20
96.0	3:25	3:00	2:30	2:05	1:35	1:05	1:10	1:00	0:50	0:40	0:30	0:20
97.0	3:10	2:45	2:20	1:55	1:25	1:00	1:10	0:55	0:45	0:40	0:30	0:20
98.0	2:55	2:35	2:10	1:45	1:20	0:55	1:05	0:50	0:40	0:35	0:25	0:15
99.0	2:40	2:20	2:00	1:40	1:15	0:50	0:55	0:45	0:40	0:30	0:25	0:15
100.0	2:30	2:10	1:50	1:30	1:10	0:45	0:50	0:45	0:35	0:30	0:20	0:15
101.0	2:20	2:00	1:40	1:25	1:05	0:45	0:45	0:40	0:35	0:25	0:20	0:15
102.0	2:10	1:50	1:35	1:15	1:00	0:40	0:40	0:35	0:30	0:25	0:20	0:10
103.0	2:00	1:45	1:25	1:10	0:55	0:35	0:40	0:35	0:30	0:25	0:15	0:10
104.0	1:50	1:35	1:20	1:05	0:50	0:35	0:35	0:30	0:25	0:20	0:15	0:10
105.0	1:40	1:30	1:15	1:00	0:45	0:30	0:35	0:30	0:25	0:20	0:15	0:10
106.0	1:35	1:25	1:10	0:55	0:45	0:30	0:30	0:25	0:20	0:20	0:15	0:10
107.0	1:30	1:15	1:05	0:50	0:40	0:25	0:30	0:25	0:20	0:15	0:10	0:10
108.0	1:20	1:10	1:00	0:50	0:35	0:25	0:25	0:25	0:20	0:15	0:10	0:05
109.0	1:15	1:05	0:55	0:45	0:35	0:25	0:25	0:20	0:15	0:15	0:10	0:05
110.0	1:10	1:00	0:50	0:40	0:30	0:20	0:25	0:20	0:15	0:15	0:10	0:05
111.0	1:05	1:00	0:50	0:40	0:30	0:20	0:20	0:20	0:15	0:10	0:10	0:05
112.0	1:00	0:55	0:45	0:35	0:25	0:20	0:20	0:15	0:15	0:10	0:10	0:05
113.0	0:55	0:50	0:40	0:35	0:25	0:15	0:20	0:15	0:15	0:10	0:05	0:05
114.0	0:55	0:45	0:40	0:30	0:25	0:15	0:15	0:15	0:10	0:10	0:05	0:05
115.0	0:50	0:45	0:35	0:30	0:20	0:15	0:15	0:15	0:10	0:10	0:05	0:05
116.0	0:45	0:40	0:35	0:25	0:20	0:15	0:15	0:10	0:10	0:10	0:05	0:05
117.0	0:45	0:40	0:30	0:25	0:20	0:10	0:15	0:10	0:10	0:05	0:05	0:05
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 3 - Safe Exposure Times Aboard Ship

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

$$RSST = [(1 - (Et - R/2) / At1)] \times At2$$

Where:

RSST = remaining safe stay time (in minutes)

Et = elapsed time on station (in minutes)

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

At1 = 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].¹³⁹ It is to be noted that PHEL curves apply only to shipboard use (see [OPNAVINST 5100.19 series](#)).

C2.3.16.2. Flag Conditions and Activity Limitations

The Navy uses a set of flags to indicate when certain heat stress hazards exist ([Table 4](#)). These are based on the Marine “Heat Condition Flag Warning System” (Enclosure 4 of Marine Corps Order 6200.1E).¹⁴⁰

WBGT Index	Activity Level Hazards and Limitations	Flag
Less than 80	Extremely intense physical exertion may precipitate heat exhaustion or heat stroke.	White
80 - 84.9	Discretion is required in planning heavy exercise for unacclimatized personnel.	Green
85 - 87.9	Curtail strenuous exercise and activity for unacclimatized personnel during the first 3 weeks of heat exposure. Avoid classes in the sun.	Yellow (Amber)
88 - 89.9	Strenuous exercise must be curtailed for all personnel with less than 12 weeks training in hot weather.	Red
90 or above	Physical training and strenuous exercise must be suspended for all personnel (excludes operational commitment not for training purposes).	Black

Table 4 - Flag Conditions and WBGT

C2.3.16.3. Screening criteria for Heat Stress Exposure Ashore

While the flag system communicates heat stress conditions in a local geographic area, it does not identify job-specific or work area-specific heat stress conditions. For example, WBGT may be 75° F (23.9° C) outdoors at a Navy installation (white flag conditions), but the laundry workers could be working indoors with a WBGT of 88° F (31.1° C). The following table is adapted from the ACGIH 2005 TLVs and BEIs [ACGIH].¹⁴¹

Work Demands	Acclimatized				Unacclimatized			
	Light	Moderate	Heavy	Very Heavy	Light	Moderate	Heavy	Very Heavy
100% Work	85.1	81.5	78.8		81.5	77	72.5	
75% Work 25% Rest	86.9	83.3	81.5		84.2	79.7	76.1	
50% Work	88.7	85.1	83.3	81.5	86	82.4	79.7	77

50% Rest								
25% Work 75% Rest	90.5	87.8	86	85.1	87.8	84.2	82.4	79.7

Table 5 - Screening Criteria for Heat Stress Exposure (ACGIH) based on WBGT

For woven cloth overalls add 6.3° F (3.5° C) to the WBGT. For double-cloth overalls add 9° F (5° C) to the WBGT.

C2.3.16.4. Muscle Performance in Heat Stress conditions

Intermittent supramaximal running performance in the heat is reduced, which is not caused by greater muscle glycogenolysis or lactate accumulation [[PMID10331887](#)].¹⁴² Heat stress reduces maximum metabolic rate (VO₂max) [[PMID 4039255](#)],¹⁴³ [[PMID 8175568](#)].¹⁴⁴ Acclimatization and aerobic fitness increase VO₂max, but a reduction in VO₂max is still caused by heat stress [[PMID 4039255](#)].¹⁴⁵

C2.3.16.5. Sleep Deprivation and Heat Tolerance

Sleep loss reduces heat tolerance [[PMID 1763248](#)].¹⁴⁶

C2.3.17. Mean Metabolic Rate

The mean metabolic rate is a value referred to in some scientific reports related to heat stress. It is generally restricted to use in research.

C2.3.18. Attributes of Surrounding Materials

In one study, skin temperature and moisture were affected by the material or construction of furniture coverings, although test subjects attributed personal thermal comfort more to room climate than to material properties of furniture coverings [[PMID 7148203](#)].¹⁴⁷ Cotton continues to be the preferred clothing material during hot weather. Special evaporative polyester fabric clothing compared to cotton clothing does not alter physiological, thermoregulatory, or comfort sensation responses during exercise in a moderately warm environmental condition [[PMID 11740309](#)].¹⁴⁸

C2.3.19. 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, or 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](#)].¹⁴⁹

C2.3.20. Electric Blankets

Cases of heat stroke (including two fatal cases) in users of electric blankets have been reported; first and second degree burns were noted in the victims [[PMID 17133030](#)].¹⁵⁰

C2.4. Heat Stress Vs. Heat Strain

C2.4.1. Heat Stress

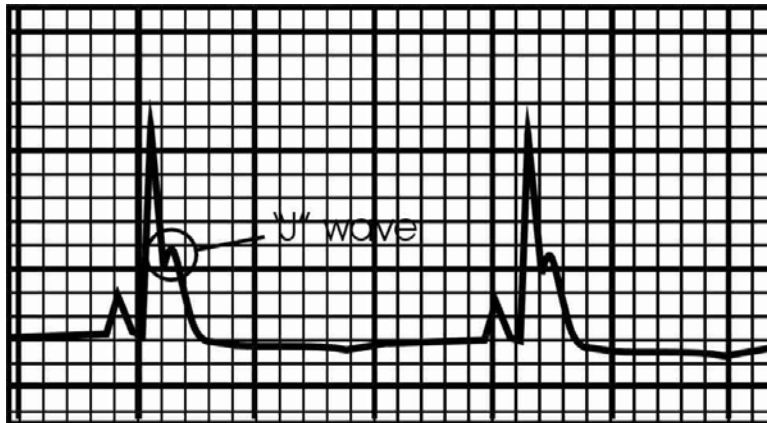
Heat stress refers to the combination of all of those factors which result in heat gain (or loss) to the body. Heat stress is the force or load acting upon the body. At a cellular level, cells produce heat-shock (stress) proteins, increased levels of which induce transient tolerance to a second heat stress [[PMID 12075060](#)].¹⁵¹ Heat-shock protein production may play a role in acclimatization [[PMID 9375300](#)].¹⁵² At the tissue level, there is an acute-phase response to heat stress that protects against tissue injury and promotes repair [[PMID 12075060](#)].¹⁵³ 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](#)]¹⁵⁴ [[PMID 11219501](#)].¹⁵⁵

C2.4.2. Heat Strain

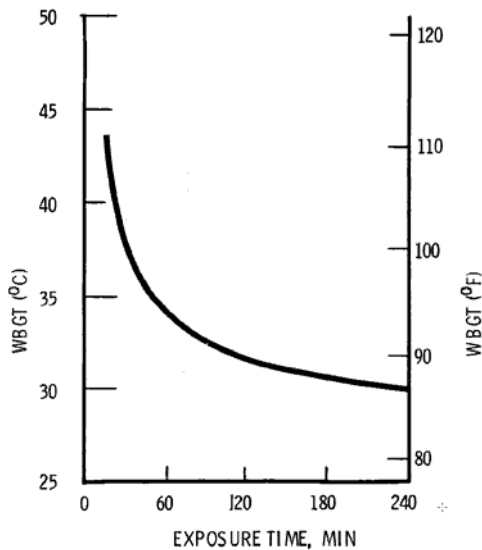
Heat strain is the resulting abnormality or “distortion” of the body’s physiology when exposed to more heat stress than the body is prepared to compensate for at that time. The extreme result is the failure of the body to cool itself (thermoregulation failure), and core temperature rises (often precipitously).

Heat increases myocardial oxygen demand. Electrocardiogram (EKG) changes may include increased J-point displacement (J wave, also seen in hypothermia [Wagner]),¹⁵⁶ S-T segment flattening (0.08 s), and prolongation of the Q-T interval with reduction in T-wave amplitude [[PMID 7362568](#)].¹⁵⁷

Figure 3 - J Wave Appearance on Electrocardiogram



Mental performance is also affected by the heat. The following graph illustrates the impact on mental performance of heat stress exposure. [HEW].¹⁵⁸

Figure 4- Upper Limits of Exposure for Unimpaired Mental Performance

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](#)],¹⁵⁹ [[PMID 16916922](#)].¹⁶⁰

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](#)].¹⁶¹

The recommended threshold WBGT Index value for instituting hot weather practices is 75° F or 23° C [[CHPPM](#)].¹⁶²

C2.5. Prevention Of Heat Stress Injuries

Heat stress injuries and illnesses are preventable threats to health. In the United States, annual heat-related deaths between 1979 and 1996 ranged from 148 to 1,700 [[MMWR](#)].¹⁶³ The U.S. Army had 196 hospitalizations due to heat illness or injury (“excessive heat”) in 1994, with a rate of 0.36 per thousand per year, while the U.S. Air Force had 19 such hospitalizations [[AMSUS](#)].¹⁶⁴ From 1981 to 1991, an average of 135 British servicemen and women were hospitalized yearly for heat-related conditions [[PMID 8904496](#)].¹⁶⁵

C2.5.1. 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 [Drinks, above](#)), light clothing, and appropriate exercise or work limitations for the level of heat stress and PPE used (see [PHEL Curves, above](#)).

C2.5.2. 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.

C2.5.3. 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](#)].¹⁶⁶

INDIVIDUAL FACTORS	ENVIRONMENTAL FACTORS
Advanced age Alcohol use Antihistamine use Bromocriptine use Cocaine use Heavy clothing, including MOPP gear 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 Young age (<5 years [PMID 2371104]) ¹⁶⁷	High temperature (>85° F) High humidity Midday Hot large objects nearby (close enough to provide significant radiant heat) No air movement (wind speed = 0) Deep mines

Table 6 - Heat Stress Injuries—Risk Factors and Predisposing Conditions

C2.6. Temperature Measurement And Thermometers

C2.6.1. Skin Temperature

Skin temperature is affected by dermal blood flow and environmental conditions, and is a poor indicator of body core temperature. Skin temperature is unreliable in the diagnosis of heat stress injury severity [[PMID 2805574](#)].¹⁶⁸

C2.6.2. Expired Breath

Expired breath temperature does not correlate with body core temperature, even after holding a breath for 30 seconds [[PMID 11007321](#)].¹⁶⁹

C2.6.3. Oral Thermometers

Oral thermometers are popular and give a reasonable approximation of body temperature (usually reading lower than core temperature) in many circumstances. Recent drinking of hot (within 5 minutes [[PMID 11606822](#)])¹⁷⁰ or cool (within 30 minutes [[PMID 11606822](#)],¹⁷¹ [[PMID](#)

[3722670](#))¹⁷² 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.

C2.6.4. Tympanic (Ear) Electronic Thermometers

Tympanic (ear) electronic thermometers can significantly underestimate core temperatures. Readings may be affected by external conditions (such as facial fanning [[PMID 9116786](#)]),¹⁷³ liquids in the ear canal (e.g., cold water used in cooling), and improper technique. Thus, tympanic thermometers do not reliably measure core body temperature and their use for this purpose is not recommended. The use of fiber optic devices may overcome the affect of some of those factors [[PMID 9729565](#)].¹⁷⁴

C2.6.5. Rectal Temperature

Rectal temperatures generally are a good reflection of core body temperature. However, with aggressive cooling techniques, readings may lag behind actual core body temperature [[PMID 1608386](#)].¹⁷⁵ Thus, if used during aggressive treatment of heat injuries, caution must be exercised not to over-treat (over-cool).

C2.6.6. Esophageal Thermometers

Esophageal thermometers give accurate core body temperature measurements. They are more difficult to use than oral, rectal, or ear thermometers, and may not be readily available. Readings in patients with tracheal intubation may be affected by placement of the thermometer and temperature of inspired air, especially in children [[PMID 8478651](#)].¹⁷⁶ Esophageal temperatures may fluctuate rapidly as a result of subjects swallowing liquids or saliva [[PMID 6853278](#)],¹⁷⁷ [[PMID 5060664](#)].¹⁷⁸

C2.6.7. Bladder Temperature

Bladder temperatures can accurately indicate core body temperature during anesthesia without the lag during rewarming noted with rectal temperatures [[PMID 9382206](#)].¹⁷⁹ The use of bladder temperatures may be restricted to advanced hospital settings under limited conditions.

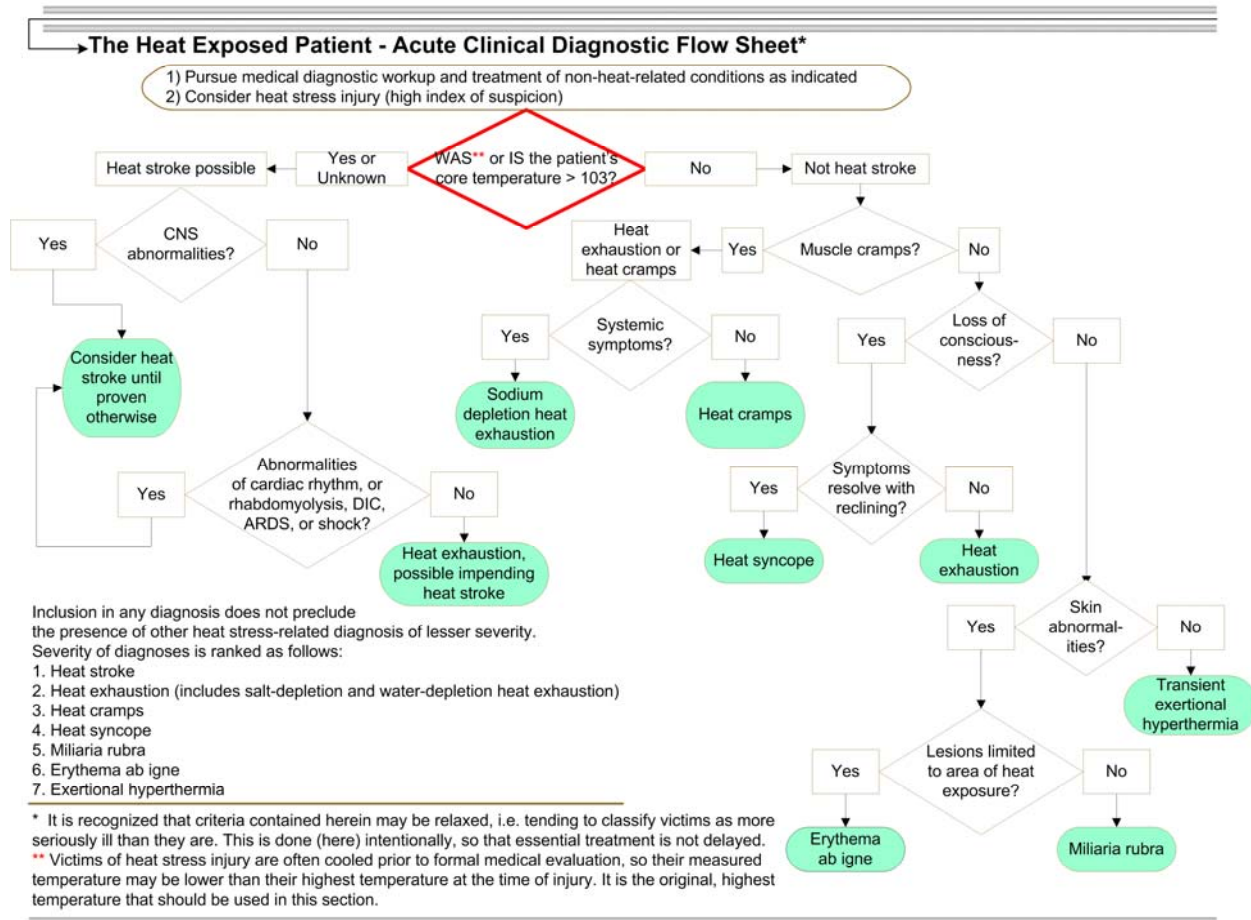
Chapter 3:

C3. 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 obviously high-risk settings. However, hot workspaces, inadequate fluid replacement, or the impact of protective equipment may be less obvious to health care workers. Military cases of heat stroke, although more common in summer, have occurred in the coldest part of the year [[PMID 8904496](#)].¹⁸⁰ 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. Specific symptoms, signs, and findings noted below will give further guidance in the recognition of heat injuries and illnesses. An algorithm ([Figure 5](#)) is available to assist in the diagnosis of heat stress injury.

It is to be noted that heat stress injuries represent a continuum rather than discreet injury categories. Also, 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.

