INTRODUCTION
In attempts to master the environment, we have circumvented our phylogenetic heritage as a tropical mammal by developing a sophisticated technology to control the temperature, humidity, and pressure of our immediate surroundings. For the Seabee, operational emergencies and enemy action remain ever-present hazards. Situations may develop almost instantaneously forcing them to survive extremes of temperature. Seabees must frequently live and work under adverse environmental conditions, and maintenance of their functional effectiveness both ashore and afloat is an area of concern to the operational physician. The physician needs to understand the operational, clinical, and preventive medicine aspects of environmental thermal stress.

The response to environmental extremes of temperatures has been of interest for a long time. The response to climate has influenced geographical migrations and cultural development. Before commencement of modern tropical and polar explorations and subsequent military operations, an individual's ability to lead a healthy and productive life in such environments was problematic. Temperature extremes have physiological and psychological effects. This chapter will consider primarily the former.

Mammals are homeothermic organisms and maintain the internal body or "core" temperature within a minimal range of variation, independent of ambient temperature. This contributes to the normal pattern of human biochemical reactions. The biochemical reactions responsible for metabolism impose several requirements upon the mechanisms regulating body temperature. Since most biochemical reactions are effected through the action of enzymes, which are particularly sensitive to temperature change, a fall or rise of only a few degrees in core Temperature may so retard metabolism that normal behavior is impossible and death may ensue. In the course of biochemical oxidation, heat is liberated; and unless provision is made for its dissipation, overheating of the body may occur. This could cause death as a result of irreversible damage to the various enzymatic systems and cells of the central nervous system. It is mandatory, therefore, that thermal equilibrium of the body be maintained.

THERMAL EQUILIBRIUM
Thermal equilibrium is preserved by the body's ability as a whole to alter its rate of heat production and heat loss. Body temperature is a measure of heat content or storage. A fall in body temperature indicates a decrease in the total heat content of the body, while a rise denotes an increase. Although the core temperature varies normally over a range of only a single degree, the variation in temperature of the exposed portions of the body reflects its continual effort to achieve equilibrium. First, the rate of heat production and then, the rate of loss are altered by environmental or physiological changes. A normal sized, unclothed human at rest in a postabsorptive state can, with minimum body effort, reach thermal equilibrium at a room temperature of 86°F (30°C). Under such conditions, the individual retains only that amount of heat formed by the basic metabolic processes which comprise normal storage. The remainder is
lost to the environment without utilization of any of the reserve mechanisms of heat loss. The preceding example is only one instance of countless states of thermal equilibrium. Its significance lies in the fact that no reserve mechanisms of heat production or heat loss are required to achieve equilibrium. Under different conditions of clothing, activity, or environment, additional physiological adjustments would be needed to maintain an equivalent state.

Another expression of thermal equilibrium has been designated as the "comfort zone." The comfort zone may be grossly defined as the set of environmental conditions which causes neither sweating nor shivering. A more precise specification is possible through reference to the Effective Temperature (ET) Scale, which has become an accepted index of environmental comfort.

A scale of effective temperature may be presented as a family of curves formed by a plot of all combinations of relative humidity and temperature that yield the same subjective sensation of temperature (Figure 1). It is generally agreed that the optimum comfort range for persons wearing normal indoor clothing is +65°F ET to +73°F ET. As can be seen, this corresponds roughly to a temperature range between +70°F and +80°F with relative humidity between 40 and 60 percent. Although in practice the Effective Temperature Scale has been found workable, it should be noted that the stated comfort range will be altered as a function of any variation in amount of clothing worn, level of physical activity, and with the introduction of the factor of air movement.

Figure 1. Scale of effective temperature

Heat Transfer
Heat is transferred to and from the body by several different physical processes: radiation, conduction and convection, and the vaporization of water.

Within the body, heat is transferred from the core to the shell or skin surface primarily by conduction. The agent of transport is the circulating blood. Therefore, thermal regulation is closely related to the regulation of peripheral circulation through the cutaneous vascular bed. The extremities represent about 65 percent of the total body surface. At certain times, as much as ten
percent of the total blood volume of the body may be located in the skin, within a surface layer only two millimeters thick (Krog, 1974). The mechanisms by which circulatory control is accomplished either to preserve or dissipate heat are many and complex. These mechanisms are mediated both centrally and locally and include vascular dilation and constriction, arteriovenous shunts, cold-induced vasodilatation, counter-current exchange, and cold adaptation. The existence, as well as the relative importance, of some of these mechanisms, however, has been challenged.

Radiation. Radiation is the transfer of heat from the surface of one object to another without physical contact between the two. The magnitude of heat loss in man is directly dependent on skin surface area and the average temperature gradient between the skin and surrounding objects. The heat loss from radiation varies widely with environmental conditions. In a temperate climate, a resting individual, wearing ordinary clothes, loses about 60 percent of heat production by radiation. At a temperature of 90°F, this loss may drop to zero. Conversely, at subzero temperatures, heat loss by radiation may reach levels higher than 60 percent.

Conduction and Convection. Conduction and convection are less important methods of heat loss in temperate climates but assume major roles in polar climates. By conduction, the cold air is warmed by immediate contact with the skin; then the skin is warmed; the heated molecules move away, and cooler ones approach to take their places. These in turn are warmed, and the process perpetuates itself. The air movements constitute convection currents. Any process, such as wind, which tends to increase the rate of movement of the ambient air relative to the skin surface, intensifies heat loss. Siple and Passel (1945) have incorporated the phenomenon into the concept of windchill. A unit of windchill is defined as the amount of heat that would be lost in an hour from a square meter of exposed skin surface which has a normal temperature of 91.4°F. Given a hypothetical situation wherein the wind velocity is 20 miles per hour and the temperature is 34°F, reference to the windchill chart (Figure 2) discloses that at the given wind and temperature conditions of the hypothetical situation, the rate of cooling of all exposed flesh is the same as at minus 38°F with no wind. It is easily concluded that under the climatic conditions observed in polar operations, conduction and convection are significant causes of heat loss and potential contributory factors in the causation of cold injuries.

Heat loss by conduction also occurs from transfer of heat to tidal air as it is warmed in the respiratory passages and lungs, to water and foodstuffs taken into the gastrointestinal tract, and to waste materials (urine and feces) as they are eliminated.

Vaporization of Water. Vaporization of water removes heat from the skin surface and the moist mucous membranes of the respiratory epithelium. When one gram of water is converted into water vapor, 0.58 kilocalories of heat must be supplied from the surroundings for the conversion to occur. Although the actual amount of heat loss depends on the ambient relative humidity, in Antarctica, where humidity is very low, respiration alone may account for ten percent (375 kcal)
of an individual's total daily heat loss. Insensible perspiration accounts for an additional loss of about 400 kcal.

Loss of heat by vaporization of perspiration from eccrine sweat glands may account for a large part of the total heat loss at temperatures of 93°F to 95°F, or above. In polar climates, vaporization of perspiration assumes importance only under certain clothing conditions which will be discussed later. It can be seen, then, that loss of heat from the body occurs primarily at two surfaces, the skin and the epithelium of the respiratory system. Under constant environmental conditions, the amount of heat loss depends upon surface area, temperature gradient, humidity, vapor pressure gradient, and the rate of airflow over the surface.

Heat Regulation in Cold Environments
Physiological Mechanisms to Diminish Heat Loss. Some of the physiological methods used to diminish heat loss in lower mammals are unsuitable or impractical for man. Such procedures as rolling up into a ball and thereby markedly decreasing the area of exposed skin, or diminishing heat loss from vaporization of water at respiratory surfaces by cessation of panting, obviously have little application to human beings.

Since approximately 80 to 85 percent of heat loss occurs from the body surface, any reduction in skin temperature should conserve body heat. Changes in the temperature of body surfaces are mediated through the activity of three physiological mechanisms. Unfortunately, one of the three mechanisms, although of some value in retaining body heat, is of greater importance when heat dissipation is desired.

Figure 2: Windchill nomogram.

The first mechanism depends upon the means by which heat is transported from the depths to the
surface of the body. Blood is 80 percent water by volume; because of the water’s high heat capacity, circulating blood is the primary source of heat transfer to the body surface. The total amount of heat brought to a given area is a function of the rate of blood flow through the area. If the rate of flow is retarded by local constriction of the superficial arterioles, the total heat transfer from blood to skin tends to be small and the skin remains cool, thereby decreasing the temperature gradient between it and the surrounding cold air. Although this physiological mechanism is an important means of heat conservation, a certain minimum blood flow must be maintained through the skin to prevent localized anoxia and cellular death.