Figure 5 - Acute Heat Exposed Patient Diagnostic Flow Chart



Four major types heat stress injuries are commonly recognized: heat rash, heat cramps, heat exhaustion, and heat stroke. Some authors elaborate and include more heat exposure-related conditions. An attempt is made here to include most such conditions.

Certain underlying conditions have specific symptoms that may be precipitated by exposure to heat stress. Migraine headaches may be precipitated by heat (or cold) stress exposure [PMID 6935858].¹⁸¹ Intermittent hyperthyreosis is a condition in which hyperthyroid patients during heat stress exposure experience various symptoms (insomnia, irritability, headache, tension, tachycardia, palpitations, precordial pain, dyspnea, flushes with sweating or chills, tremor, abdominal pain or diarrhea, polyuria, weight loss in spite of ravenous appetite, fatigue, exhaustion, depression, weakness, lack of concentration and confusion) [PMID 52584]¹⁸² [PMID 6935858].¹⁸³ Therapy is medical and related to treating the thyroid condition rather than oriented at heat stress exposure control. Although migraines and intermittent hyperthyreosis may be precipitated by heat stress exposure, they are not considered true “heat illnesses,” as they are primarily unrelated (to heat) underlying conditions.

C3.1.1. 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; wildfire rash [Steadman's].¹⁸⁴ It is a cutaneous reaction to heat stress exposure, with redness and inflammatory skin reaction. It consists of papules and vesicles at sweat glands ([photo](#)). Symptoms include pruritus and a burning sensation, as well as heat intolerance [[PMID 10994246](#)].¹⁸⁵

Treatment is removal from heat stress exposure. Application of cool wet cloths may alleviate symptoms. Cool starch baths, calamine lotion, corticosteroid lotion [Noojin],¹⁸⁶ sometimes with 0.25% menthol added [[Merck](#)],¹⁸⁷ may also be tried if necessary. Oral antihistamines to control itching have been suggested [Noojin],¹⁸⁸ but this should be limited to use for severe itching in air conditioning, as caution must be used in hot environments as antihistamines may inhibit sweating.

Miliaria can impair sweating and reduce heat tolerance. One study found that heat intolerance due to heat rash was not resolved until after 21 days [[PMID 7435594](#)].¹⁸⁹ 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.

C3.1.2. 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](#)],¹⁹⁰ including chronic exposure to heat from fires, chairs with built-in heaters, car heaters [[PMID 9040977](#)],¹⁹¹ hot water bottles, infrared lamps, and heating pads [[PMID 7845500](#)].¹⁹² Central heating and not using open fires has largely reduced the incidence of erythema ab igne [[PMID 7999279](#)],¹⁹³ [[PMID 7845500](#)].¹⁹⁴ Occupational exposures causing this condition have included cooking [[PMID 1828060](#)]¹⁹⁵ and baking [[PMID 8772030](#)].¹⁹⁶ While it has been noted to be a marker for underlying disease, including cancer [[PMID 4067962](#)],¹⁹⁷ some authors have proposed a pathogenic role for erythema ab igne [[PMID 2685415](#)]¹⁹⁸ ([photo](#)). Treatment is removal from the heat source.

C3.1.3. Exertional Hyperthermia

Exertional hyperthermia refers to a significant (for example, greater than 0.9° F or 0.5° C) rise in body temperature that occurs with exercise. It is not a diagnosis, but rather is used (usually by researchers) to describe a physiologic response to exercise in heat stress conditions [[PMID 10484593](#)].¹⁹⁹

C3.1.4. 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](#)].²⁰⁰ A common scenario is when personnel are required to stand at attention or in formation in the heat. It is syncope from vascular insufficiency (hypotension). Hypovolemia (due to diuretics or medications decreasing vascular tone) increases risk of heat syncope [Nadel].²⁰¹

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 as for fainting: supine position, elevation of the feet, and oral fluids (those containing sodium may be preferable). Recovery is expected to be prompt and complete.

C3.1.5. Heat Edema

Heat edema is dependent (lower extremity) edema that develops or worsens soon after heat stress exposure (usually within 48 hours). It is probably hormonally mediated [Nadel].²⁰² Victims have swelling or increased pre-existent edema of the lower extremities. No specific treatment is required, as the condition is expected to resolve as the acclimatization process continues. In cases of excessive edema due to underlying disease, some authors recommend removing the victim from further heat stress exposure, rather than giving diuretics to enable them to complete heat acclimatization [Nadel].²⁰³

C3.1.6. 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]²⁰⁴ [[PMID 2178579](#)].²⁰⁵ 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 (pO₂). Treatment is temporary removal from heat stress. Some authors recommend that workload be decreased before resuming acclimatization [Nadel].²⁰⁶

C3.1.7. 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](#)].²⁰⁷ Continuous strenuous exercise in the heat, such as a several-day sports tournament or building project, may eventually lead to decreased sodium and favor development of heat cramps. Symptoms are cramps in the affected muscles (generally those muscles that have been exercised), and other than the fatigue associated with the exercise, are not accompanied by constitutional complaints. Treatment for heat cramps is increased sodium intake, which has eliminated heat cramps even in highly competitive sport competition [[PMID 8653105](#)].²⁰⁸

C3.1.8. Heat Exhaustion

Heat exhaustion has been defined both as simply “the inability to continue exercise in the heat” [[PMID 1763248](#)]²⁰⁹ (which could be taken to imply no distinct medical illness) and “a form of reaction to heat, marked by prostration, weakness, and collapse, resulting from severe dehydration” [Stedman’s]²¹⁰ (implying a distinct medical illness or injury). It is treated here as a distinct medical illness or injury.

C3.1.8.1. Symptoms of Heat Exhaustion

Heat exhaustion is characterized by dizziness, weakness, or fatigue often following several days of sustained exposure to hot temperatures, and results from dehydration or electrolyte imbalance [[MMWR](#)].²¹¹ Heat exhaustion is a serious heat injury, and may be classified according to the underlying physiologic mechanism as sodium depletion (ICD-9-CM code 992.4, “heat exhaustion due to salt depletion”) or water deficient (ICD-9-CM code 992.3, “heat exhaustion, anhydrotic”) [[WHO](#)].²¹²

C3.1.8.2. Sodium-depletion Heat Exhaustion

Sodium-depletion heat exhaustion occurs when individuals exposed to excessive heat stress consume sufficient water but insufficient salt. Hyponatremia (low sodium levels, serum sodium less than 130 mEq/L [[PMID 10530529](#)])²¹³ 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. Symptoms include nausea, vomiting, diarrhea, headache, dizziness, weakness, alterations of mental status, and minimal or no thirst. Seizures were noted in 31% of victims in a study involving Army trainees [[PMID 11370203](#)].²¹⁴ The victim usually has cool, moist skin that may be sticky and pale, and often is hypotensive and tachycardic with normal (or even low) body temperature and normal urine volume. Hyponatremia always is present. The existence of hyponatremia is suggested by altered mental status or by seizures without hyperpyrexia or hypoglycemia during prolonged exercise in the heat [[PMID 10530529](#)].²¹⁵

Treatment of sodium-depletion heat exhaustion is removal of the victim to a cool place and administration of sodium replacement (oral or IV fluids of normal or high tonicity, such as NS or hypertonic saline) until symptoms clear and pulse and urine findings normalize (including urine sodium content of at least 10 mEq/L). Although 1 to 2 liters of saline solution is usually sufficient, up to 4 liters of IV fluids may be required [Schmidt].²¹⁶ Unless more fluid is needed to maintain blood pressure, current clinical recommendation is to limit IV saline administered in the field (i.e., pre-hospital) to 2 liters to avoid pulmonary edema [Noltkamper].²¹⁷ Heavy clothing should be removed from patients and they should be allowed to rest in a shaded and ventilated space, while active cooling is initiated (see [Cooling](#), below). Heat exhaustion victims should improve rapidly with shaded rest, cooling, and replacement of sodium. If sodium has been very low (less than 120 mEq/L) for more than 48 hours, sodium replacement may lead to cerebral edema if done too rapidly. In such cases, sodium replacement should be monitored [[PMID 12074531](#)]²¹⁸ or paced [Nadel].²¹⁹ In hyponatremia of shorter duration, sodium replacement can be more rapid. Avoidance of aspirin and other non-steroidal anti-inflammatory agents has been recommended to prevent possible paradoxical hyperthermia [Nadel].²²⁰

C3.1.8.3. 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 ability to tolerate heat, with virtually no ability to exercise in severe dehydration (loss of body water of 7% of body weight) [[PMID 1763248](#)].²²¹ Symptoms include malaise, vomiting, confusion [[PMID 12074531](#)],²²² anxiety, weakness, agitation, and even delirium [Nadel].²²³ Victims are dehydrated, oliguric, hyperthermic (temperature of 100.4° F or 38° C or greater [[PMID 8775579](#)]),²²⁴ and may exhibit hyperventilation and tetany. Hypernatremia is characteristic. Heat stroke may be imminent with this heat stress injury.

Treatment is removal from heat (remove heavy clothing, move victim to a shaded, ventilated area, begin active cooling, see [Cooling](#), below) and emergent transport to a hospital for water replacement. IV fluids should be used to replace volume (primarily) and lower serum sodium (at 0.5 to 1 mEq/hour but no more than 10-15 mEq/24 hours, to avoid cerebral edema as a result of decreasing sodium too rapidly). Initially, NS is appropriate until tissue perfusion is restored, at which time 0.5NS or other hypotonic solution may be used. A target of 0.5 ml/kg hourly urine output has been recommended [Londer].²²⁵ Aggressive cooling should be instituted if necessary to maintain core temperature less than 102° F (38.9° C).

C3.1.9. Heat Stroke

Heat stroke is the most serious heat stress exposure-related illness (injury). Unfortunately, early or impending heat stroke may go unrecognized [[PMID 2910771](#)].²²⁶ It is defined as a seriously elevated temperature (> 104° F or 40° C [[PMID 12075060](#)])²²⁷ that causes CNS injury, caused by heat stress conditions beyond the compensatory (cooling) ability of the body. It is a life-threatening emergent medical condition. Without prompt treatment, victims will sustain permanent injury or death. Multiple organ dysfunction syndrome has been common in some case series [[PMID 10229168](#)].²²⁸

C3.1.9.1. Classic Heat Stroke

Classic heat stroke is the most common type of heat stroke [[MMWR](#)].²²⁹ It generally affects the elderly and those with underlying disease or other debilitating condition. 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].²³⁰ However, maximum daily temperature and humidity have not been found to be good predictors of the number of heat stroke injuries [[PMID 11235827](#)].²³¹

- previous heatstroke
- age (the young and the elderly)
- social circumstances (e.g., living alone, the urban poor)
- medical history and chronic health conditions (e.g., cardiovascular disease, respiratory diseases, schizophrenia with neuroleptic treatment [[PMID 661049](#)],²³² amyotrophic lateral sclerosis receiving nortriptyline, multiple sclerosis, attention deficit disorder, cystic fibrosis [[PMID 9167437](#)],²³³ hyperthyroidism [[PMID 11510526](#)])²³⁴
- other conditions that might interfere with the ability to care for oneself
- alcohol consumption (which may cause dehydration)
- physical activity (e.g., exertion in exceptionally hot environments during work or recreation—see [Exertional Heat Stroke](#), below), specifically indoor activity with malfunctioning air-conditioning [[PMID 9167437](#)]²³⁵
- use of medications that interfere with the body's heat regulatory system, such as neuroleptics (antipsychotics or major tranquilizers) and medications with anticholinergic effects (e.g., tricyclic antidepressants, antihistamines, some antiparkinsonian agents, and some over-the-counter sleeping pills) [[MMWR](#)].²³⁶ A patient who developed hyperpyrexia and heat stroke after taking overdoses of a monoamine oxidase inhibitor, benzodiazepines and a beta-adrenergic receptor blocking agent has been described [[PMID 550464](#)].²³⁷ Heat stroke has also been reported in a patient taking perphenazine and amitriptyline [[PMID 6842224](#)].²³⁸

Table 7 - Risk Factors for Classic Heat Stroke

C3.1.9.2. 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. According to a recent article, it is the third leading cause of death among American athletes [[PMID 12172074](#)].²³⁹ Increased heat

production from exercising skeletal muscles is a major contributing mechanism [Simon].²⁴⁰ Acute renal failure may occur in 25% of patients with exertional heat stroke [PMID 9351070].²⁴¹ While hot weather or surrounding conditions increase the heat stress on an individual, exertional heat stroke has been documented in physically conditioned military personnel at ordinarily comfortable temperatures, even below 75° F [PMID 2107465].²⁴²

- sleep loss
- generalized fatigue [PMID 2406545]²⁴³
- inappropriately heavy clothing
- exposure to direct sunlight
- dehydration
- lack of cardiovascular conditioning
- lack of acclimatization to heat [Simon]²⁴⁴
- 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])²⁴⁵

Table 8 - Predisposing Factors for Exertional Heat Stroke

Heat intolerance (inability to adapt to heat stress) occurs in a small percentage of prior heatstroke patients, and may be transient or persistent [PMID 2406545].²⁴⁶ Recovery from exertional heatstroke is idiosyncratic and may require up to 1 year in severe cases [PMID 2406545].²⁴⁷ In one study, no measured variable predicted recovery from exertional heatstroke, or heat acclimation responses [PMID 2406545].²⁴⁸

C3.1.9.3. 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. Victims occasionally have feelings of euphoria. Sweating may or may not be present. Heat stroke victims frequently have diarrhea and vomiting. In exertional heat stroke, metabolic acidosis is the predominant acid-base change, especially in victims presenting with higher temperatures [PMID 2692177],²⁴⁹ followed by respiratory alkalosis (a study that may have included both exertional and classic forms of heat stroke found similar results [PMID 11398693]).²⁵⁰ In classic heat stroke, reports are inconsistent between metabolic acidosis or respiratory alkalosis as the predominant finding [PMID 2355761],²⁵¹ [PMID 12075060].²⁵²

The hemodynamic changes in severe heat exposure reflect a hyperdynamic circulation with tachycardia and high cardiac output [PMID 10517377].²⁵³ In one study, relative hypovolemia was more pronounced in patients with heatstroke compared to patients with heat exhaustion; and, signs of peripheral vasoconstriction were more often present in patients with heatstroke, while patients with heat exhaustion more often demonstrated peripheral vasodilatation [PMID 10517377].²⁵⁴

Arrhythmias are common in heat stroke. One study of heat stroke patients found prolonged Q-T interval (61 percent), sinus tachycardia (43 percent), diffuse nonspecific ST-T

changes (26 percent), conduction defect (22 percent), and ST-T changes consistent with myocardial ischemia (21 percent) [[PMID 8339628](#)].²⁵⁵ Pericardial effusion has been reported in heat stroke [[PMID 1452370](#)],²⁵⁶ [[PMID 11496687](#)].²⁵⁷ When interpreting ST-T elevation in the ECG of a heat stroke patient, caution should be used so as to not misdiagnose it as an acute myocardial infarction [[PMID 16043949](#)].²⁵⁸

Rhabdomyolysis is common in severe heat injury, with extremely high serum and urine myoglobin and serum creatinine kinase concentrations (for example, 100 times normal levels). (It is to be noted that exertion without heat stress injury may cause creatinine kinase levels of nearly 5000 international units/liter [[PMID 9827835](#)].)²⁵⁹

Carboxyhemoglobin levels may be elevated in heat stroke, even without exposure to carbon monoxide; elevated carboxyhemoglobin levels may be due to heat shock protein up-regulation (specifically hemoxygenase-1, classified as heat-shock protein 32) [[PMID 10088844](#)].²⁶⁰ Oxyhemoglobin is partially oxidized to met-hemoglobin in severe heat stress conditions, but carboxyhemoglobin is resistant against heating [[PMID 11516900](#)].²⁶¹

System	Symptom, Sign, or Finding
CNS	<ul style="list-style-type: none"> • Delirium • Coma • Euphoria • Hallucinations • Rapid eye movement • Tremors • Tonic contractions • Seizures • Cerebellar dysfunction (dysarthria, ataxia, downbeat nystagmus [PMID 16311159])²⁶² • Hemiplegic episodes • CNS hemorrhage • Cerebrospinal fluid normal pressure, elevated protein levels to 150 mg/dl, occasional pleocytosis
Liver	<ul style="list-style-type: none"> • Acute hepatic failure [PMID 17226914]²⁶³ • Serum transaminase elevation (100x normal not uncommon) • Jaundice • Hypoglycemia
Kidneys	<ul style="list-style-type: none"> • Acute renal failure • Acute tubular necrosis (associated with rhabdomyolysis) • Myoglobinuria • Pyuria • Proteinuria • Microscopic hematuria • Granular casts

System	Symptom, Sign, or Finding
Muscles	<ul style="list-style-type: none"> • Cramps • Rigidity • Rhabdomyolysis • Elevated serum myoglobin • Hyperphosphatemia • Hyperuricemia • Elevated plasma creatinine kinase (CK)
Blood	<ul style="list-style-type: none"> • Coagulation abnormalities • Altered clotting time and clot retraction • Fibrinolysis • Platelet count usually low • Factors V and VIII usually low • Purpura • Conjunctival hemorrhages • DIC • White blood cell count may be 30,000 - 40,000/ul
Cardiovascular	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • Diarrhea • Vomiting • Mesenteric vascular constriction • Local areas of mucosal ulcerations • Hematemesis • Melena
Pulmonary	<ul style="list-style-type: none"> • Hyperventilation • Respiratory alkalosis • Hemoptysis • Pulmonary edema

Table 9 - Symptoms, Signs, and Findings in Heat Stroke

C3.1.9.4. 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](#)]²⁶⁴ to 105.8° F (41° C) [[PMID 9694423](#)].²⁶⁵

HEAT STROKE SHOULD BE SUSPECTED IN ALL PATIENTS WITH ALTERED MENTAL STATUS AND ELEVATED TEMPERATURE OR HIGH LEVELS OF EXERTION. If other etiology is not apparent, it should be considered heat stroke until proven otherwise. Rapid cooling should be instituted in such cases, while further studies (such as lumbar puncture, etc.) are pursued.

The table in [C6. Criteria for the Diagnosis of Heat Stroke](#) may assist in the diagnosis of heat stroke. (Note: criteria have been established for the diagnosis of heat stroke as a cause of death [[PMID 9095294](#)].)²⁶⁶

C3.1.10. Severe Exertional Heat Injury (“Exertional Heat Illness”)

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.

Chapter 4:

C4. Treatment of heat stroke

C4.1. Treatment of Heat Stroke - General

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.

C4.1.1. Cooling

The goal in the treatment of heat stroke is rapid cooling to (theoretically) normal body temperature. Clinically, to prevent over-cooling (reported to occur in 33% of heat stroke cases [[PMID 1608386](#)]),²⁶⁷ the target temperature of cooling is to 101° F (38.3° C) [Schmidt]²⁶⁸ (literature reports range from 99.5° F [[PMID 2692177](#)]²⁶⁹ to 102.2° F [[PMID 10959266](#)]).²⁷⁰ Rapid cooling to less than 102° F (38.9° C) is associated with a significantly reduced mortality rate [[PMID 3741557](#)].²⁷¹ To avoid “overshoot” (over-cooling), some clinicians clinical use a target temperature of 102° F (38.9° C) when using aggressive cooling (e.g., iced water immersion) and a rectal thermometer to monitor body core temperature; when the target temperature is reached, a fan with cool misted water or a lukewarm shower may be used [Noltkamper].²⁷²

Cooling should be started immediately on diagnosing heat stroke or serious heat exhaustion, preferably while the victim is being transported to the hospital. With the notation that overcooling should be avoided, the more rapid the cooling, the lower the mortality [[PMID 11510526](#)].²⁷³ However, although important in determining prognosis, early treatment may not prevent severe complications [[PMID 10959266](#)].²⁷⁴

Cooling may be done through three mechanisms: cooling the skin while maintaining cutaneous blood flow, cooling internal organs directly, and cooling blood directly by removal, cooling, and re-introduction of blood.

1) Cooling the skin while maintaining cutaneous blood flow

C4.1.1.1.1. Clothing Removal

Unless prohibited by operational personal protection requirements (e.g., battle, chemical or biological or radiological threat), the victim’s clothing should be removed immediately.

C4.1.1.1.2. Fanning

Cooling with large amounts of airflow is often the only effective cooling method immediately available. Helicopter downdraft cooling has been used successfully on heatstroke victims [[PMID 3579826](#)].²⁷⁵

C4.1.1.1.3. Cold or Ice Water Immersion

IMMERSION OF THE VICTIM IN COLD OR ICE WATER IS THE MOST EFFECTIVE TREATMENT TO RAPIDLY DECREASE CORE TEMPERATURE, AND SHOULD BE PERFORMED IMMEDIATELY [[PMID 9366461](#),²⁷⁶ [PMID 15248787](#)].²⁷⁷ As heat stroke victims may be shunting blood from the skin, and as cooling causes cutaneous vasoconstriction, effort may be necessary to restore or increase cutaneous blood flow. Cold or ice water immersion techniques are labor-intensive. Vigorous rubbing of the skin, or intermittent warm air or warm water exposure is done to maintain cutaneous blood flow.

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)²⁷⁸ may be effective at cooling the victim while avoiding the need for rubbing the skin. 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.

C4.1.1.1.4. Cold Packs, Cooling Blankets, Fanning, Mists, Cooling Units

One alternative to cold or ice water immersion is the application of cold packs or ice packs or ice water slush to part of the body. Another alternative is the use of cooling blankets (blankets with tubing containing a circulating coolant), which cool the patient without wetting. However, contact area with the body is less than can be achieved with immersion. They are unlikely to be available in a field situation. Evaporative cooling techniques may also be used, including fanning the patient with or without water or mist, and using a “body cooling unit” (a special bed spraying water at 59° F or 15° C and blowing air at 113° F or 45° C) [[PMID 12075060](#)].²⁷⁹

2) Cooling internal organs directly

Ice water NG lavage and/or ice water enemas may be used to rapidly cool the body. Iced peritoneal lavage may be effective when other methods, including evaporative cooling techniques and iced gastric lavage, fail [[PMID 2803356](#)].²⁸⁰ Iced urinary bladder lavage only removed a trivial amount of heat in one study [[PMID 9366461](#)].²⁸¹ Recent animal studies suggest that flushing of the nasal cavities with cooled gas (e.g., cooled nasal air) may protect the brain against hyperthermal damage [[PMID 12018975](#)].²⁸²

3) Cooling blood directly by removal, cooling, and re-introduction of blood

Advanced techniques such as this require specialized equipment. For example, successful cooling of victims (resistant to other methods of rapid cooling) using cold hemodialysis (initially 86° F or 30° C) with cold continuous hemodiafiltration has been reported [[PMID 16202019](#)].²⁸³

4) Medications

No medication has been shown to decrease core temperature. Dantrolene sodium did not prove beneficial to patients with heatstroke in a randomized, double-blind, placebo-controlled trial [[PMID 1989755](#)].²⁸⁴ However, research suggests dantrolene (a direct-acting skeletal muscle relaxant) may lead to attenuated heat production and peripheral vascular relaxation [[PMID 10519477](#)].²⁸⁵

C4.1.2. Cardiovascular Support

Preliminary work in animals suggests that maintenance of mean arterial pressure may be important in the treatment of heat stroke [[PMID 11508869](#)].²⁸⁶ IV hydration should be done with caution to avoid shifts of osmolality which may lead to cerebral edema and central pontine myelinosis if done too rapidly [[PMID 9267966](#)].²⁸⁷ Correction of reduced antithrombin III levels to supranormal by therapeutic administration of antithrombin III concentrate and steroids in disseminated intravascular coagulation of heat stroke has been reported [[PMID 12168565](#)].²⁸⁸ Treatment of heat stroke-associated conditions (DIC, rhabdomyolysis, arrhythmias, hypotension, shock, ARDS, renal failure, liver failure, electrolyte abnormalities) may be required (including

the use of aggressive techniques such as mechanical ventilation, blood purification therapy [[PMID 10229168](#)],²⁸⁹ etc.)

C4.1.3. Concomitant Therapy

Heat stroke has been reported to be a risk factor for the development of disseminated zygomycosis (one case of paranasal and GI zygomycosis in which *Rhizopus schipperae* was cultured [[PMID 10405417](#)]).²⁹⁰ Mechanisms regulating body heat may remain disturbed for days following early treatment and apparent stabilization, mandating continued hospitalization [[PMID 10959266](#)].²⁹¹ After recovery, re-exposure to heat stress conditions must be done with caution. Gradual acclimatization may eventually be successful, allowing the victim to be re-exposed safely to heat stress conditions [[PMID 2406545](#)].²⁹²

C4.2. Treatment of Heat Stroke - Clinical

C4.2.1. Preliminary Studies and Procedures

Treatment of heat stroke in the field should include immediate attempts to lower body temperature to 101°F (38.3°C). The victim's clothes should be removed. If there is a source of cool water nearby, the victim should be immersed in it. Otherwise, water should be sprinkled over the victim and evaporation hastened by fanning. Attendants should rub the victim's extremities and trunk briskly to increase circulation to the skin. 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].²⁹³ During transportation, cooling efforts should be continued by permitting passage of air currents through the open door of the field ambulance or helicopter.

Rectal temperature should be obtained on presentation of the victim to the medical facility. Laboratory studies should include a complete blood count (including hemoglobin, hematocrit, white blood count, and platelet count), PT/PTT, electrolytes (sodium, potassium, bicarbonate, chloride), blood urea nitrogen, creatinine, glucose, hepatic transaminases (AST, ALT), lactate dehydrogenase, creatinine kinase, and urinalysis. A full (12 lead) EKG and urine drug screen should be obtained. A chest x-ray and arterial blood gas (ABG) should be done if indicated. If ABGs are deemed necessary, the lab should be informed of the patient's body temperature in order to make corrections in determining the results. As in any patient with altered mental status and fever, a toxicologic screen and lumbar puncture may be indicated if the etiology is unclear. As cocaine may cause hyperthermia, special consideration should be given to assessing for cocaine. Blood osmolality, myoglobin, and urine myoglobin may be useful in patient management, although they may not be readily available. Calcium, magnesium, phosphorous, uric acid, protein, albumin, and lactic acid levels may be helpful in managing severe cases, and are recommended if there is suspicion of internal organ damage.

If heat stroke (or impending heat stroke) is a diagnostic possibility, continuous body core temperature readings should be taken via esophageal thermometer. If that is not available, rectal temperatures should be measured frequently, at least every 15 minutes. In addition, the victim's mental status should be checked frequently, as should blood pressure and urine output and color.

Definitive medical treatment should begin with general supportive therapy, including oxygen (by face mask or nasal cannula) or endotracheal intubation, as increased tissue oxygen requirements induced by hyperthermia are expected [[PMID 3063579](#)].²⁹⁴ Endotracheal intubation should be used in any patient with a reduced level of consciousness and in any heat stroke patient that has been sedated. IV fluid administration should be started, with the notation

that dehydration and volume depletion may be limited in heat stroke [[PMID 1852063](#)].²⁹⁵ NS or LR at 250 ml/hour may be required; however, heat stroke victims may be especially prone to congestive cardiac failure and pulmonary edema from excessive fluid administration [[PMID 1852063](#)].²⁹⁶ An NG tube should be placed to prevent aspiration in non-alert patients and for potential gastric lavage or administration of medications. A foley catheter should be placed and urine output monitored.

Exercise in heat stress conditions may cause hypoglycemia [[PMID 1602940](#)],²⁹⁷ [[PMID 2132167](#)],²⁹⁸ and hypoglycemia in heat stroke has been reported [[PMID 3398504](#)].²⁹⁹ (However, hypoglycemia in heat stroke is reported to be rare by other authors [[PMID 12075060](#)].)³⁰⁰ It is recommended that hypoglycemia is confirmed (for example, fingerstick blood glucose determination) prior to using IV fluids contain glucose.

If there is inadequate urine output after continued IV fluid resuscitation, osmotic diuretics (e.g., mannitol) may be considered. However, central line or Swan-Ganz monitoring may be desirable to ensure appropriate fluid administration [[PMID 1852063](#)].³⁰¹ Whether hypotensive patients who do not respond to saline should receive inotropic support is uncertain. Use of norepinephrine, if deemed necessary for hemodynamic control, probably should be avoided until after successful cooling has been accomplished. Increases in both norepinephrine and epinephrine have been found in patients with heat stroke, indicating activation of the sympatho-adrenal system, and researchers suggested that the alpha-mediated effect of catecholamines may be important in impairing heat dissipation and may contribute to the pathogenesis of heat stroke [[PMID 2499843](#)].³⁰² Additionally, experiments in rats demonstrated increased extracellular concentrations of dopamine, serotonin or norepinephrine in the hypothalamus and other brain regions during heatstroke-induced cerebral ischemia and neuronal damage; heatstroke-induced cerebral ischemia and neuronal damage were attenuated and survival increased by depletion of brain dopamine or serotonin [[PMID 9100936](#)].³⁰³

C4.2.1.1.1. Shivering

Overcooling may result in shivering [[PMID 1608386](#)].³⁰⁴ 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](#)],³⁰⁵ [[PMID 12075060](#)].³⁰⁶ Antipyretic agents are not indicated (although aspirin may have some efficacy in treatment of platelet aggregation abnormalities in heat stroke [[PMID 231070](#)]).³⁰⁷

C4.2.1.1.2. Electrolyte Abnormalities

In both classical and exertional heatstroke and in various animal models of human heat injury, clinical manifestations have included observations of normokalemia, hyperkalemia, and hypokalemia [[PMID 11990141](#)].³⁰⁸ (Some authors report severe hyperkalemia to be common in patients with exertional heat stroke but uncommon in those with classical heat stroke [[PMID 7078400](#)],³⁰⁹ while other recent reports found hypokalemia or normokalemia instead of hyperkalemia [[PMID 7644768](#)].)³¹⁰

C4.2.1.1.3. Rhabdomyolysis

Rhabdomyolysis may be recognized by discoloration of urine, and should be suspected in all cases of heat stroke. It may cause release of creatinine kinase and myoglobin into the vascular

system, and may be associated with elevated uric acid, phosphate, and potassium levels (as well as 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](#)].³¹¹ Urine alkalization 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](#)].³¹²

C4.2.1.1.4. Renal Injury

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](#)].³¹³ Uric acid may play a role in heat-related renal injury [[PMID 6611841](#)].³¹⁴ Continuous venovenous hemofiltration has been reported to be a good alternative to dialysis in hemodynamically unstable patients [[PMID 7644768](#)].³¹⁵ Core body temperature should be monitored closely until stable.

C4.2.1.1.5. Other Associated Injury

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](#)].³¹⁶ Extensive hepatocellular damage requiring liver transplant has been reported [[PMID 15838872](#)],³¹⁷ as has spontaneous recovery of a case that initially was thought to require liver transplantation [[PMID 15105986](#)].³¹⁸

Lower leg compartment syndrome has rarely been seen as a complication of or in conjunction with heat stroke [Noltkamper],³¹⁹ especially in sickle trait [[PMID 8677839](#)];³²⁰ health care providers should be aware that compartment syndrome may itself cause rhabdomyolysis [[PMID 7569117](#)].³²¹

Chapter 5:

C5. Follow-up of Heat Stress Injuries

After a heat stress injury, victims are less resistant to 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](#)].³²² 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.

C5.1. 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. MRI and/or neuropsychiatric testing may be indicated.

C5.2. 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](#)].³²³ One series of young heat stroke victims concluded that prominent neurological or behavioral sequelae in heat stroke victims are rare [[PMID 8372119](#)].³²⁴ 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](#)],³²⁵ [[PMID 7788975](#)],³²⁶ [[PMID 7575855](#)].³²⁷

C5.3. Re-Exposure to Heat

C5.3.1. Minor 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.

C5.3.2. Major Injuries

C5.3.2.1.1. 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.

C5.3.2.1.2. 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](#)].³²⁸ Some victims will not successfully acclimatize until months later, if at all [[PMID 2406545](#)].³²⁹ Some researchers have recommended testing 8 to 12 weeks after heat stroke to detect possible inability to cope with heat stress adequately [[PMID 2406544](#)].³³⁰

C5.4. Reporting

All Navy heat stress-related injuries should be reported through the Naval Disease Reporting System. A simultaneous report to the Naval Safety Center should be made using the Web Enabled Safety System ([WESS](#)). 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 6220.12A](#) (which stipulates Naval Disease Reporting System electronic report, or written, fax, e-mail or phone report to the cognizant NAVENPVNTMEDU).³³¹

C5.5. 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 (see [OPNAV 5100.23 series Chapter 29](#)).³³² 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 6:

C6. Criteria for the Diagnosis of Heat Stroke

The following table is set forth for use in non-emergent care of severe heat stress injury victims. For diagnostic criteria for use in acute care situations, see [Figure 5](#) (page 26).

Required for diagnosis of heat stroke	Abnormality	Criteria	Notations	Points
	Temperature elevation	105° F or higher (40.6° C)	<ul style="list-style-type: none"> • Taken by any means of instrumentation (i.e., not only with hand on forehead) • No known underlying condition to explain the high body temperature (for example, fever due to infection) 	4
		103° F to 104.9° F (39.4° C to 40.5° C)	<ul style="list-style-type: none"> • Taken by any means of instrumentation (i.e., not only with hand on forehead) • No known underlying condition to explain the high body temperature (for example, fever due to infection) 	2
		101° F or higher (38.4° C) after cooling	<ul style="list-style-type: none"> • 105° F or higher (40.6° C) suspected from history but not documented • No known underlying condition to explain the high body temperature (for example, fever due to infection) 	2
		Undocumented	<ul style="list-style-type: none"> • Suspected to have been 105° F or higher (40.6° C) based on history 	1
	Disordered mentation	<ul style="list-style-type: none"> • Confusion, extreme and persistent • Delirium • Seizures • Coma 	<ul style="list-style-type: none"> • Not due to other causes (no CNS trauma, no toxicant, no CNS infection) 	4
		<ul style="list-style-type: none"> • Mild confusion • Disoriented 	<ul style="list-style-type: none"> • Does not clear or resolve immediately 	2
	Cardio-vascular abnormalities	<ul style="list-style-type: none"> • Arrhythmias • Hypotension • Ischemia • Pulmonary edema 	<ul style="list-style-type: none"> • Not present prior to exposure to heat stress exposure 	2
	Coagulation abnormalities	Disseminated intravascular coagulopathy (DIC)	<ul style="list-style-type: none"> • Abnormal clotting studies • Hemorrhage (skin, gastrointestinal, genitourinary) 	2
		<ul style="list-style-type: none"> • PT or PTT ≥ twice normal • Platelets < 100,000 	<ul style="list-style-type: none"> • Without other known explanation 	1

Rhabdomyolysis	<ul style="list-style-type: none"> • Myoglobinuria (positive urine myoglobin test) • Elevated serum myoglobin (radio-immunoassay > 100) 	<ul style="list-style-type: none"> • Without other explanation (crush injury, overexertion, alcohol abuse, medicines, toxicants) • If serum myoglobin, without other explanation of elevated serum myoglobin (such as myocardial injury) 	2
Sweating abnormalities	Cessation of sweating	<ul style="list-style-type: none"> • In spite of exposure to heat stress conditions 	1
Respiratory distress	Adult respiratory distress syndrome (ARDS)	<ul style="list-style-type: none"> • Without other explanation 	2
Shock	BP < 90/60	<ul style="list-style-type: none"> • With evidence of inadequate organ perfusion • Without other explanation 	2
Blood chemistry abnormalities	Hypernatremia	<ul style="list-style-type: none"> • Sodium > 150 	1
	Hypophosphatemia	<ul style="list-style-type: none"> • Inorganic phosphorus < 2.5 mg/dL 	2
	Hypokalemia	<ul style="list-style-type: none"> • Potassium < 3.1 	1
	Hyperkalemia	<ul style="list-style-type: none"> • Potassium > 5.5 	1
<p>Total required for heat stroke diagnosis</p> <ul style="list-style-type: none"> • Must be 6 or more • Must include points from both temperature and CNS categories • Treatment for heat stroke should not be delayed until a definite diagnosis of heat stroke is made using the above criteria (which may take days to develop). Instead, Figure 5 - Acute Heat Exposed Patient Diagnostic Flow Chart, page 26, should be used. 			

Table 10 - Diagnostic Criteria for Heat Stroke

Chapter 7:

C7. Physiology of Cold Stress

C7.1. Introduction

In contrast to heat stress, cold stress presents challenges to the body in retaining heat produced by metabolism. Air temperatures 88° F (31.1° C) and lower may represent cold stress conditions to healthy people (for example, in newborns 2 to 10 days old) [[PMID: 3970567](#)],³³³ and temperatures below 95° F (35° C) may pose a risk of cold stress injury to individuals without insulation or without functioning thermoregulatory mechanisms [Currier].³³⁴

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](#)].³³⁵ Cold temperature also can lower the immune system's resistance to respiratory infection, causing an increase in respiratory disease mortality [[CDC](#)]³³⁶ [[PMID: 9149695](#)].³³⁷ Hypothermia decreases performance [[PMID: 16538942](#)],³³⁸ increases the risk of bleeding [[PMID: 15211129](#)],³³⁹ and is an independent risk factor of mortality in trauma patients [[PMID: 16385283](#)].³⁴⁰ Hypothermia may also alter the response to certain medications (e.g., increased rather than decreased blood pressure from thiopental) [[PMID: 17023288](#)].³⁴¹

C7.1.1. 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.

C7.1.2. 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).

C7.2. Environmental Cold Stress Factors

C7.2.1. 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 [below](#)) 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](#)].³⁴² (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.)