The second mechanism is dependent on the phenomenon of horripilation, or erection of body hair, which increases the thickness of the layer of nonconducting air entrapped between the hairs. Since surface temperature depends upon the ease with which heat is transferred from the body to the environment, horripilation affords a satisfactory method for retarding conduction by reducing the temperature gradient between the skin and environment even though the skin temperature may remain at a high level. Due to evolutionary processes, humans are poorly equipped to take advantage of horripilation. We substitute a different insulating material, clothing, for hair. A single layer of clothing limits heat exchange by replacing the single temperature gradient between a nude subject and the environment with three such gradients. One of these three exists between the skin and the inner surface of the insulation, another exists between the outer surface of the insulation and the environment, and the third gradient is found between the inner and outer surfaces of the insulation. The effectiveness of clothing in decreasing heat loss is proportional to the magnitude of this third gradient, which in turn depends upon the nature and thickness of the nonconducting substance.

A third mechanism modifies the temperature of body surfaces by varying the amount of moisture available for vaporization. Since insensible heat loss due to perspiration is not subject to wide variation in cold climates, the primary value of this mechanism is in the dissipation of body heat rather than its retention.

**Heat Production.** When heat loss exceeds heat production in spite of utilization of the previously discussed physiological mechanisms, the body, in an effort to regain thermal equilibrium, increases heat production. Heat production is essentially chemical in nature and is developed from at least two different sources. When body temperature decreases in a resting human exposed to cold, involuntary muscular contractions (shivering) ensue. Since only 25 percent of the energy liberated by chemical changes in contracting muscle is converted to work, heat production is equivalent to three or four times that of the muscle at rest. Even more efficient in heat production than isotonic involuntary exercise is voluntary isometric exercise (contraction of both extensors and flexors simultaneously) which converts all of the energy produced to heat.

Various investigators in animals have demonstrated another source of heat production that does not involve skeletal muscle contractions. Small experimental animals like the rat are able to vary
their rate of heat production within several hours by some mechanism in which the hormones of
the thyroid gland, the adrenal cortex, and possibly the adrenal medulla participate without any
detectable change in either voluntary or involuntary muscular activity (Davis, 1963). This
nonshivering thermogenesis is produced by increased tissue sensitivity to norepinephrine, and the
small amount of subcapsular brown tissue plays an important role in this mechanism. Recent
research suggests that other tissues' increased heat production in the cold acclimatized animal is
mediated through an intermediate mechanism in the brown fat tissue. Although Davis (1963)
suggested that nonshivering thermogenesis also exists in humans, humans have no brown fat
tissue; and nonshivering thermogenesis does not seem to occur to any significant extent in
mammals larger than the rabbit (Jansky, 1969).

Those regulatory responses discussed above can succeed in maintaining thermal balance for hours
within the compensable zone. Figure 3 shows the approximate limits of the compensable zones
on both sides of the comfort zone. When temperatures fall below the compensable zone for cold
tolerance, thermoregulation fails and extraneous methods must be utilized to maintain thermal
balance.

Figure 3: Comfort and compensable zones.

Heat Regulation in Warm Environments
Physiological Mechanisms. As the body becomes heated, whether it is from exposure to a high
temperature environment, physiological exercise, or a combination of both, superficial
vasodilatation occurs, thereby increasing blood flow to the skin. This allows additional heat to be
lost primarily by convection and radiation. Furthermore, heat is lost through insensible
perspiration and respiration. When the body becomes heated beyond the limits of the comfort
range, these processes are insufficient to maintain thermal equilibrium.

A second response to heat exposure is an increased rate of excretion by the eccrine glands.
Through the evaporation of water produced by sweating, a large increase in the rate of heat loss can be realized. Although the body can produce sweat at a high rate, evaporative heat loss is limited by the physical process of evaporation, which, in turn, depends upon skin temperature, water vapor pressure gradient, and movement of the air surrounding the individual. The vapor pressure gradient is defined as the difference between the water vapor pressure at the skin and in the surrounding air.