C7.2.2. 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].³⁴³ 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 °F³⁴⁴

No adjustments are made for sky conditions; a clear night sky is assumed [NWS].³⁴⁵

Windchill index [calculators](#) are available on the Internet.³⁴⁶

Note: Frostbite occurs in 15 minutes or less at windchill values of -18° F (-27.8° C) or lower [NWS].³⁴⁷

C7.2.3. Humidity and Moisture

Humidity affects environmental cold stress by affecting how quickly evaporation (for example, of sweat) from the skin takes place.² 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.

C7.2.4. 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].³⁴⁸ 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.

C7.2.4.1. Diving Reflex

Immersion triggers the diving reflex (bradycardia response) [PMID: 11816961].³⁴⁹ 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].³⁵⁰

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.

² The quantity of heat absorbed by water changing to steam is the “latent heat” or “heat of transformation.”

C7.2.4.2. Fluid shifts in Cold Water

Vascular fluid shifts, body cooling, and diuresis are all greater in cold water than in cold air [PMID: 3629738].³⁵¹ The percent reduction in plasma volume from cold exposure is significantly larger in cold water (-17%) than in cold air (-12%) [PMID: 3629738].³⁵²

C7.2.4.3. 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].³⁵³ Protective effects of increased body fat against cold stress, although significant on dry land [PMID: 6735815]³⁵⁴ and during immersion [PMID: 11043627],³⁵⁵ are minimal compared to the benefit of a dry suit during cold-water immersion [PMID: 2803162].³⁵⁶

C7.2.4.4. 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],³⁵⁷ has been associated with swimming and diving. Previously thought to be a cold-water phenomenon, recently (1997) it has been reported in warm water [PMID: 9068153],³⁵⁸ [PMID: 11728772].³⁵⁹ 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],³⁶⁰ [PMID: 9068153].³⁶¹ 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].³⁶²

C7.2.4.5. Awareness of Cold Strain in Cold Water

Personnel immersed in cold water cannot reliably assess how cold they are [PMID: 2803163].³⁶³ 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].³⁶⁴

C7.2.4.6. 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].³⁶⁵

C7.2.4.7. Survival Times 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].³⁶⁶ 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

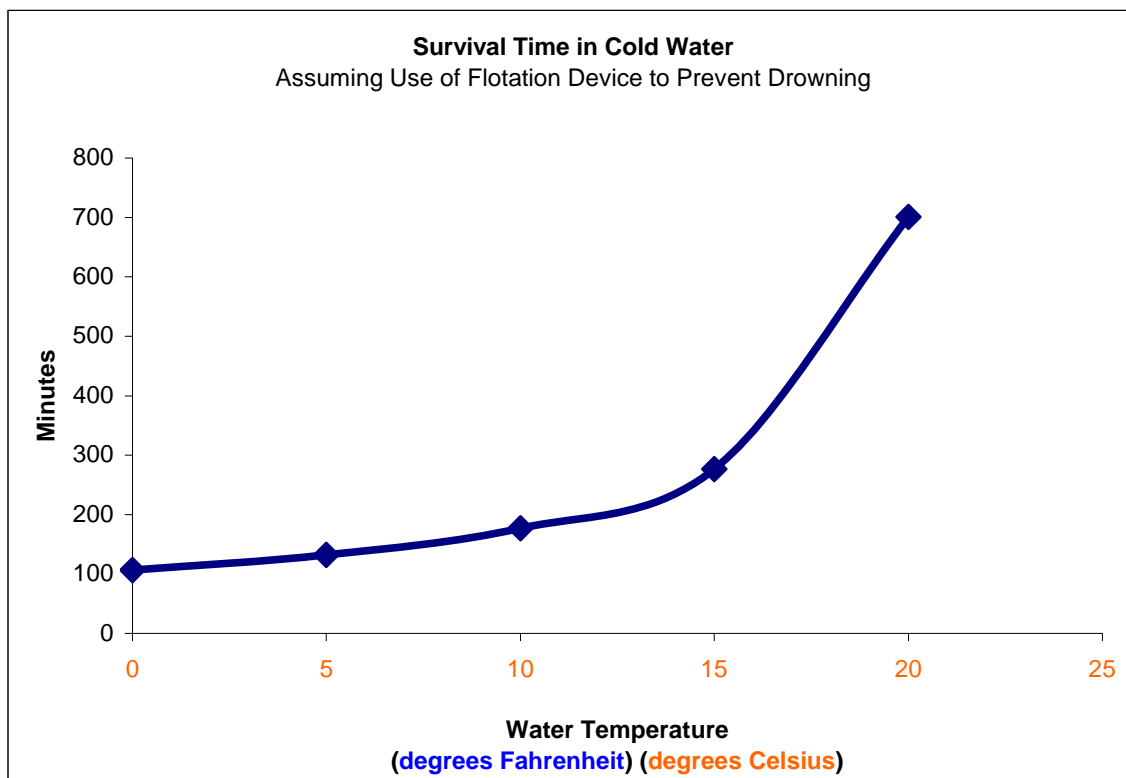
prediction equation for survival time of persons accidentally immersed in cold water (32° F to 71.6° F, or 0° C to 22° C) is as follows [[PMID: 1139445](#)].³⁶⁷

$$\text{Survival Time} = 15 + 7.2 / (0.0785 - (0.0034 \times \text{water temperature in } ^\circ\text{C})).$$

Survival time may be shorter in very cold water (e.g., one hour at 32° F or 0° C) [[PMID: 6721816](#)],³⁶⁸ and in rough seas [[PMID: 3606516](#)].³⁶⁹ 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].³⁷⁰

In falls through thin ice, the greatest risk comes from drowning (both to the individual and to the rescuers). The victim should control their breathing, make their way to the side where they fell in (where the ice is stronger), attempt to extricate themselves (using implements such as ski poles via the high dagger method, if available), and hold on to the side, perhaps even freezing to the ice with their sleeves. Rescuers should plan the rescue properly, not rushing to the edge or jumping in to save the victim and perishing themselves: there is some time before hypothermia will become deadly.³⁷¹

Figure 6 - Survival Time in Cold Water (estimated)



C7.2.4.8. 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 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](#)].³⁷²

C7.2.5. Elevation (Altitude)

Altitude may accentuate certain responses to cold stress. The respiratory responses during acute cold exposure (see [below](#)) are similar to those of initial altitude responses [[PMID: 8468097](#)].³⁷³ It is felt by some that cold can predispose individuals to high altitude pulmonary edema [[PMID: 9152300](#)].^{374,375}

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](#)].³⁷⁶

Frostbite is common in mountain climbers; one study of self-reported history of frostbite injury showed a mean incidence of 366/1000 population per year, significantly related to lack of proper equipment and to not having a guide [[PMID: 16306495](#)].³⁷⁷

C7.2.6. 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].³⁷⁸

One experimental investigation concluded that metal surfaces in contact with bare hands should not be below 39.2° F (4° C) surface temperature, and that lower temperatures require insulating material or the wearing of protective gloves [[PMID: 7957157](#)].³⁷⁹ Contact pressure, contact material mass, surface temperature, and whole body thermal balance all were found to significantly impact contact skin temperature change with time. Of special concern is that in persons handling cold metal objects with their fingers, pain and temperature sensation do not correlate well with skin temperature or change in temperature [[PMID: 8184801](#)].³⁸⁰ 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](#)].³⁸¹ Some clinicians feel that allowing limited ungloved handling of metal objects may be safe for experienced personnel.³⁸²

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) (see [below](#), [Dexterity when Handling Cold Objects](#)) [[PMID: 8049001](#)].³⁸³ Local hypothermia (cooling the forearm to 86° F or 30° C and to 82.4° F or 28° C) was found to increase local bleeding time but did not affect bleeding time at other parts of the body [[PMID: 17096671](#)].³⁸⁴

C7.2.6.1. Time to Reach Contact Temperature

In examining the time it takes to cool the hand when gripping a cold object, researchers found that women were found to reach a “contact temperature” (i.e., the temperature of the palm of the hand—in the study, 59° F or 15° C) significantly faster than men (in approximately only 70% of the time it takes men’s palms to reach the same temperature) [[PMID: 12074025](#)].³⁸⁵ Interestingly, the same study found that time to reach a contact temperature was actually longer

when subjects were standing in a cold room, rather than standing in a warm room and inserting their hands into a cold box [PMID: 12074025].³⁸⁶ The little finger is the limiting factor in exposure of the hand to cold air, as it is particularly vulnerable to rapid cooling [Daniels].³⁸⁷

C7.2.6.2. Dexterity when Handling Cold Objects

Exposure to the cold may negatively influence manual dexterity [PMID: 12074025].³⁸⁸ 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].³⁸⁹ 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].³⁹⁰

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].³⁹¹

C7.2.6.3. 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].³⁹²

C7.2.7. 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].³⁹³ 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).

C7.3. Individual Cold Stress Factors

C7.3.1. Body Mass and Fat

Individuals with higher body mass and more body fat can better tolerate cold stress exposure [PMID: 984737].³⁹⁴

C7.3.2. 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],³⁹⁵ but not all studies support that finding [PMID: 3780704].³⁹⁶ At least one review has concluded that individual body size, physical fitness, and state of acclimatization play for more important roles than gender in determining human thermal responses [PMID: 750842].³⁹⁷

C7.3.3. Age

In a cold environment, children have lower skin temperatures, reflecting greater vasoconstriction [PMID: 9587181].³⁹⁸ 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].³⁹⁹

C7.3.4. Race and/or Ancestral Geographic Location

In one study, high altitude (11,500 feet or 3500 meters above sea level) natives maintained significantly higher oral temperature, mean body temperature, and skin temperature during sea level cold stress; they shivered much less, and showed higher peripheral blood flow than “lowlanders;” however, there was no difference found in the rise in oxygen consumption in cold stress [PMID: 464958].⁴⁰⁰ Different types of general cold adaptation have been described in different people groups, including metabolic adaptation (Alacaluf Indians, Arctic Indians), insulative adaptation (coastal Aborigines of tropical northern Australia), hypothermic adaptation (bushmen of the Kalahari desert, Peruvian Indians) and insulative hypothermic adaptation (Central Australian Aborigines, nomadic Lapps, Korean and Japanese diving women) [PMID: 1483765].⁴⁰¹ Whether genetics or climate characteristics are responsible in each case is not established; acclimatization (see [below](#)) to cold stress is possible in men from tropical climates as well as men from temperate zones [PMID: 1297856].⁴⁰²

C7.3.4.1. Metabolic Adaptation

Indigenous circumpolar people groups have systematic and statistically significant elevations in basal metabolic rate (BMR) ranging from 7% to 19% above predicted values for indigenous men and from 3% to 17% for indigenous women [PMID: 12203815].⁴⁰³ This is considered a metabolic adaptation to cold stress (also see [Metabolism, below](#)).

C7.3.4.2. Insulative Adaptation

A group of acclimatized cross-country skiers was found to have lower skin temperatures after exposure to cold stress; metabolic heat production was not increased [PMID: 1555569].⁴⁰⁴ This is an insulative adaptation to cold stress.

C7.3.4.3. Hypothermic Adaptation

A group of study subjects exposed to cold for 10 days showed an increase in the delay before the onset of shivering and a decrease in the core temperature at the onset of shivering [PMID: 11507988].⁴⁰⁵ termed a hypothermic adaptation.

C7.3.5. 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].⁴⁰⁶ A recent case report noted that severe ethanol poisoning, in the absence of any other contributing factors, may explain hypothermia [PMID: 17251602].⁴⁰⁷ 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]⁴⁰⁸ [PMID: 8897037].⁴⁰⁹

C7.3.6. Hydration

Cold exposure may lead to significant dehydration (via cold diuresis, high energy expenditures, and poor access to water) [PMID: 7639888].⁴¹⁰ 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](#)].⁴¹¹ 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](#)].⁴¹² The same study found that inadequate drinking water availability was associated with inadequate nutritional intake (troops preferred to go hungry) [[PMID: 3116457](#)].⁴¹³

C7.3.7. Central Nervous System (CNS) Abnormalities

CNS abnormalities involving the corpus callosum (including agenesis of the corpus callosum, multiple sclerosis, and previous head injury) have been associated with abnormal body temperature regulation in what has been termed “spontaneous hypothermia” or Shapiro’s syndrome [[PMID: 16459729](#),⁴¹⁴ [PMID: 17250734](#)].⁴¹⁵ The disorder has an unpredictable course, generally with long periods of remission [[PMID: 15990200](#)].⁴¹⁶

C7.3.8. Heat Debt

Heat debt refers to the net amount of heat lost to the environment that would be necessary to return the entire body to normal temperature in thermo-neutral conditions. The term is often used in studies of the effects of cold stress exposure, but is generally not of use in clinical practice.

C7.4. Compensation for Cold Environments

C7.4.1. Functioning in Cold Environments

Moderate cold exposure (even that does not produce core hypothermia) can impair performance of complex cognitive tasks [[PMID: 2818396](#)].⁴¹⁷ Mechanisms to compensate with cold environmental temperatures include behavior changes (voluntary muscular activity and exercise, staying indoors, wearing warm clothing, growing—or allowing to grow longer—a beard and/or long hair) and physiological responses. Physiological responses to cold stress include increasing body fat (which acts as insulation and as an energy reserve for increased metabolic needs), increased appetite (related to increased body fat), increased (or decreased) metabolism, increased activity, decreased cutaneous blood flow (resulting in cool skin, which may appear pale, mottled, or blue), shunting of blood from skin and extremities (to decrease cutaneous loss of heat, but which may increase likelihood of Chilblains and frostbite), and shivering. Certain individuals demonstrate a response termed paradoxical undressing (a pathologic response occasionally seen in alcoholics exposed to cold stress [[PMID: 541627](#)]⁴¹⁸ [[PMID: 2036058](#)]).⁴¹⁹ Infants, but not adults, can also increase thermogenesis by the metabolism of brown fat [[PMID: 9180091](#)].⁴²⁰

Manual dexterity is decreased after cold exposure [[PMID: 11374119](#)].⁴²¹ Maximum grip strength improves somewhat immediately following the application of cold and then declines [[PMID: 4083336](#)].⁴²² Peripheral motor nerve conduction velocity has been found to decrease by 15 meters per second for every 18° F (10° C) fall in tissue temperature; at a tissue temperature of 46° F to 50° F (8-10° C), a complete nervous block was established [[PMID: 1115693](#)].⁴²³

Exposure to cold is frequently in high altitude environments. 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](#)].⁴²⁴ Absorption of protein and fat has not been shown to be a factor, at least up to 5000 feet above sea level [[PMID: 1490954](#)].⁴²⁵

C7.4.2. Metabolism

C7.4.2.1. 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](#)].⁴²⁶

C7.4.2.2. 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](#)].⁴²⁷ Individuals with less body fat tend to expend more calories when exposed to cool water [[PMID: 11990094](#)].⁴²⁸

Increased oxidation of carbohydrates and free fatty acids is a well-known phenomenon during cold stress [[PMID: 12079880](#)].⁴²⁹ Human studies have shown that cold exposure increases lipid oxidation [[PMID: 2233284](#)].⁴³⁰ 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](#)].⁴³¹ In one study, during exercise for 90 minutes in cold air, fat oxidation was found to be diminished, thought by researchers to reflect either a reduction in lipolysis and/or mobilization of free fatty acids or impairment in the oxidative capacity of muscle [[PMID: 11984294](#)].⁴³² Free fatty acid levels are not higher with exercise in cold air or water [[PMID: 1925184](#)].⁴³³ Cold-induced vasoconstriction of peripheral adipose tissue may account, in part, for the decrease in lipid mobilization [[PMID: 1925184](#)].⁴³⁴

C7.4.2.3. Hormones

Plasma concentrations of epinephrine, insulin, cortisol, and growth hormone are unaffected by cold exposure; norepinephrine increases about threefold [[PMID: 12079880](#)].⁴³⁵

Decreased fertility has been noted with cold exposure, which is thought to change metabolic fuel oxidation, affecting viscera and parts of the brain (the hindbrain in animals), which in turn affect the activity of gonadotropin-releasing hormone neurons in the forebrain [[PMID: 8772468](#)].⁴³⁶

C7.4.2.4. Calories and Cold Exposure

C7.4.2.4.1. Caloric requirements

The change in core temperature that occurs as a result of exposure to cold air or water affects all body systems [[PMID: 11816961](#)].⁴³⁷ The caloric 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](#)].⁴³⁸ Exercising in cool water has not been found to increase energy expenditure and weight loss (beyond the effect of exercising in general) [[PMID: 7068314](#)].⁴³⁹

One investigation reported that during prolonged, low-intensity shivering, the energy for heat production is from lipids (50%), carbohydrates (40%, with 30% from muscle glycogen and 10% from plasma glucose), and proteins (10%) [[PMID: 12070189](#)].⁴⁴⁰ Another study reported carbohydrate (rather than fat) oxidation represented the major fuel for thermogenesis in the cold (51%) [[PMID: 2767069](#)].⁴⁴¹ Lipolysis and free fatty acid turnover are greatly increased by cold

stress, but only about half the rate of free fatty acid turnover is ultimately oxidized [[PMID: 9895020](#)].⁴⁴²

Certain metabolic conditions (obesity and diabetes in women) are associated with an abnormal metabolic response to cold stress, possibly related to abnormal thyroid responses [[PMID: 3254262](#)].⁴⁴³ A study of obese women exercising in cool water concluded that while cold exposure does not increase caloric expenditure significantly in obese individuals, exercising regularly in cool water may motivate obese people to exercise at higher intensity for thermal comfort [[PMID: 7068314](#)].⁴⁴⁴

C7.4.2.4.2. Appetite

Decrease in appetite and food intake has been noted in cold exposure at high altitudes [[PMID: 1582718](#)];⁴⁴⁵ however, this appears to be due to hypobaric hypoxia [[PMID: 10409600](#)].⁴⁴⁶ Conversely, although a cold-induced increase in appetite may be expected, evidence for such a phenomenon is poor [[PMID: 10817145](#)].⁴⁴⁷ Water availability may affect food intake, as noted previously (see [above](#)).

C7.4.3. Cardiovascular

C7.4.3.1. Shunting

Cold constricts cutaneous blood vessels by increasing the reactivity of smooth muscle alpha (2)-adrenergic receptors [[PMID: 10749700](#)].⁴⁴⁸ Vasoconstriction via the sympathetic nervous system is most pronounced in the extremities, but is minimal in the head and neck [[PMID: 1811574](#)]⁴⁴⁹ [[PMID: 2221434](#)].⁴⁵⁰

C7.4.3.2. Blood Pressure

Cold exposure increases blood pressure [[PMID: 11374119](#)].⁴⁵¹ Exposure to cold air increased average systolic and diastolic pressures approximately 20 millimeters of mercury each [[PMID: 11214769](#)].⁴⁵² Blood pressure is noted to be higher in winter than in summer [[PMID: 9314429](#)].⁴⁵³

C7.4.3.3. Heart Rate and Cardiac Output

Heart rate may be lower or unchanged during exercise in the cold [[PMID: 1925184](#)].⁴⁵⁴ For those with coronary artery disease, heart rate during exercise in the cold may be unchanged or higher [[PMID: 8529083](#)].⁴⁵⁵ Cardiac output increases with cold exposure (e.g., 10% increase at 50° F or 10° C) [[PMID: 3968010](#)].⁴⁵⁶ Cold-water immersion including the face triggers the diving reflex (bradycardia response) [[PMID: 11816961](#)].⁴⁵⁷ Immersion of only a hand causes increased heart rate [[PMID: 11209666](#)].⁴⁵⁸ Cold increases the risk of arrhythmia during exercise [[PMID: 11505864](#)].⁴⁵⁹

C7.4.3.4. 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](#)].⁴⁶⁰ Plasma volume decreases on exposure to cold stress [[PMID: 3629738](#)].⁴⁶¹

C7.4.4. 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].⁴⁶²

Minute ventilation is substantially increased upon initial exposure to cold [PMID: 1925184].⁴⁶³ Acute cold stress exposure early in acclimatization causes a decrease in ventilation parameters.³ As acclimatization continues, there is a gradual recovery continuing up to 9 weeks [PMID: 8468097].⁴⁶⁴

C7.4.5. Shivering

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.

C7.4.5.1. Metabolism of Shivering

Shivering can increase the metabolic rate to a maximum of approximately five times the resting rate [PMID: 11394237].⁴⁶⁵ 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].⁴⁶⁶ Thermoregulatory responses of cold stress exposure are not related to muscle glycogen levels of major skeletal muscle groups; other metabolic substrates may enable sparing of muscle glycogen during shivering [PMID: 2732173].⁴⁶⁷ Hypoglycemia may delay shivering onset until core temperature is lower than what would cause shivering with normal blood sugar levels [PMID: 8964720].⁴⁶⁸

C7.4.5.2. Peak Shivering Metabolic Rate Equation

An equation to calculate the peak shivering metabolic rate has been formulated as follows [PMID: 11394237].⁴⁶⁹

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

$$30.5 + 0.348 \times \text{maximal oxygen uptake (in milliliters of oxygen per kilogram per minute)} - \\ 0.909 \times \text{body mass index (in kilograms per square meter)} - \\ 0.233 \times \text{age (in years)}.$$

C7.4.6. Fatigue

Vasoconstrictor responses to cold, but not shivering responses, are impaired after multiple days of severe physical exertion [PMID: 11181604].⁴⁷⁰ 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].⁴⁷¹ These findings suggest that susceptibility to hypothermia is increased by exertional fatigue [PMID: 11181604].⁴⁷²

C7.4.7. Circadian Rhythm (Body Clock)

Cold exposure at night lowers core body temperature and increases blood pressure more than cold exposure in the afternoon; skin temperatures, however, decrease less after cold exposure at night than they do after cold exposure in the afternoon, and may explain the difference in core temperatures [PMID: 11374119].⁴⁷³ Researchers report sensations of thermal cold and pain and manual dexterity are less after cold exposure at night than during the day,

³ Vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in the first second (FEV₁), peak expiratory flow rate (PEFR), and maximum voluntary ventilation (MVV).

which were felt to suggest that there is an increased risk of both hypothermia and accidents for those who work at night [[PMID: 11374119](#)].⁴⁷⁴

C7.5. Acclimatization

C7.5.1. 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.⁴

C7.5.2. 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](#)];⁴⁷⁵
- A slight (less than 0.5° F) decrease of core body temperature levels at the onset of shivering [[PMID: 3597234](#)];⁴⁷⁶
- Lower core body temperature in thermoneutrality (less than half a degree F) [[PMID: 3597234](#)];⁴⁷⁷
- A decrease of heat debt calculated from the difference between heat gains and heat losses (by about one third) [[PMID: 3597234](#)];⁴⁷⁸
- Reduced sensitivity to the pressor effect of norepinephrine [[PMID: 8299617](#)];⁴⁷⁹
- Reduced cold-induced muscle tenseness [[PMID: 8299616](#)];⁴⁸⁰
- Less shivering on cold exposure [[PMID: 1483764](#)];⁴⁸¹
- Lower central and peripheral body temperatures at rest and during cold immersion [[PMID: 8765994](#)];⁴⁸²
- Delayed metabolic response to cold [[PMID: 8765994](#)];⁴⁸³
- Attenuated subjective shivering [[PMID: 8765994](#)];⁴⁸⁴
- Lowered cold sensation [[PMID: 8765994](#)];⁴⁸⁵
- Increased vasoconstriction, evidenced by lowered skin temperature [[PMID: 8765994](#)];⁴⁸⁶
- Increased plasma potassium concentration during cold exposure (no change in plasma sodium concentration) [[PMID: 3629738](#)];⁴⁸⁷
- Increased rate of rewarming [[PMID: 655993](#)];⁴⁸⁸
- Lowered diastolic pressure and an increase in peripheral vasoconstriction [[PMID: 8891513](#)];⁴⁸⁹
- 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](#)];⁴⁹⁰
- Increased minute ventilation during cold air and cold-water exposure [[PMID: 3388627](#)];⁴⁹¹
- Lower oxygen consumption in cold air (but not in cold water) [[PMID: 3388627](#)];⁴⁹²
- Cold acclimation attenuates the onset of metabolic heat production during cold air exposure [[PMID: 3388627](#)].⁴⁹³

⁴ While “acclimatization” and “acclimation” are not identical, use in this text will be generally as used by authors.

C7.5.3. 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](#)].⁴⁹⁴
- Cold acclimation does not affect the minute ventilation - carbon dioxide production relationship or the pattern of breathing in cold air or water [[PMID: 3388627](#)].⁴⁹⁵
- Cold acclimation does not alter the magnitude of metabolic heat production [[PMID: 3388627](#)].⁴⁹⁶
- Cold acclimation has no effect on cardiac output or arterial-venous oxygen difference [[PMID: 3388627](#)].⁴⁹⁷
- 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](#)].⁴⁹⁸
- Acclimatization does not induce non-shivering thermogenesis in adults [[PMID: 8299617](#)].⁴⁹⁹

C7.5.4. Development of Acclimatization

Repeated cold-water immersion can induce the development of an insulative type of cold acclimation in man, specifically including changes in thermoregulation [[PMID: 3710973](#)].⁵⁰⁰ However, vascular fluid responses to cold stress are not altered by repeated cold-water immersion [[PMID: 3629738](#)].⁵⁰¹

C7.5.4.1. 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](#)].⁵⁰²

C7.5.4.2. 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](#)].⁵⁰³ 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](#)].⁵⁰⁴ Local cold acclimatization is possible regardless of what climate a person (or a person's ancestors) is from [[PMID: 1297856](#)].⁵⁰⁵

C7.5.5. 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](#)].⁵⁰⁶ Habituation is “the method by which the nervous system reduces or inhibits responsiveness during repeated stimulation” [Stedman's].⁵⁰⁷ 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](#)].⁵⁰⁸

C7.5.6. Non-Shivering Thermogenesis

In newborn humans and in many mammals, brown adipose tissue is metabolized to maintain body temperature (non-shivering thermogenesis) [[PMID: 1891665](#)].⁵⁰⁹ However, during metabolic cold acclimation, brown adipose tissue is not a major site for non-shivering thermogenesis in adult humans [[PMID: 7744360](#)].⁵¹⁰

Chapter 8:

C8. Prevention of Cold Stress Injuries

C8.1. Measurement of Cold Stress Effects on the Body

C8.1.1. Core Body Temperature

The most reliable method generally available in hospital settings for determining core body temperature is the esophageal thermometer (see section on [Prevention Of Heat Stress Injuries](#)). 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.

Esophageal temperatures, but not rectal or tympanic temperatures, have been found to be representative of cardiac temperature [[PMID: 6322264](#)].⁵¹¹

C8.1.2. 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.

C8.1.3. Cold Strain Index

A cold strain index based on rectal and mean skin temperatures has been proposed [[PMID: 10444564](#)],⁵¹² but has been shown to require further refinement before being considered valid for widespread use in quantifying cold strain [[PMID: 11705759](#)].⁵¹³

C8.2. Identifying Risk Factors

Prevention of cold stress injuries begins with recognition of risk factors. Arctic locations or freezing weather are not the only conditions in which cold stress injuries occur. Severe hypothermia in a tropical setting has been reported [[PMID: 9791596](#)].⁵¹⁴

C8.2.1. Protective Clothing

Collectively, the limited experimental work and the results of simulation modeling argue against any increased risk of hypothermia associated with wearing nuclear, biological, or chemical (NBC) protective clothing while working in the cold. However, 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](#)].⁵¹⁵

C8.2.2. 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](#)].⁵¹⁶

C8.2.3. Vasospastic Syndrome

People with vasospastic syndrome have cold hands and feet and abnormal vasoconstriction after local cold exposure [[PMID: 11463418](#)].⁵¹⁷

C8.2.4. 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](#)].⁵¹⁸

C8.2.5. Shapiro's Syndrome

Shapiro's Syndrome (Spontaneous Periodic Hypothermia) is a very rare disorder of temperature regulation, rather than merely a risk factor for hypothermia (see previous section on [Central Nervous System \(CNS\) Abnormalities](#)).

Chapter 9:

C9. Diagnosis and Treatment of Cold Stress Injuries

C9.1. Dermatological (Skin) injuries

C9.1.1. Conditions Unmasked or Exacerbated by Cold Exposure

C9.1.1.1. Acrocyanosis

Acrocyanosis (also known as Crocq's disease), “a circulatory disorder in which the hands, and less commonly the feet, are persistently cold and blue” [Stedman's],⁵¹⁹ is more intense in cold weather. Symptoms are permanent and painless cyanosis of extremities, local hypothermia, permanent sweateness, and elastic infiltration of the integument; capillaroscopy visualizes capillarovenular stasis [[PMID: 9814068](#)].⁵²⁰ Some forms of acrocyanosis are related to Raynaud's phenomenon (see [below](#)).

C9.1.1.2. Rosacea

Rosacea (also known as acne erythematosus 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](#)].⁵²¹

C9.1.1.3. Cold Agglutinin Disease

Cold agglutinin disease is a symptom-producing monoclonal B-cell lymphoproliferative disorder [[PMID: 9201236](#),⁵²² [PMID: 11722415](#)].⁵²³ The dysfunction of auto-reactive B-cell clones may be triggered by infection of some viruses and bacteria, or by certain medications [[PMID: 8890588](#)].⁵²⁴ Cold agglutination may also be associated with underlying disease (lymphoma, post-mycoplasma or infectious mononucleosis infections) [Schrier].⁵²⁵

Cold agglutinin disease may present as an autoimmune hemolytic anemia, characterized by high titers of serum IgM agglutinins (antibodies) maximally active at 39.2° F (4° C) [Schrier].⁵²⁶ It may be difficult to draw blood, and the red cells may visibly agglutinate in a cold syringe and on the blood smear. Symptoms are livedo reticularis of the thighs and a history of acrocyanosis and Raynaud's phenomenon upon cold exposure [[PMID: 11455160](#)].⁵²⁷ (See [Cold Agglutinin Disease](#), [below](#), for a further discussion.)

C9.1.1.4. 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](#)].⁵²⁸ Also known as Haxthausen's disease in children [[PMID: 3370518](#)],⁵²⁹ 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](#)].⁵³⁰

C9.1.1.5. Xerosis

Impaired desquamation may be one consequence of cold exposure, resulting in xerosis (dry skin); xerosis on the limbs due to impaired desquamation is not rare [[PMID: 10667045](#)].⁵³¹

C9.1.1.6. 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].⁵³²

C9.1.2. Chilblain

Chilblain (chilblains, Milker's chilblains, erythema pernio, perniosis) is a vascular (erythrocyanotic) discoloration of the acral skin occurring after exposure to the cold (generally moist cold). It is most common in young women although it can happen at every age, and relapses characteristically in autumn and winter [PMID: 9814070].⁵³³ Chilblain results from reversible alterations of the dermal vasculature [PMID: 10667045],⁵³⁴ including an obstructive and thrombotic microangiopathy, especially of venules [PMID: 3958629].⁵³⁵ Histology shows vascular and perivascular capillary and venular lymphocyte infiltrates without necrosis [PMID: 1431611].⁵³⁶ It also may be associated with underlying connective tissue disorders (specifically lupus erythematosus [PMID: 11388094]),⁵³⁷ especially if it lasts longer than one month [PMID: 11388094].⁵³⁸

Symptoms include pruritic, painful (especially burning) red patches on the fingers and/or toes, generally bilaterally. Sunlight may aggravate the lesions [PMID: 8474715].⁵³⁹ Ultraviolet irradiation can induce cutaneous lesions during winter [PMID: 10667045].⁵⁴⁰ Laboratory studies are normal. Diagnosis may be difficult [PMID: 8474715].⁵⁴¹ The differential diagnosis includes lupus, cold urticaria, acrocyanosis, erythromelalgia (erythermalgia, Gerhardt's disease; Mitchell's disease; red neuralgia; rodalgia), vasculitis and the Blue Toe Syndrome (tissue ischemia secondary to cholesterol crystal or atherothrombotic embolization leading to the occlusion of small vessels [PMID: 12555011])⁵⁴² [PMID: 1431611].⁵⁴³ Spontaneous healing is common when spring arrives and relapse is frequent during the following winters [PMID: 1431611].⁵⁴⁴ Significant scarring may result [PMID: 8474715].⁵⁴⁵

Treatment is removal from the cold (including passive warming). Nifedipine may hasten resolution of symptoms, signs, and biopsy findings [PMID: 2647123].⁵⁴⁶ Prevention includes prophylactic measures against cold; nifedipine is also suggested as a prophylactic (vasodilator) therapy [PMID: 8474715].⁵⁴⁷ In more severe cases, thyrocalcitonin and hemodilution might be helpful [PMID: 1431611].⁵⁴⁸

C9.2. Injuries of the Extremities Due to Cold Exposure

C9.2.1. Frostbite

Frostbite (congelation, dermatitis congelationis, "Teruel feet" [PMID: 16155617])⁵⁴⁹ 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, and are risk for other cold-related injuries, such as hypothermia and snow blindness.

C9.2.1.1. Mechanisms (Pathophysiology) of Frostbite

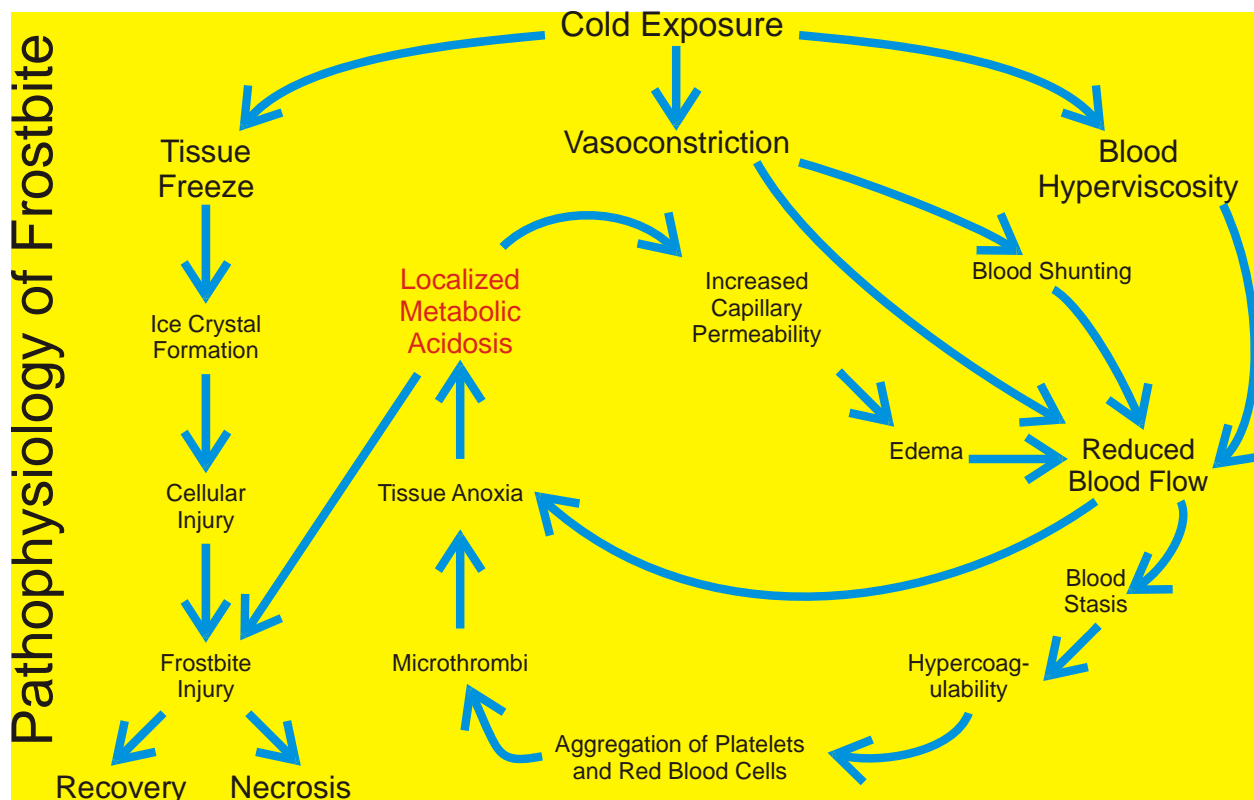
The central pathogenic mechanism is ice crystal formation in tissue, resulting in cellular injury; ensuing and concomitant ischemic anoxia and acidosis contribute to the injury (see [Figure 7](#)). As rewarming (thawing) of frozen tissue allows re-perfusion, vascular permeability and intravascular thrombi 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.

C9.2.1.1.1. Hunting reaction

The hunting reaction (also called the Lewis hunting reaction, the hunting phenomenon, and the hunting response) occurs when extremities are cooled (for example, immersed in water at 41° F to 59° F, or 5° C to 15° C). Vasoconstriction is alternated with vasodilation in irregular repeated sequences in digital blood vessels exposed to cold [Stedman's].⁵⁵⁰ The mechanism may be a cold-induced increased affinity of the post-junctional alpha-adrenoceptors for norepinephrine, leading to vasoconstriction; vasoconstriction leads to further decreased tissue temperature and sympathetic nerve conduction, leading to vasodilatation; vasodilation restores blood flow, nerve conduction is reestablished, and increased affinity of the alpha-adrenoceptors for norepinephrine leads to renewed vasoconstriction [PMID: 6131011].⁵⁵¹ When core temperature is threatened, the hunting response is superseded by vasoconstriction alone [Gonzalez].⁵⁵²

Figure 7 - Pathophysiology of Frostbite



Adapted from Foray J. Mountain frostbite. Current trends in prognosis and treatment (from results concerning 1261 cases). *Int J Sports Med* 1992 Oct;13 Suppl 1:S193-6. Used with permission.