Effects of Clothing. Thermal comfort and stability are modified considerably by the clothing an individual wears. With suitable clothing, a person can achieve a level of comfort and survival over a much greater range of environmental temperatures than those shown in Figure 3. Heat transfer through clothing is a function of the thermal resistance of the clothing and the temperature and humidity differential between the inner and outer surfaces. Thermal resistance of clothing is expressed in terms of "clo" units. One clo is defined as the equivalent to normal indoor clothing and is that clothing insulation required to keep a resting or sitting man with a metabolism of 50 Kcal per square meter per hour indefinitely comfortable in an environment of 69.8°F (21°C) with an air movement of 20 feet per minute, and with a relative humidity of less than 50 percent (Kerslake, 1965). The insulation value of clothing is a function of the air trapped between its fibers and is roughly equal to four clo per inch thickness of fabric.

The biophysics of clothing is interdisciplinary (physiology, psychology, physics, clothing design, and textile science) which relates human work efficiency and comfort to a specific task in a particular environment. Prior to this realization, the physiologist was consulted for information about the human, and the climatologist was sought out for knowledge of the environment. Little was known about the physics of how clothing materials interacted with each other or with the complex human-clothing-environment system. Most experimental measurements utilized "steady state" conditions for simplicity and the reduction of variables. Utilization of the biophysical approach should result in an end product clothing system in which human, environment, and clothing are united into a functional and comfortable whole.

In a cold environment, an individual frequently find themselves wearing more insulation than needed during work and less than needed at rest. This is readily explained by considering the biophysics of the situation. For maintenance of thermal equilibrium in any given environment, the optimum insulation is five to six times as much at rest as at work. The problem, then, is to design clothing which optimizes the balance between static and dynamic insulating efficiencies. Specifically, the rate of heat loss must be minimized when activity level is low and increased when activity level rises.

Primary functions of protective clothing are to ensure adequate ventilation for the escape of both insensible and sensible perspiration, and to provide, around the body, an insulating zone of dead airspace which is compartmentalized in sufficiently small pockets so that currents of air will not be set up by movements of the body and thus disperse heat.
Polar explorers have cautioned repeatedly against the danger of sweating profusely because during later periods of diminished activity, excessive heat loss occurs when the vaporized perspiration condenses on the cold outer cloth, thereby permitting direct heat transfer by conduction. Because retention of vaporized perspiration in clothing diminishes the effectiveness of the sweat mechanism in cooling the skin surface, increased production of perspiration ensues and a potentially dangerous situation develops.

Conversely, in hot environments, clothes become a barrier to the evaporation of perspiration from the skin because sweat evaporated from wet clothing is much less effective in removing heat from the body than moisture evaporated directly from the skin.

Figure 4 illustrates the interaction of clothing insulation, activity, and ambient temperature in the maintenance of thermal balance.

**Figure 4: Prediction of required insulation.**

![Diagram](image_url)

**Hypothermia**
When heat loss exceeds heat production, hypothermic injury may develop. Hypothermia is classified as general or local, depending on whether the injury affects the individual as a whole or affects only a particular part of the body.

**General Hypothermia**
Accidental general hypothermia may result from total or partial immersion in cold water or from exposure to cold ambient air temperatures alone. Almost four-fifths of the world's surface is covered with water, and apart from the equatorial regions, the temperatures of the world's seas are substantially below body temperature. Deck personnel and particularly aviators who fly above these waters are at risk of being placed precipitously into the cold sea and sustaining the physiological insults imposed by accidental immersion. Local cold injury is less a threat of to deck
and shore station crews who must work outside in very cold weather or under conditions of a high windchill index. They are usually prepared and adequately clothed to resist the deleterious effects of exposure to cold air. Immersion in cold water is different from exposure to cold air because of two physical properties of water:

1. The thermal conductivity of water is approximately 26 times greater than that of air. Therefore, during immersion, heat is conducted away from the body at 26 times the rate it would be in air.

2. Water has a specific heat approximately 1,000 times that of air. Therefore, each cubic centimeter of water in contact with the skin can extract and hold 1,000 times more heat from the body than a comparable volume of air for any given increase in temperature.

The rate of heat loss from the immersed body is, therefore, a function of the temperature differential between the skin and the water immediately adjacent to it and the rate of heat transfer from the body core to the skin.