C9.2.1.2. 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].⁵⁵³ A review of cold-

weather injuries among soldiers noted the following frostbite risk factors: Negro race, inadequate clothing, wet clothing, dehydration, inactivity, fatigue, and previous cold weather injury [[PMID: 9433082](#)].⁵⁵⁴ Specific factors associated with increased risk of frostbite include not wearing a hat with earflaps, not wearing a scarf, using protective ointments, having hands and feet that sweat profusely, and being transported in the open or in open vehicles under windy conditions [[PMID: 8541749](#)].⁵⁵⁵ The Marine ski-march leather boot and smoking were related to foot cold injuries in a winter mountain training exercise [[PMID: 11149059](#)].⁵⁵⁶ Smoking has been shown to be a contributing factor in high altitude frostbite, as has peripheral vascular disease and altitude (with greatest risk above 17,000 feet above sea level) [[PMID: 9623370](#)].⁵⁵⁷ A review of peacetime military frostbite injuries showed no risk associated with gender or rank [[PMID: 1361671](#)].⁵⁵⁸

In civilian populations in the United States, homelessness, alcohol use, psychiatric illness, and motor vehicle breakdown have been associated with frostbite [[PMID: 9456445](#)].⁵⁵⁹ Workers with vibration white finger have been found to be at increased risk of frostbite [[PMID: 8318122](#)].⁵⁶⁰

C9.2.1.3. Classification of Frostbite

Traditional classification of frostbite injuries (first, second, third, and fourth degree) is similar to (but not the same as) classification of burns. A recent classification based on early bone scan results and lesion characteristics has been proposed, but has not been widely accepted [[PMID: 11769921](#)].⁵⁶¹

In an investigation of contact cooling of the hand, researchers observed what may have been frostnip or even early frostbite, with severity depending on the finger involved and direct contact with a cold object. Immediately after hand exposure to cold metal objects (-20° F or -28.9° C), finger skin showed white patches that rapidly disappeared on rewarming. Some areas had a burning sensation, associated with diffuse redness with a slightly cyanotic hue. After 24 hours, scattered patches of redness and hardening of skin in contact areas were noted. The little finger tip pad was mottled gray with a pinkish cast surrounded by erythema, painful to slight pressure and not anesthetic, although light touch was diminished. Pink areas of skin blanched with pressure and rapidly recovered pinkness. Peeling skin developed in many areas of metal contact, while blistering developed over the ball of the little finger [Daniels].⁵⁶²

C9.2.1.3.1. Frostnip

This is the mildest freezing injury of the skin, and involves freezing of 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.

C9.2.1.3.2. 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.

C9.2.1.3.3. 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](#)].⁵⁶³ 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), paresthesias, 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 recent review of Army Experience in Alaska [[PMID: 9433082](#)].⁵⁶⁴

C9.2.1.3.4. 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.

C9.2.1.3.5. 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 paresthesias may develop days to weeks later [NAVEDTRA 13147-A].⁵⁶⁵

C9.2.1.4. 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](#)].⁵⁶⁶ 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](#)].⁵⁶⁷

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](#)].⁵⁶⁸ Two-phase technetium-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](#)],⁵⁶⁹ but not in a study of mild to moderately severe frostbite using technetium-99m pertechnetate [[PMID: 11926378](#)].⁵⁷⁰ Successful use of technetium-99m-sestamibi scintigraphy in evaluation of frostbite has also been reported [[PMID: 11822694](#)].⁵⁷¹

Magnetic resonance imaging (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](#)].⁵⁷²

C9.2.1.5. Treatment of Frostbite

C9.2.1.5.1. Rewarming

Initial treatment is immediate removal from cold exposure. If that cannot be done, further treatment (rewarming) should be delayed to avoid 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](#)].⁵⁷³

Rapid rewarming in water (as opposed to gradual rewarming) is the definitive treatment for frostbite [[PMID: 10791170](#)].⁵⁷⁴ Water temperatures from 96.8° F (36° C) [[PMID: 1483773](#)]⁵⁷⁵ to 108° F (42.2° C) [[PMID: 9460447](#)]⁵⁷⁶ have been recommended. Some clinicians add a mild antiseptic to the water, and limit immersion to 20 to 30 minutes twice daily [[PMID: 1483773](#)].⁵⁷⁷ Rewarming may be quite painful and require analgesics and sedatives [[PMID: 9556318](#)].⁵⁷⁸

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 evacuated as soon as possible to a definitive treatment facility, since the extent of injury may not be readily apparent and convalescence is usually prolonged.

C9.2.1.5.2. Rest

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

C9.2.1.5.3. 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](#)].⁵⁷⁹ Dextran is often used to limit edema [[PMID: 1483773](#)].⁵⁸⁰

Thrombolytics have been used in treatment of frostbite [[PMID: 8214384](#)].⁵⁸¹ A report notes a smaller area of damaged tissue using fibrinolytic in combination with other drugs in a clinical setting [[PMID: 2165688](#)].⁵⁸² Limited animal data indicates streptokinase administered within 48 hours of frostbite results in less tissue damage from frostbite [[PMID: 2820216](#)].⁵⁸³

Anticoagulants have been advocated in the treatment of frostbite [[PMID: 8214384](#)].⁵⁸⁴ [[PMID: 1483773](#)].⁵⁸⁵ Heparin has been used, with benefits possibly related to anti-inflammatory effects rather than from anticoagulation [[PMID: 1483773](#)].⁵⁸⁶ A recent series reported positive results using heparin and IV tissue plasminogen activator in treatment of severe frostbite [[PMID: 16394908](#)].⁵⁸⁷

Other anti-inflammatory agents (non-steroidals such as ibuprofen and aspirin) have also been recommended to decrease systemic levels of thromboxane [[PMID: 2243830](#)].⁵⁸⁸ (suspected

to have a role in frostbite tissue damage [[PMID: 7204918](#)].⁵⁸⁹ Several other agents targeting thromboxane and prostaglandin (to inhibit the “arachidonic acid cascade” [[PMID: 3631670](#)])⁵⁹⁰ have been promoted, including Aloe vera [[PMID: 2243830](#)],⁵⁹¹ methimazole [[PMID: 3631670](#)],⁵⁹² and methylprednisolone [[PMID: 3631670](#)].⁵⁹³ Some authors recommend early aspiration of blister fluid [[PMID: 9734425](#)],⁵⁹⁴ on the grounds that the thromboxane and prostaglandin known to play a role in burn pathogenesis have been found in frostbite blisters [[PMID: 7204918](#)].⁵⁹⁵ However, it has not been established that the presence of those substances in frostbite blisters worsen recovery outcome, or that early blister aspiration improves outcome.

Pentoxifylline to increase red blood cell flexibility also has been suggested for use in frostbite [[PMID: 11070801](#)].⁵⁹⁶

Vasodilators (buflomedil hydrochloride, naphthidrofuryl, or ketanserin) have been advocated by some authors, but efficacy in frostbite is not established [[PMID: 1483773](#)].⁵⁹⁷ Similarly, although nifedipine has been useful in treating (and suggested for prophylaxis against [[PMID: 8474715](#)])⁵⁹⁸ Chilblain [[PMID: 2647123](#)],⁵⁹⁹ its efficacy in frostbite is not established in the scientific literature.

Smoking is absolutely prohibited during recovery from frostbite [NAVEDTRA 13147-A].⁶⁰⁰

C9.2.1.5.4. Neurological

Sympathetic blockade may be used to both decrease sympathetic tone and relieve pain (e.g., epidural bupivacaine [[PMID: 1483773](#)]),⁶⁰¹ or for pain relief alone (e.g., continuous epidural morphine [[PMID: 9556318](#)])⁶⁰² [[PMID: 3659437](#)]⁶⁰³ [[PMID: 10459266](#)].⁶⁰⁴ However, effectiveness in improving outcome is not established [[PMID: 7411663](#)].⁶⁰⁵

C9.2.1.5.5. Hyperbaric Oxygen

Hyperbaric oxygen has been successfully used on frostbite [[PMID: 11348755](#)].⁶⁰⁶

C9.2.1.5.6. Surgery

Debridement without anesthesia may be done to help visualization of tissue [[PMID: 1483773](#)].⁶⁰⁷

Surgical amputation, if necessary, should be delayed 60 to 90 days (minimum of 3 weeks), unless sepsis occurs [[PMID: 1483773](#)]⁶⁰⁸ [NAVEDTRA 13147-A].⁶⁰⁹

C9.2.1.5.7. Prevention of Infection

Tetanus toxoid booster is appropriate, if required, as tetanus is a known complication of frostbite [[PMID: 8323232](#)].⁶¹⁰ Prophylactic antibiotics have not been found to prevent wound infection [[PMID: 8356126](#)].⁶¹¹

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](#)],⁶¹² or show signs of infection.

C9.2.1.5.8. 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](#)].⁶¹³

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 anaesthesia (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

Table 11 - Frostbite Rewarming Protocol

C9.2.2. Trench Foot (Trenchfoot)

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.⁶¹⁴ (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,” both 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](#)].)⁶¹⁵

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, and improper technique used to rewarm an injured limb.⁶¹⁶ In addition to military personnel, those involved in adventure tourism and the homeless are at particular risk for trench foot [[PMID: 12363167](#)].⁶¹⁷

Trench foot is a very serious injury that may result in permanent nerve or tissue damage, or, untreated, may require amputation [[PMID: 6115374](#),⁶¹⁸ [CHIPPM TN/02-2](#)].⁶¹⁹ In contrast to frostbite, trench foot injuries that have been rewarmed intermittently during cold exposure may be less severe,⁶²⁰ (hence the need to distinguish between non-freezing and freezing cold exposure injuries).

C9.2.2.1.1. Stages of Trench Foot

Trench foot has three clinical “stages”: prehyperemic, hyperemic, and posthyperemic [[MilDerm](#)].⁶²¹ Symptoms begin with cold, then numbness, paresthesias, 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](#)].⁶²² This is the “prehyperemic stage.”

On or after rewarming, sensation returns proximally first, with paresthesias and burning or throbbing pain. Heat sensitivity is increased. Hypoesthesia, further swelling, and erythema may develop. Mottling and discoloration may appear or increase. This is the hyperemic or inflammatory stage. 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 [[MilDerm](#)].⁶²³

More serious cases have a “posthyperemic stage,” a prolonged post-inflammatory phase involving compromised blood supply. Signs and symptoms include cyanosis, mottling, pain (distal and small joints), hyperesthesia, paresthesia, 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 [[MilDerm](#)].⁶²⁴

Trench foot may result in a 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](#)].⁶²⁵ In severe non-freezing cold injury cases, all nerve populations (myelinated and unmyelinated) may be damaged [[PMID: 9306996](#)].⁶²⁶ 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](#)].⁶²⁷

C9.2.2.1.2. 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](#)]).⁶²⁸ Non-steroidal or even narcotic analgesics may be necessary. Tetanus immunization should be given if not up to date.

Other forms of therapy suggested for frostbite (rapid rewarming, low molecular weight dextran, sympathetic blockade, continuous epidural anesthesia, anticoagulation, and regional sympathectomy) have not been specifically investigated for non-freezing injuries and are not recommended [[MilDerm](#)].⁶²⁹ Diet should have adequate protein. Smoking and other use of tobacco products (or nicotine-containing aids to smoking cessation) are prohibited.

Treatment of posthyperemia is directed at rehabilitation, including physical therapy, exercise, and surgical correction of deformities [[MilDerm](#)].⁶³⁰

C9.2.2.1.3. 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 to return to full duty.

Military personnel who have previously suffered trench foot injury may be at increased risk for future cold injury [[PMID: 1969264](#)].⁶³¹

C9.2.3. Vibration White Finger

Occupational cold exposure may be a contributing factor in the development of vibration white finger [[PMID: 8022312](#)].⁶³² Workers should have adequate hand protection when handling cold objects (see [Contact and Handling of Cold Objects, above](#)).

C9.2.4. 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].⁶³³ It manifests as "episodic vasospastic ischemia of the digits . . . characterized by digital blanching, cyanosis, and rubor after cold exposure and rewarming" [SAM-CD].⁶³⁴ Low blood pressure may be a risk factor for Raynaud's Phenomenon [Creager].⁶³⁵ As noted in the section on vibration white finger ([above](#)), workers should have adequate hand protection when handling cold objects (see [Contact and Handling of Cold Objects, above](#)). 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.

C9.2.5. Cold Agglutinin Disease

Cold agglutinin disease is discussed [above](#). Exposure to cold stress (even without being extreme) may precipitate or exacerbate symptoms. The post-infectious variant is usually mild and self-limited and requires no specific management. However, patients with the idiopathic variety who have acral symptoms must either move to a warmer climate or keep their ears, nose, hands, and feet covered during cold weather [Schrier].⁶³⁶

C9.2.6. Paroxysmal Cold Hemoglobinuria

Paroxysmal cold hemoglobinuria is a rare disorder involving cold-induced signs and symptoms of intravascular hemolysis. Like cold agglutinin disease, it may present as a hemolytic anemia. Episodic hemoglobinuria provoked by cold is reported [[PMID: 2046431](#)].⁶³⁷ Paroxysmal

cold hemoglobinuria may be associated with viral infections, non-Hodgkin's lymphoma, or syphilis [Schrier].⁶³⁸

C9.3. Ocular (Eye) injuries Due to Cold Exposure

C9.3.1. 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].⁶³⁹ It is caused by exposure of the cornea to ultraviolet 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 are 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. Chronic exposure (decades or more) to ultraviolet radiation may cause spheroidal degeneration (Labrador keratopathy) [PMID: 7236572,⁶⁴⁰ PMID: 3778857],⁶⁴¹ and may lead to blindness [PMID: 6478308].⁶⁴²

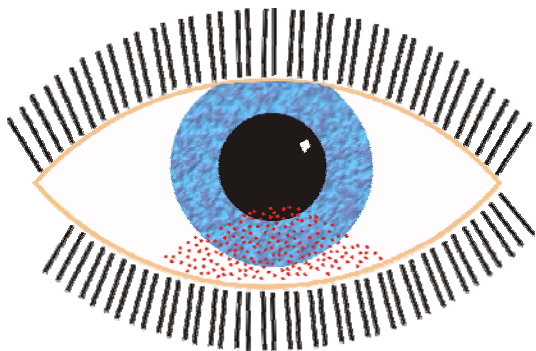
Treatment with a short-acting cycloplegic drop (e.g., cyclopentolate 1% or 2%, or tropicamide 0.5% or 1%, or scopolamine 0.25%, to relieve painful ciliary spasm) and a topical anesthetic given by the health care provider should be adequate for immediate pain relief. (Potent topical anesthetics, such 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].⁶⁴³

Commercially available UV-protective eye drops have not been found to offer adequate protection against solar UV radiation under realistic conditions [PMID: 9704331].⁶⁴⁴

C9.3.2. Cold Keratopathy (Corneal Epithelial Cold injury)

Cold keratopathy is a superficial cold injury of the corneal epithelium. It is commonly found in cross-country skiers competing for long distances in cold weather (e.g., 9.3 miles in 3.2° F or -16° C). More severe cases may cause transient blurred vision. The apparent mechanism of injury is cold exposure (rather than ultraviolet light exposure) of the lower cornea due to incomplete closure of the eyelids. Cases may show punctate red staining (after instillation of Rose-Bengal) of the lower cornea. Contact lenses may help prevent such injury. Spontaneous healing within 24 hours is expected.⁶⁴⁵

Figure 8 - Corneal Staining in Cold Keratopathy



Redrawn from Kolstad A, Opsahl R Jr. Cold injury to corneal epithelium. A cause of blurred vision in cross-country skiers. *Acta Ophthalmol (Copenh)*. 1969;47(3):656-9.

C9.3.3. 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.

C9.4. Hypothermia

Whereas exposure of the extremities or limited areas of skin may cause localized cold stress injuries, exposure of the whole body, or significant portions of the body, to uncompensable cold stress may cause hypothermia. Although hypothermia has been defined as “body temperature significantly below 98.6°F (37°C)” [Dorland],⁶⁴⁶ a more specific definition of hypothermia applicable to clinical practice is the unintentional lowering of body core temperature below 95.0° F (35.0° C) [CDC].⁶⁴⁷ Although hypothermia is a serious cold stress injury, lowered body temperature has limited application in certain medical procedures and treatments. For example, in addition to use during cardiac surgery and neurosurgery, clinical and experimental evidence has suggested hypothermia as an effective therapeutic adjunct in the treatment of decompression sickness [PMID: 7187221].⁶⁴⁸

C9.4.1. Public Health Impact of Hypothermia

During 1979 to 1998, approximately 700 persons (range 420 to 1,024) died annually in the United States from hypothermia [CDC].⁶⁴⁹ During 1999, exposure to excessive natural cold (ICD-10 code X31) was listed as the underlying cause of death for 598 persons in the United States, and hypothermia (ICD-10 code T68) was listed as an injury that occurred to the decedent in 1,139 deaths.