Etiology. The most important factors in determining the rate of onset and the depth of the hypothermia are the water temperature and duration of immersion. In 1946, Molnar, in his classical paper, emphasized the relationship between survival times and water temperatures below 59°F (15°C). Immersion in water at 28 to 35°F (-2.2°C to +1.7°C) may result in unconsciousness in five to seven minutes and in death in ten to twenty minutes. The ungloved hand becomes useless in one to five minutes due to loss of tactile sensation. Figure 5 illustrates the effect of variations in water temperature on the rate of fall of body temperature.

**Figure 5: Rectal temperature as a function of various water temperatures.**
Another factor influencing the rate of onset of hypothermia is the amount of water movement relative to the surface of the immersed body. Currents, turbulence, and body movement each cause the water molecules next to the body surface to be exchanged more frequently, thereby promoting increased heat loss due to conduction and convection. In water at 41°F (5°C) for twelve minutes, it has been found that moderate work doubled the rate at which rectal temperature fell because of increased blood circulation. Working as hard as possible only slightly decreased the rate of temperature loss at this temperature. Collis (1976) determined that survival time could be increased by about one-third by holding still in the water instead of swimming. Infrared thermograms taken of individuals who had remained nearly motionless while immersed demonstrated the areas of greatest heat loss to be the inguinal and thoraco-axillary areas. Collis developed a position called HELP (heat escape lessening posture) for survivors in the water by themselves in which the knees are drawn up to the chest and the arms are clasped together at the chest. Survival time in 50°F (10°C) water proved to be four hours, or double the survival time of a swimmer in the same temperature water. Another significant finding from the series of 5000 immersions in water with temperatures ranging from 39°F (4°C) to 59°F (15°C) was that the drownproofing technique of flotation resulted in a cooling rate 50 percent faster than that observed in subjects treading water with the head above the surface.

A factor which may influence the rate of onset of hypothermia is the presence of body fat. Keatinge (1960) described a linear relationship between fall in rectal temperature and the reciprocal of the mean skinfold thickness in men. It might be expected then that, all other factors being equal, females, who tend to have a higher total percentage of body fat than males of similar height and weight, should have a slower cooling rate on immersion than males of similar height and weight. Golden and Hervey (1972) demonstrated that this is not the case (Table 1) and that the overall rate of cooling of unclothed individuals depends on a complex interplay of many factors, notably heat production, body size, and fat insulation.

Table 1: Skin fold thickness and cooling rates in males and females.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>% Fat (skin fold thickness)</th>
<th>Subject</th>
<th>Mean cooling rate °C/hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wendy</td>
<td>F</td>
<td>30</td>
<td>Andrew</td>
<td>10.7</td>
</tr>
<tr>
<td>Christine</td>
<td>F</td>
<td>20</td>
<td>Christine</td>
<td>9.1</td>
</tr>
<tr>
<td>Brenda</td>
<td>F</td>
<td>18</td>
<td>Brenda</td>
<td>5.6</td>
</tr>
<tr>
<td>Stewart</td>
<td>M</td>
<td>17</td>
<td>Tim</td>
<td>5.1</td>
</tr>
<tr>
<td>Ian</td>
<td>M</td>
<td>16</td>
<td>Will</td>
<td>4.4</td>
</tr>
<tr>
<td>Tim</td>
<td>M</td>
<td>14</td>
<td>Stewart</td>
<td>4.0</td>
</tr>
<tr>
<td>Will</td>
<td>M</td>
<td>14</td>
<td>Ian</td>
<td>3.1</td>
</tr>
</tbody>
</table>
The importance of protective clothing as a factor in the etiology of hypothermia has been demonstrated experimentally by many investigators and is confirmed by accounts of survivors. The mechanism of protection at its most basic level, and with only a single layer of clothing is to reduce the rate of exchange of the water molecules immediately adjacent to the skin.

Physiology of Immersion Hypothermia. In an excellent review article, Golden (1974) summarized the physiological changes seen as a result of immersion hypothermia. A precise understanding of the physiological changes in humans is hampered by lack of hard data. The majority of the literature describes either the results of experimentally induced, relatively mild states of hypothermia or anesthetic hypothermia in which shivering was abolished and respiration controlled. With the exception of the Dachau experiments during WW II, most case studies relate instances of hypothermia which had a relatively long duration of onset. Nevertheless, the understanding of physiological mechanisms in humans has been extended by cautious extrapolation from experimental findings in animals.