In 1994, excessive cold was reported as the reason for hospitalization for 30 men (27 Army and 3 Air Force) and 14 women (Army) [AMSUS].⁶⁵⁰ The male to female hypothermia ratio is 2.8 to 1 for the U.S. population [MMWR].⁶⁵¹

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].⁶⁵² 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].⁶⁵³

C9.4.2. Symptoms and Signs of Hypothermia

Individuals may not reliably assess whether they are experiencing hypothermia, especially under certain conditions (e.g., during immersion) [PMID: 2803163,⁶⁵⁴ PMID: 3795105].⁶⁵⁵ Symptoms of hypothermia begin subtly with fatigue and loss of concentration [PMID: 1901977].⁶⁵⁶ Ataxia, impaired judgment, oliguria, and slight confusion may be subtle symptoms [PMID: 12092964],⁶⁵⁷ but may progress to stupor, coma, and resemble rigor mortis [PMID: 1901977].⁶⁵⁸ The differential diagnosis in older adults includes cognitive decline, cerebral vascular accident, hypothyroidism, or myxedema coma [PMID: 10024873].⁶⁵⁹ The most important differential diagnosis is death; patients who are cold and could be resuscitated must be differentiated from patients who are cold because they are dead [PMID: 10994374].⁶⁶⁰

In addition to decreased body temperature, neonatal hypothermia signs are lethargy, refusal to feed, coldness to touch, cyanosis, apnea, and pedal edema [PMID: 10957833].⁶⁶¹

Hypothermia shifts the oxyhemoglobin-dissociation curve to the left, resulting in decreased oxygen delivery to tissue [PMID: 16730,⁶⁶² PMID: 9239580,⁶⁶³ PMID: 7984198].⁶⁶⁴ A review article estimated a hematocrit increase of 2 percent for every 1.8° F (1° C) decline in temperature [PMID: 7984198].⁶⁶⁵

As in heat stress, EKG changes may be seen in hypothermia (see [Figure 3 - J Wave Appearance on Electrocardiogram](#)). One investigation of 59 cases of hypothermia reported QT interval (adjusted for heart rate) prolongation in over 70 percent of patients, and bradycardia, J wave, and T wave changes in approximately 50 percent of hypothermic patients; an inverse and significant correlation between J wave voltage and core temperature was noted [PMID: 17027018].⁶⁶⁶

C9.4.3. 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].⁶⁶⁷ 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. For example, a German article categorizes hypothermia as mild (89.6°F to 95°F, or 32°C to 35°C), moderate (82.4°F to 89.6°F, or 28°C to 32°C), or severe (less than 82.4°F, or 28°C) [PMID: 1882213].⁶⁶⁸ At least one author has added another category: extreme (“body temperature below 18-20 degrees C; no recordable EEG activity”) [PMID: 8236180].⁶⁶⁹

Severity	Fahrenheit Core Temperature	Celsius Core Temperature
Mild	90.0° to 95.0°	32.2° to 35.0°
Moderate	82.5° to < 90.0°	28.0° to < 32.2°
Severe	< 82.5°	< 28.0°

Table 12 - Classification of Hypothermia

Hypothermia may be thought of as primary (due to cold exposure) or secondary (related to underlying or predisposing health-related conditions, such as illness, injury, intoxication or extremes of age), and acute (due to cold exposure of less than 6 hours' duration) or chronic (due to cold exposure of greater than 6 hours' duration) [Currier].⁶⁷⁰

C9.4.4. Survival of Hypothermia

A study of survival of hypothermia found that negative survival factors are asphyxia, with an odds ratio (OR) of 30, invasive rewarming methods (OR 20), slow rate of cooling (OR 10), asystole on arrival (OR 9), pulmonary edema or adult respiratory distress syndrome (ARDS) during hospitalization (OR 8), elevated serum potassium (OR 2 / millimole / liter) and age (OR 1.03/year); positive survival factors are rapid cooling rate (OR 10), presence of ventricular fibrillation in cardiac arrest patients (OR 9) and presence of narcotics and/or alcohol during hypothermia (OR 5) [[PMID: 1882213](#)].⁶⁷¹

Factor	Odds Ratio	For (+) or Against (-) Survival
Asphyxia	30	-
Invasive rewarming methods	20	-
Slow rate of cooling	10	-
Asystole on arrival	9	-
Pulmonary edema or ARDS	8	-
Elevated serum potassium (OR given per millimole per liter)	2	-
Age (OR given per year)	1.03	-
Presence of narcotics and/or alcohol during hypothermia	5	+
Presence of ventricular fibrillation in cardiac arrest patients	9	+
Rapid cooling rate	10	+

Based on Locher T, Walpoth B, Pfluger D, Althaus U. Accidental hypothermia in Switzerland (1980-1987)-- case reports and prognostic factors. [German]. Schweiz Med Wochenschr. 1991 Jul 9;121(27-28):1020-8.

Table 13 - Hypothermia Survival Factors

Survival has been reported with a core temperature as low as 58.1° F (14.5° C) in a child [[PMID: 8601876](#)].⁶⁷² Shock, requiring treatment with vasoactive drugs, is an independent risk factor for mortality, while initial core temperature is not [[PMID: 11742934](#)].⁶⁷³ Hyperkalemia may be a useful diagnostic tool in discerning dead from hypothermic victims [[PMID: 10994374](#)].⁶⁷⁴ In urban adult victims of hypothermia, decreased rates of rewarming were associated with increased mortality and infection; rewarming rates seemed to reflect intrinsic capacity for thermogenesis (i.e., hypothermic patients with underlying infection took longer to rewarm and were less likely to survive) [[PMID: 16946289](#)].⁶⁷⁵

Stage of Hypothermia	Organ System				
	Central Nervous System	Cardio-vascular	Respiratory	Dermatologic/Neuro-muscular	Renal/Gastro-intestinal
Mild (Excitation Phase)	Apathy Hyperreflexia Disorientation	Tachycardia Hypertension Increased cardiac output	Tachypnea Bronchorrhhea Bronchospasm	Shivering Vasoconstriction Increased tone	Cold-induced diuresis Decreased GI motility Constipation
Moderate (Slowing Phase)	EEG abnormalities Hyporeflexia Paradoxical undressing	Bradycardia Hypotension Increased atrial arrhythmias	Bradypnea Diminished gag Decreased O2 consumption	Decreased shivering Spasm	Ileus GI erosions Hepatic necrosis/pancreatitis
Severe	Coma Areflexia EEG flattening	Asystole Ventricular arrhythmias	Pulmonary edema Apnea	Rigidity Compartment syndrome	Oliguria Decreased renal blood flow

Reproduced from Chang AK. Chapter 351 hypothermia. In Harwood-Nuss' Clinical Practice of Emergency Medicine. Wolfson AB, Hendey GW, Hendry PL, Linden CH, Rosen CL, Schaider J; Sharieff GQ, Suchard JR, eds. 4th Edition. Copyright © 2005 Lippincott Williams & Wilkins, Philadelphia. Used with permission.

Table 14 - Physiologic Changes During Hypothermia

C9.4.5. Predisposing Factors for Hypothermia

Decreased Heat Production	Increased Heat Loss	Hypothalamic Dysfunction	Iatrogenic Cooling
CNS depression (metabolic or traumatic) Immobility (age, neuromuscular disorders) Endocrine failure (adrenal / pituitary / thyroid) Hypoglycemia / malnutrition	Environmental exposure (inadequate clothing or shelter, wind chill, low humidity, perspiration, wet clothing) Exfoliative skin disease Drugs / alcohol Neuropathy Sepsis Shock Burns	Acidosis / anoxia CNS hemorrhage / infarction Drugs (e.g., phenothiazines) Encephalopathy Multiple sclerosis ^{676,677} Poisoning (e.g., maneb) ⁶⁷⁸ Previous Head Injury ⁶⁷⁹ Prior Cold Injury	Use of large volumes of cool fluids (<95° F or 35° C) for lavage or IV administration Overly aggressive treatment of hyperthermia (“overshoot”)

Adapted from Chang AK. Chapter 351 hypothermia. In Harwood-Nuss' Clinical Practice of Emergency Medicine. Wolfson AB, Hendey GW, Hendry PL, Linden CH, Rosen CL, Schaider J; Sharieff GQ, Suchard JR, eds. 4th Edition. Copyright © 2005 Lippincott Williams & Wilkins, Philadelphia. Used with permission.

Table 15 - Predisposing Factors in Hypothermia

Hypothalamic dysfunction and decreased ability to seek shelter from the cold also may contribute. Sepsis, endocrine dysfunction (diabetic ketoacidosis, hypoglycemia, hypothyroidism, hypopituitarism, hypoadrenalism), central nervous system (CNS) disorders (stroke, brain tumors, spinal cord injury), and skin conditions (burns, erythroderma, psoriasis, ichthyosis) are additional predisposing factors [Currier].⁶⁸⁰

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].⁶⁸¹ History of orthostatic hypotension is a risk factor for developing accidental hypothermia [PMID: 4065579].⁶⁸² Hypothermia subsequent to chemotherapy for Hodgkin's disease with paraneoplastic fever has been reported [PMID: 17240740].⁶⁸³

C9.4.5.1. Ethanol and Medications

Ethanol and drug use may increase heat loss due to cutaneous vasodilatation and impaired shivering; substance abuse may also affect behavior so as to increase cold exposure (see [Alcohol](#), [above](#)).

Pyridostigmine bromide (30 mg) had no significant effect on physiologic responses to cold-water immersion, but limited cold tolerance because of marked abdominal cramping [PMID: 1938714].⁶⁸⁴ Nitrous oxide (30%) has been shown to blunt the metabolic and shivering responses to cold-water immersion [PMID: 1550280].⁶⁸⁵ Triazolam has been found not to significantly affect heat production or core temperature during cold-water immersion [PMID: 7575314].⁶⁸⁶ Naphazoline 0.05-0.1% nasal decongestion solution has been reported as a cause of hypothermia in children [PMID: 8067910].⁶⁸⁷

Hypothermia may be due to nerve agent (sarin) exposure [PMID: 12387297].⁶⁸⁸

C9.4.5.2. 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].⁶⁸⁹ A recent review, including simulation modeling, of the military Extreme Cold Weather Clothing System during cold stress exposure reported that:

- Nuclear, biological, and chemical (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].⁶⁹⁰

C9.4.6. Treatment of Hypothermia

Rapid core rewarming, airway control, and prolonged cardiopulmonary resuscitation have been noted to be key factors in managing the hypothermic patient [PMID: 8121213].⁶⁹¹

Medical management of the older patient with moderate to severe hypothermia may require in-hospital intensive care [[PMID: 10024873](#)].⁶⁹² 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](#)].⁶⁹³

C9.4.6.1. Rewarming Methods

The method used to rewarm a patient with severe accidental hypothermia should be adjusted to the hemodynamic status [[PMID: 8719198](#)].⁶⁹⁴

Of conventional rewarming methods, trunk immersion has been found to have the smallest afterdrop (see [below](#)), shortest recovery period, and most rapid rewarming [[PMID: 7417132](#)].⁶⁹⁵ One article reported that surface and conventional core rewarming methods result in an average temperature increase of 4.3° F (2.4° C) per hour [[PMID: 3729178](#)];⁶⁹⁶ however, it has been claimed that immersion rewarming can increase cardiac temperature at least twice as fast as inhalation rewarming, and inhalation rewarming may increase cardiac (i.e., body core) temperature twice as fast as spontaneous rewarming [[PMID: 6322264](#)].⁶⁹⁷ When hypothermia is associated with cardiac arrest, rewarming by extracorporeal support is recommended (see [below](#)) [[PMID: 8236180](#)].⁶⁹⁸

Inhalation rewarming of mild (body core temperature of 95° F or 35° C) hypothermia with heated, humidified oxygen may be as effective as rewarming by immersion in a hot bath [[PMID: 1180782](#)].⁶⁹⁹ However, one study found no benefit from pre-hospital inhalation and peripheral rewarming for the treatment of mild hypothermia [[PMID: 1854075](#)].⁷⁰⁰

In more severe hypothermia, inhalation therapy is the recommended treatment in the field [[PMID: 7417132](#)].⁷⁰¹ Evaluation of rewarming techniques shows that results from forced air rewarming are equivalent to or better than results from invasive rewarming methods, except for rewarming with cardiopulmonary bypass [[PMID: 11107727](#)].⁷⁰² Severe hypothermia has been managed with warm IV fluids, peritoneal dialysis with warm fluids [[PMID: 8698555](#)],⁷⁰³ and hemodialysis with an average rise of 3.4° F/hour (1.9° C/hour) [[PMID: 11479182](#)].⁷⁰⁴

C9.4.6.2. Afterdrop

Afterdrop is a phenomenon of conductive heat loss. Afterdrop refers to a continued decrease in measured body temperature after removal from cold stress exposure. In one study, surface to volume ratio and body mass index predicted afterdrop duration [[PMID: 11043627](#)].⁷⁰⁵ Afterdrop and hypothermia can occur commonly after recreational cool-water swimming, and some researchers suggest that participants should be observed for signs of temperature decrease following removal from cold stress [[PMID: 11043627](#)].⁷⁰⁶

Afterdrop of core temperature, ventricular fibrillation precipitated by rough handling, hypovolemia, or fluid overload all have been noted as potential contributors to hypothermia deaths [[PMID: 1483774](#)].⁷⁰⁷

C9.4.6.3. Extreme Cases of Hypothermia

C9.4.6.3.1. Cardiopulmonary Bypass

Cardiopulmonary bypass for core rewarming allows circulatory support while avoiding myocardial damage from prolonged external cardiac massage, rapidly increases the myocardial temperature, counteracts myocardial temperature gradients so that direct current cardioversion is successful, avoids "rewarming shock," and improves microcirculatory flow [[PMID: 3729178](#)].⁷⁰⁸ Immediate cardiopulmonary bypass for rewarming is recommended for patients in ventricular

fibrillation with core temperatures below 86° F (30° C) [[PMID: 3729178](#)]⁷⁰⁹ who have no documented contraindications to resuscitation [[PMID: 7984198](#)].⁷¹⁰ Femoral flow rates of 2 to 7 liters per minute may be used in cardiopulmonary bypass rewarming [Long].⁷¹¹ One review noted that with the warmer set at 100.4° to 104° F (38° to 40° C), core temperature will rise at 1.8° to 3.6° F (1° to 2° C) every 3 to 5 minutes [[PMID: 7984198](#)].⁷¹²

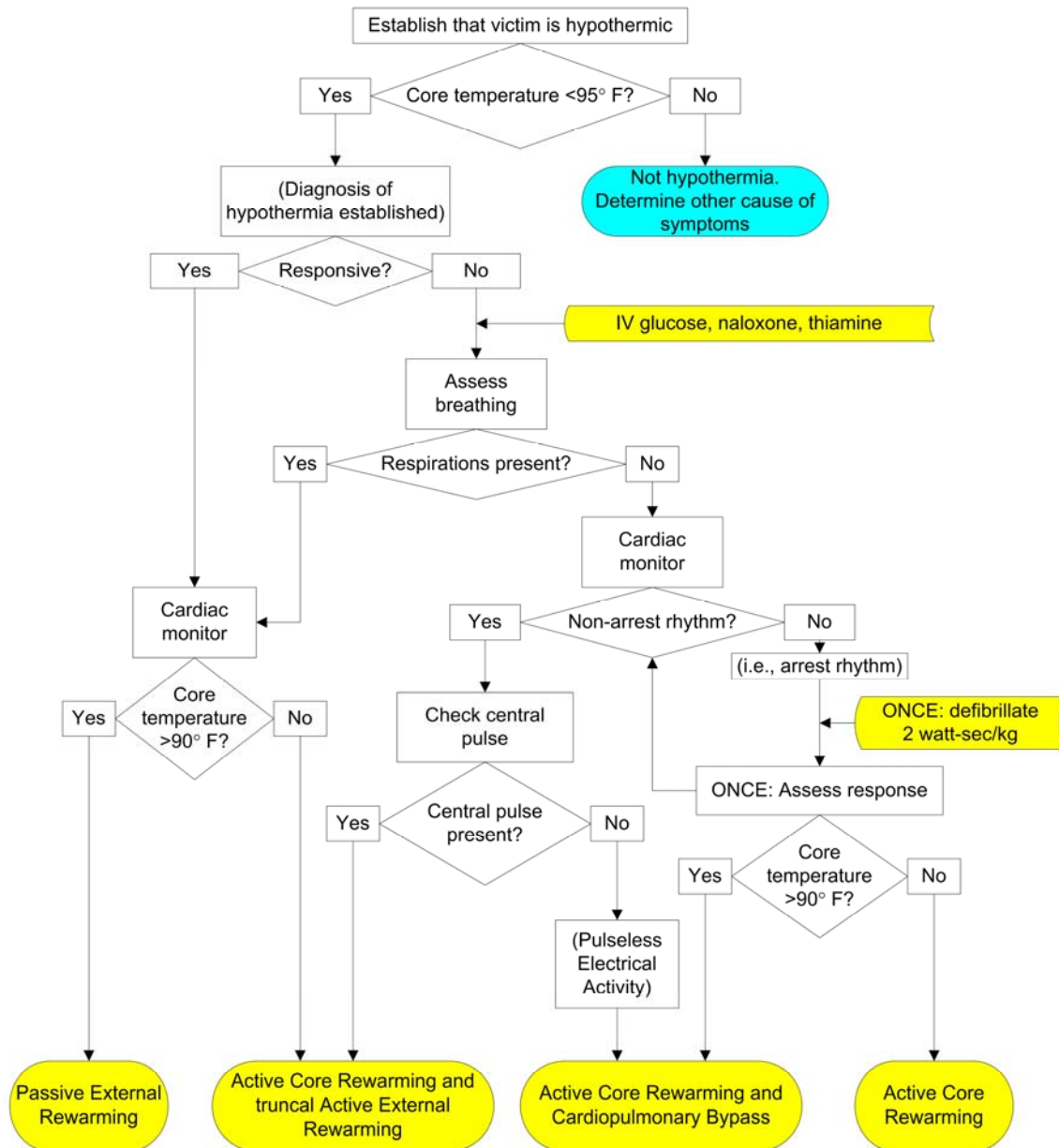
C9.4.6.3.2. Bretylium

Bretylium, no longer generally recommended for treatment of ventricular fibrillation [AHA],⁷¹³ has been found to increase the threshold for ventricular fibrillation in dogs if administered prior to hypothermia [[PMID: 1616517](#)].⁷¹⁴ However, resuscitation of hypothermic dogs in ventricular fibrillation using bretylium was no more effective than using saline [[PMID: 6486552](#)].⁷¹⁵ Bretylium may be of value for prevention of ventricular fibrillation in hypothermia during rewarming; some clinicians think it should be considered a first-line agent for treatment of ventricular fibrillation in hypothermia [Currier].⁷¹⁶

Extremely aggressive measures, including median sternotomy with cardiopulmonary bypass, have been advocated to rewarm pediatric victims of cold-water submersion who suffer severe hypothermia (<82.4° F or 28° C) and cardiac arrest (asystole or ventricular fibrillation) [[PMID: 11886730](#)].⁷¹⁷

Figure 9 - Hypothermia Treatment Algorithm

Hypothermia Treatment Algorithm



C9.4.6.3.3. Triage

In multiple-victim situations, triage may be essential to allocate limited resources (e.g., cardiopulmonary bypass equipment). A study of avalanche victims (in whom asystole is generally due to asphyxia and not primarily to hypothermia [PMID: 8701103])⁷¹⁸ found that plasma potassium (greater than 9 millimoles per liter), pH (equal to or less than 6.50) or an activated clotting time (exceeding 400 seconds) may identify hypothermic arrest victims in whom death preceded cooling (and thus have no chance of resuscitation after rewarming)

[PMID: 8191027].⁷¹⁹ Another study suggested potassium of 10 millimoles per liter as a cut-off [PMID: 8701103].⁷²⁰ However, there is no variable that can with certainty predict successful resuscitation efforts [PMID: 10925611].⁷²¹

C9.4.6.3.4. Potential Complications

Serious complications of hypothermia include hemorrhagic pancreatitis, lung edema and myxomatous skin edema [PMID: 10998830].⁷²²

Survivors of neonatal hypothermia may show severe developmental delay, with abnormal cerebral computed tomography findings, including diffuse cerebral edema, with reversal of the normal density relationship between grey and white matter and a relative increased density of the thalami, brainstem and cerebellum (the “reversal sign”), cerebral atrophy, and multicystic encephalomalacia [PMID: 9634453].⁷²³ Acutely, intracranial pressure monitoring may be important in pediatric victims, especially those who survive drowning [PMID: 3550722].⁷²⁴

Mild perioperative hypothermia is significantly associated with infection of surgical wounds [PMID: 11395189].⁷²⁵ In one report, all 4 surviving victims of hypothermia treated with cardiopulmonary bypass developed temporary pulmonary problems, and 2 developed wound infections; average hospital stay was 21 days [PMID: 3729178].⁷²⁶

C9.4.7. Follow-up of Hypothermia

Follow-up of hypothermia victims may include neuropsychiatric testing and brain imaging studies. Magnetic resonance imaging showed cerebellar atrophy in one patient that received rewarming with cardiopulmonary bypass with mild clinical signs [PMID: 9366581].⁷²⁷