Metabolism. Immediately following cold water immersion, the body attempts to maintain its thermal integrity, but the rate of heat loss exceeds even the most violent efforts of the body to increase metabolic heat production through exercise. Involuntary shivering reaches a maximum at a core temperature of 95°F (35°C), but it declines thereafter to be replaced by a rigidity of muscles during the range of 91.4°F to 86°F (33°C to 30°C), which, in turn, is abolished around 80.6°F (27°C). An extrapolation from animal data would indicate that the relationship of oxygen consumption and body temperature is almost linear, and in dogs, oxygen consumption at 20°C was only 15 percent of normal. Since oxygen consumption in humans is largely dependent on the shivering response, all evidence suggests that the increase in metabolic rate with cooling does not increase below a core temperature of 95°F (35°C).

The hyperglycemia resulting from hypothermia was first described by Claude Bernard. It was also observed in the Dachau experiments in which it was found that the blood sugar level was a mirror image of the rectal temperature. In the presence of shivering, blood glucose levels rise if there is an ample supply of carbohydrates. But as metabolism declines with decreasing body temperature, glucose utilization diminishes, and the early hyperglycemia continues. Where there is no shivering during cooling, such as in anesthesia, the blood glucose level is maintained or decreased. Therefore, in an individual whose hypothermic state was rapidly induced (acute), hyperglycemia should be observed, while in those individuals who developed hypothermia slowly (chronic) or while expending energy at high rates (subacute), the blood sugar level should be normal or subnormal. At temperatures below 86°F (30°C), glucose is metabolized slowly if at all, and insulin's effect on glucose transport across the cell membrane is significantly impaired. There is an immediate significant increase of free fatty acids in response to cold exposure which is still demonstrable eight hours after exposure. This suggests that lipids rather than glucose are the
preferred source of energy in hypothermia.

**Respiratory System.** Hyperventilation with respiratory rates of 60 to 70 per minute is seen as a result of the initial shock of entry into cold water. There is a marked reduction in end tidal PCO$_2$, but it gradually returns to a little above the original level. This initial hyperventilation results in a reduction of arterial CO$_2$ which causes a dramatic rise in pH, but as blood temperature falls, CO$_2$ solubility increases and the arterial pH, falls. During rewarming, this acidosis, which is due in part to a metabolic component, may intensify, and the pH may fall as low as 7.1. The shift to the left of the oxygen dissociation curve resulting from the decrease in temperature is counteracted in part by a shift to the right due to lowering of pH. Respiration becomes progressively depressed as hypothermia deepens, and at near lethal core temperatures, it is extremely difficult to detect.

**Cardiovascular System.** The cause of death in hypothermia is almost always cardiac in origin. There is an initial stimulatory phase during which cardiac rate increases dramatically and central venous pressure rises. During this period, a marked peripheral vasoconstriction occurs. As the hypothermic condition deepens, cardiac rate decreases due to a direct effect of cold on the pacemaker, and cardiac output decreases as a direct consequence. Duration of systole increases, and the refractory period of the atrioventricular bundle is increased. Arrhythmias are common in hypothermia. Extrasystoles are commonly seen on cold water entry. Atrial fibrillation appears to be more a feature of acute hypothermia and has been described in Dachau victims, World War II immersion survivors, in surgical patients with induced hypothermia, and even in an English Channel swimmer. Atrial fibrillation usually occurs at a body temperature of 91°F (33°C). As temperature falls to 82.4°F to 77°F (28°C to 25°C), there is a sharp increase in the incidence of ventricular arrhythmias, ectopic beats, and A-V dissociation, and if the heart is mechanically stimulated at these temperatures, development of ventricular fibrillation is likely to occur.

In humans, death due to cardiac arrest appears to occur between 78.8°F and 75.2°F (26°C to 24°C); however, there are documented cases of accidental hypothermia victims surviving core temperatures of 64.4°F (18°C). The nature of terminal cardiac arrest has not been precisely described. Extrapolations from animal experiments would suggest that when the heart is not mechanically stimulated, arrest is due to simple asystole, but when irritation occurs, ventricular fibrillation is the cause of death.