C9.4.8. Prognosis in Hypothermia

The prognosis is excellent in patients in whom no hypoxic event precedes hypothermia and no serious underlying disease exists [PMID: 8719198].⁷²⁸

Out of a group of 32 victims, age 15 to 36 years, of severe hypothermia (core temperature less than 82.4° F or 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].⁷²⁹

C9.4.8.1. Predicting Outcome in Hypothermia

In one report, emergency room Glasgow coma score and trauma score were not indicative of outcome of victims of hypothermia [PMID: 3202789].⁷³⁰

C9.4.8.2. Elderly Victims

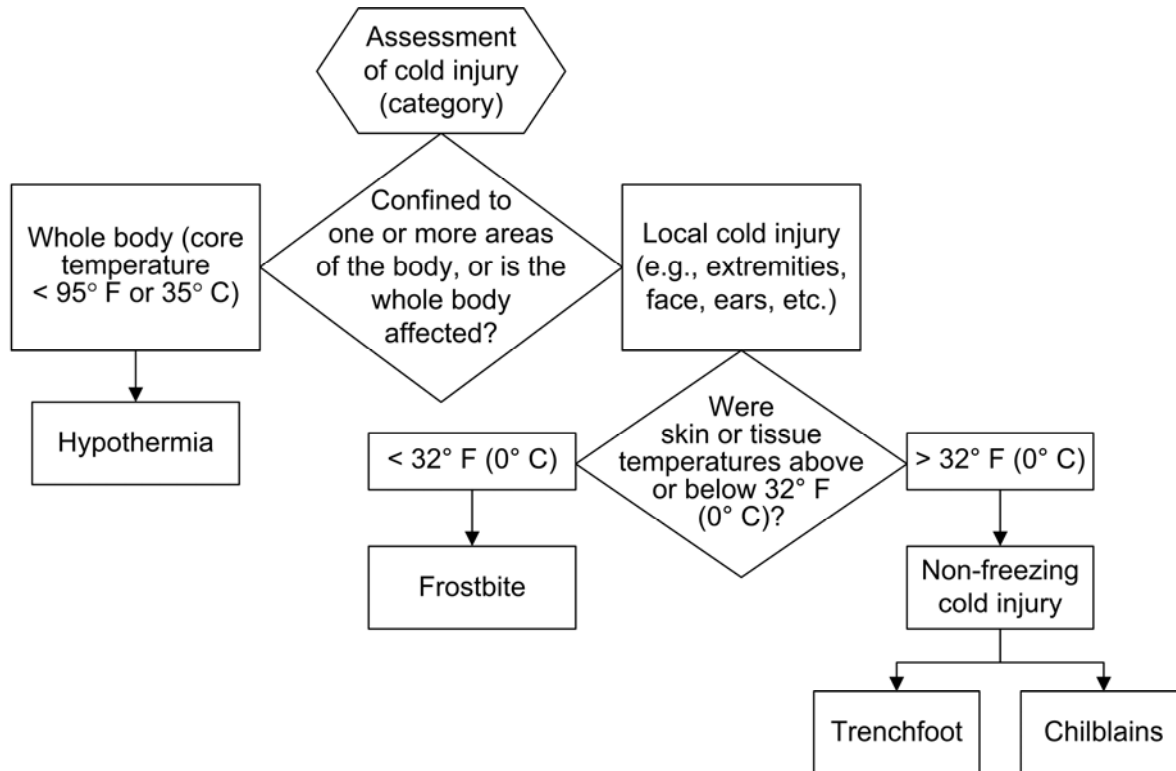
In a series of hypothermia victims aged over 65 years, of those who survived the index admission, the 3-year mortality was 100% in those with primary hypothermia and 24% in those with secondary hypothermia [PMID: 3577949].⁷³¹

C9.4.8.3. Pediatric Victims

In neonates with a birth weight of less than 700 gm, hypothermia is a factor correlating with non-survival [PMID: 6463697].⁷³²

Of 12 survivors of infant hypothermia, by ages 3 to 12 years, one had mild and one had severe psychomotor retardation (both of the latter victims had sepsis on first admission for hypothermia). All hypothermic infants without sepsis had normal developmental achievements [PMID: 10663286].⁷³³ An 8 year old child treated aggressively with open rewarming and resuscitation by thoracotomy and pleural lavage for cardiac arrest with core temperature 77° F (25° C) recovered without any postoperative complications; follow-up at two years showed some neuropsychological defects [PMID: 7857072].⁷³⁴

Figure 10 - Assessment (Category) of Cold Injury



C9.5. Prevention of Further Heat 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 injuries may be appropriate (see [OPNAV 5100.23](#) series).⁷³⁵ 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.

Chapter 10:C10. Reporting

All cold stress-related injuries should be reported through the Naval Disease Reporting System. A simultaneous report to the Naval Safety Center should be made using the Web Enabled Safety System ([WESS](#)). 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 6220.12A](#) (which stipulates Naval Disease Reporting System electronic report, or written, fax, e-mail or phone report to the cognizant NAVENPVNTMEDU).⁷³⁶

Chapter 11:

C11. Heat Stress Injuries Prevention and Treatment

C11.1. Prevention of Heat injuries

Heat stress is commonly encountered in military operations, and may cause injury or death, resulting in decreased readiness. Most heat stress injuries are preventable.

The body constantly generates heat; activity (work or exercise) generates more heat. The body cools itself in heat stress conditions primarily by sweating.

Risk factors and conditions that predispose to heat stress injuries include high humidity, little air movement (wind), overhead sun, elevation below sea level, sunburn or other skin condition, clothing or gear that hinders skin “breathing,” illness, recent immunizations, prior heat injury, recent heat stress exposure, age (very young or very old), lack of acclimatization (not taking a few weeks to “get used to” heat stress), dehydration, medications, substance abuse, fatigue, high activity level, elevated body mass (being overweight, whether obese or very muscular), and being around very hot objects (e.g., hot steel).

Preventive measures against heat stress injury are focused on limiting exposure to heat stress (by decreasing activity or avoiding hot environments), acclimatization (taking 3 weeks to gradually get used to the heat), maintaining adequate hydration and limiting strenuous activity. Water, electrolyte solutions (sports drinks), or even carbonated beverages may be used to replace fluids lost through sweating. “Forced drinking” (i.e., drinking even though not thirsty) during activity in heat stress should be done, as thirst lags behind actual need for water. Water intake should not exceed 1.5 quarts per hour and no more than 12 quarts per day (note: highly conditioned personnel may require even more water than that, and for such people water intake should not be limited; however, 12 quarts per day is very much and is not often required). Activity is controlled by observing work-rest cycles, established based on the wet bulb globe temperature index (WBGT). A specific instrument measures the WBGT; the WBGT is not the same as the temperature in the sun or shade, the “heat index,” or other reading given by weather stations.

Symptoms (feelings of being overheated or of chills) and signs of impending heat stroke (confusion, incoordination, decreased sweating, “gooseflesh”) should be closely followed. Checking body temperature can be done using oral (mouth) thermometers (provided the person has not had hot food or water for 5 minutes and cold food or water for 30 minutes, and can avoid mouth breathing) or ear probes (provided no water has splashed in the ear, and cold or hot air is not blowing into the ear); core temperature may be determined with a rectal thermometer, but accuracy is best with an esophageal thermometer.

Heat injuries may be generally classified as minor (miliaria, heat syncope, heat edema, heat tetany, heat cramps) and major (heat exhaustion, heat stroke). Minor heat injuries generally do not cause permanent injury, but may diminish operational readiness. Major heat injuries have the potential to cause permanent injury or death.

Heat injuries often do not fall into distinct categories. A case of heat injury may exhibit signs and symptoms from more than one diagnosis. Also, a heat injury may progress from mild (for example, heat cramps) to severe (heat exhaustion or even heat stroke) if adequate treatment is not given in a timely manner. Severe heat illness, such as heat stroke, does not have to be a linear progression through various heat illnesses, but can be sudden and explosive.

C11.1.1. Minor Heat Injuries

C11.1.1.1. Miliaria

Heat stress may cause miliaria (“heat rash”), an inflammatory skin reaction characterized by redness, with papules and vesicles at sweat glands. Miliaria can impair sweating and reduce heat tolerance. Treatment is removal from heat stress exposure. Application of cool wet cloths, cool starch baths, calamine lotion, and corticosteroid lotion with or without 0.25% menthol may alleviate symptoms. Antihistamines may inhibit sweating, and should be limited to use for severe itching in air conditioning.

C11.1.1.2. Heat Syncope

Heat syncope (fainting), if present, occurs during the first 5 days of heat exposure, due to vascular insufficiency (low blood pressure). Symptoms are syncope and postural lightheadedness. Victims are tachycardic (rapid pulse), have normal temperatures, are sweating, and have postural hypotension. Treatment is supine position, elevation of the feet, and oral fluids.

C11.1.1.3. Heat Edema

Heat edema is lower extremity edema (swelling) that develops or worsens soon after heat stress exposure (usually within 48 hours). No specific treatment is required, and the condition is expected to resolve with continued acclimatization. In more severe cases, diuretics may be given to enable victims to complete heat acclimatization.

C11.1.1.4. Heat Tetany

Heat tetany is due to hyperventilation after being exposed to heat stress, usually prior to acclimatization. Symptoms include muscle spasm (local or generalized) and numbness and tingling around the mouth. Blood is alkaline and may show hypocarbia. Treatment is temporary removal from heat stress.

C11.1.1.5. Heat Cramps

Heat cramps occur in heat-acclimatized individuals exercising or working in heat stress conditions, especially when continued over several days. Heat cramps are thought to be from hyponatremia (low sodium). Other than muscle cramps (and possibly fatigue), victims generally feel well. Treatment is increased dietary sodium intake.

C11.1.2. Major Heat Injuries

C11.1.2.1. Heat Exhaustion

Heat exhaustion is a major heat stress-related injury, and may be a precursor to heat stroke. It is an inability to sustain required cardiac output. There are two types of heat exhaustion: sodium (salt) depletion and water depletion (dehydration or anhydrotic).

Sodium-depletion heat exhaustion is from exposure to excessive heat stress while consuming sufficient water but insufficient salt. Symptoms are nausea, vomiting, diarrhea, weakness, alterations of mental status, and minimal or no thirst. The victim usually has cool, moist skin that may be sticky and pale. (NOTE: SKIN FINDINGS IN HEAT INJURIES MAY NOT BE CONSISTENT!) The victim often is hypotensive (low blood pressure) and tachycardic with normal body temperature and normal urine volume. Hyponatremia (serum sodium less than 130 mEq/L) always is present, and may result from inadequate dietary salt, excessive sodium

loss in sweat (more likely prior to heat acclimatization), or water intoxication (drinking too much water). Treatment is removal of the victim to a cool place, removing heavy clothes, and administration of sodium replacement (oral or IV fluids of normal tonicity, such as D5NS or Lactated Ringer's Solution) until symptoms clear and pulse and urine findings normalize (including urine sodium content of at least 10 mEq/L). If sodium is very low (less than 120 mEq/L), sodium replacement must be monitored, as it may lead to cerebral edema if done too rapidly. Avoid aspirin and other non-steroidal anti-inflammatory agents. Active cooling (see Cooling, below) may be performed as needed.

Water-depletion heat exhaustion is from excessive heat stress without adequate water replacement. Symptoms are malaise, vomiting, dizziness, confusion, anxiety, agitation, weakness, fatigue, prostration, and even delirium and collapse. Victims are dehydrated and have elevated temperatures (at least 100.4° F or 38° C). Hyperventilation, tetany, oliguria (low urine output), and hypernatremia may be present. Heat stroke may be imminent. Treatment is removal from heat, removal of heavy clothing, active cooling (aggressive cooling if necessary to maintain core temperature less than 102° F or 38.9° C), and emergent transport to a hospital for water replacement. IV fluids replacement should try to correct serum sodium at no more than 2 mEq/L per hour.

C11.1.2.2. Heat Stroke

Heat stroke is a seriously elevated temperature (> 104° F or 40° C) that causes central nervous system (CNS) injury. Heat stroke is a life-threatening heat stress exposure injury. Symptoms and signs of heat stroke include feeling overheated, weakness, fatigue, diarrhea, vomiting, irritability, bizarre behavior, euphoria, combativeness, hallucinations, loss of consciousness, and coma. Sweating may or may not be present. Complications include arrhythmias, shock, rhabdomyolysis (muscle breakdown), renal failure, liver failure, coagulation abnormalities (include disseminated intravascular coagulation), adult respiratory distress syndrome, and death. Treatment is immediate cooling (including removal of clothing) and removal to a hospital (continuing cooling by maintaining ventilation). In addition to active and aggressive cooling (see below), advanced cooling techniques (removal, cooling, and re-introduction of blood) may be attempted. In addition to oxygen, NG intubation, and other general supportive therapy, aggressive medical management is appropriate, possibly including mechanical ventilation, blood purification therapy, and treatment of infections. After recovery, re-exposure to heat stress conditions must be done with caution.

C11.2. Cooling

Active cooling may be done by fanning (anything from a fan to a helicopter downdraft, with or without water or mist), cold or ice water immersion (with intermittent warm air exposure or rubbing the skin to maintain skin blood flow), cold packs (ice packs or ice water slush), cooling blankets (containing a circulating coolant), and cooled nasal air or oxygen. Aggressive cooling may be done using ice water nasogastric (NG) lavage, ice water enemas, and iced peritoneal lavage. While certain individuals may respond to such measures, cold water immersion is considered to be the “gold standard” for rapid body cooling.

C11.3. Reporting Heat injuries

Heat stress injuries must be reported through the Naval Disease Reporting System and simultaneously to the Naval Safety Center using the Web Enabled Safety System (WESS).

Chapter 12:

C12. Cold Stress Injuries Prevention and Treatment

Cold stress exposure can decrease readiness by causing injury, illness, and death.

Predisposing factors to cold injuries are alcohol use, cold-water immersion, clothing that is inadequate or wet, inadequate shelter, using skin lotions or oils as cold protection, handling cold objects (especially metal), wind, high elevation (altitude), dehydration, inadequate rest, poor nutrition, lack of acclimatization, motion sickness, high activity levels followed by rest (causing sweating followed by chilling, as may occur through use of MOPP gear), not removing foot gear and changing socks at least daily, and underlying vascular conditions (i.e., abnormal blood vessel constriction, as may be present with previous cold-related injury, Raynaud's phenomenon, etc.).

Associated injuries may include dehydration, ultraviolet eye or skin injury (sunburn), chapped lips, slips and falls on ice, and high altitude-related conditions.

Most cold-related health consequences are preventable. Preventive measures include ensuring adequate shelter and clothing (especially gear for the face, hands, feet, and head), adequate nutrition and hydration, limiting or avoiding alcohol, gradual acclimatization (taking a few weeks "to get used to the cold"), changing socks at least daily, using proper equipment (such as dry suits for diving, anti-exposure suits on deck, and plastic-coated tools and equipment), taking wind speed and precipitation into account, and exercising sound judgment in cold stress conditions.

Superficial (skin) cold stress injury, such as chilblain, can result in discoloration or scarring. Symptoms of chilblain are burning and itching red patches on the fingers and/or toes, generally bilaterally and exacerbated by sunlight.

Cold stress injuries of acral areas (i.e., extremities, ears, nose), include trench foot and frostbite, which can result in scarring, decreased function, or even loss of the affected body part.

Trench foot is a non-freezing cold injury of the feet. Symptoms of trench foot are cold, numbness, paresthesias, itching, and painful weight bearing, progressing to anesthesia ("walking on blocks of wood"). Signs include pallor, mottled purple coloration, swelling, and edema. After rewarming, sensation returns with paresthesias, pain, and increased heat sensitivity. Blisters, circulatory compromise, local hemorrhage, and ecchymosis may characterize severe cases, which may have a prolonged post-inflammatory phase involving compromised blood supply. Trench foot may result in a peripheral neuropathy.

Frostbite is a freezing injury, classified as first (superficial), second (full-thickness, usually with clear blisters), third (skin and subcutaneous tissue, sometimes with hemorrhagic blisters), and fourth degree (deeper structures, including tendons, muscles, and bone). Symptoms range from pain (cold or burning sensation) to numbness. Signs of frostbite vary and include white patches, diffuse redness, hardening or waxy appearance of the skin, mottled gray coloration, tenderness, diminished light touch, and anesthesia. Determining depth of injury may take days. Delineating tissue viability may take months; technetium bone scanning (bone scintigraphy) may be used.

Whole-body cold stress can result in hypothermia, which, if severe or if not treated in time, can result in organ damage or death.

Cold stress may also exacerbate or unmask underlying conditions, such as acrocyanosis, rosacea, cold agglutinin disease, cold panniculitis, xerosis, cold-induced urticaria, vibration white finger, Raynaud's phenomenon, and paroxysmal cold hemoglobinuria. Persons with those

conditions should take extra precautions to wear adequate clothing and equipment, or avoid cold stress exposure entirely.

Treatment of cold injuries is rewarming. Affected parts should be elevated. Use (movement), weight-bearing, and rubbing should be avoided. Smoking is prohibited until recovery is complete.

Chilblain and trench foot injuries are best rewarmed using air at room temperature. Rewarming may be done even though re-exposure to cold is anticipated. Treatment of chilblain may include nifedipine, thyrocalcitonin, and hemodilution.

Rewarming of frostbite should be delayed until the victim has been removed from risk of re-exposure to the cold and can be kept at bed rest. Treatment of frostbite is rapid rewarming, using immersion in water at or just above body temperature (no more than 108° F or 42.2° C). Additional measures include IV fluids (to maintain hydration and circulation), dextran (to limit edema), thrombolytics (fibrinolytic, streptokinase within 48 hours), anticoagulants (heparin), anti-inflammatory agents (non-steroidals such as ibuprofen and aspirin), sympathetic blockade (epidural bupivacaine or morphine), hyperbaric oxygen, debridement without anesthesia (i.e., to avoid removing innervated, viable tissue), and delayed (60 to 90 days) surgical amputation, if necessary. Prevention of infection should include tetanus toxoid booster, if required, and may include adding mild antiseptics to whirlpool baths. Signs of infection, two-point discrimination, vibration, and movement examinations should be followed closely during frostbite treatment.

Hypothermia treatment includes rapid core rewarming, airway control, and prolonged cardiopulmonary resuscitation (if necessary). Heated, humidified oxygen (when available in the field) may be used alone or in conjunction with or replaced by trunk immersion, warm IV fluids, peritoneal dialysis with warm fluids, and hemodialysis. When hypothermia is associated with hemodynamic instability (e.g., cardiac arrest), rewarming by extracorporeal support (cardiopulmonary bypass) is recommended. Afterdrop (a continued decrease in core temperature after removal from cold), ventricular fibrillation, hypovolemia, and fluid overload are potential (and potentially deadly) complications of aggressive rewarming.

Cold stress injuries must be reported through the Naval Disease Reporting System and simultaneously to the Naval Safety Center using the Web Enabled Safety System (WESS).

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