Other cardiovascular physiological changes include an increase in stroke volume but a reduction in cardiac output due to the slowing in rate. Central venous pressure increases; blood pressure falls, and there is a progressive reduction in total peripheral resistance at temperatures below 86°F (30°C). Although coronary blood flow is reduced, it is sufficient for the needs of the hypothermic myocardium. Peripherally, the initial cold-induced vasoconstriction may be replaced by cold-induced vasodilatation on immersion in water below 50°F (10°C). This is thought to be due to a direct effect of the cold on the smooth muscles of the vessels.

Electrocardiographic findings consist characteristically of bradycardia and increased conduction
time with concomitant prolongation of the P.R, QRS, and Q-T intervals, and S-T deviations both upwards and downwards with a flattening or inversion of the T-wave. In profound cases of hypothermia, there is also development of a "J-wave" (Osborne wave), which is a positive deflection at the junction of the QRS and S-T segments. Figure 6 shows a typical J-wave in an electrocardiogram of a six year-old girl who was treated by Golden (1974) for hypothermia with a rectal temperature of 76.3°F (24.6°C). A marked increase in blood viscosity with sludging of red cells is also seen. Platelets markedly decrease and clotting time is prolonged.

Figure 6: EKG from hypothermia patient.

Kidneys. A marked cold diuresis occurs in the early stages of hypothermia as a result of the increased central venous pressure and its depressant effect on the secretion of antidiuretic hormone. As the hypothermia progresses and blood pressure falls, the glomerular filtration rate is reduced. But, due to impairment of the tubular transport mechanism, a higher percentage of the filtrate is excreted, carrying with it a proportionate amount of the electrolytes which are normally interchanged in this region. There is some evidence of lipoid accumulation in the distal tubules due to exposure to the cold, and renal failure is a frequent complication of chronic hypothermia.

Central Nervous System. As brain temperature falls, cerebral activity becomes impaired. Amnesia is reported to occur at temperatures below 93.2°F (34°C). When core temperature falls below 91.4°F (33°C), the individual becomes semiconscious, and introversion and apathy are seen. Consciousness generally seems to be lost or severely impaired at rectal temperatures of 87.8°F to 84.2°F (31°C to 29°C).

Electroencephalogram changes appear as the body temperature falls between 96.8°F and 89.6°F (36°C to 32°C). These changes consist of decrease in amplitude of the potentials in the occipital areas first, followed later by changes in the parietal and frontal areas. At about 86°F (30°C), large delta waves appear in the frontal area; the electrical activity then declines until between 68°F and 64.4°F (20°C to 18°C) when no potentials can be recorded. As temperature continues to fall, muscle reflex activity becomes increasingly more difficult to elicit. The pupils begin to dilate at
about a core temperature of 91.4°F (33°C) and are usually fully dilated and unresponsive to light when the core temperature has fallen to 86°F (30°C).

"Afterdrop." Nearly all immersion hypothermia victims experience a deterioration in their condition manifested by an "afterdrop" or paradoxical fall in their core temperature once they have been removed from the cold water. This phenomenon has been described by many investigators and is attributed to a continuation or progression of the rate of change of body temperature for an additional 10 to 20 minutes after removal from the water. It is thought to be caused by returning cooled blood to the core from the reviving peripheral circulation. In the Dachau experiments, the afterdrop averaged 1.9°C but did reach 4°C. Coincidental with the afterdrop in temperature, there is likely to be a fall in pH and some degree of hypotension. But in hypothermia due to immersion, there is usually insufficient time for renal compensation of the abnormal fluid and electrolyte changes which take place. Even though a grossly abnormal physiological condition may exist, it is primarily a thermal disturbance, and it is that thermal disturbance which must be promptly treated.

Diagnosis. Untreated accidental hypothermia carries an extremely high mortality rate. Hypothermia should be considered in the differential diagnosis of all drowning victims as well as in the less circumstantially obvious cases of unconsciousness due to alcohol or drug intoxication and trauma. The diagnosis may easily be confirmed by use of a special low-reading thermometer or thermocouple. In the absence of a special thermometer, a reasonably accurate assessment of the core temperature may be estimated from the clinical signs and symptoms whose physiological basis has been described in the preceding section. These signs and symptoms and their relationship to body temperature are depicted in Figure 7 (Golden, 1973).

The usual signs of "clinical death" are extremely unreliable in victims of hypothermia. Below 80.6°F (27°C), all evidence of life is extremely difficult to detect. At these temperatures, there is an intense bradycardia and respiratory depression coupled with hypotension and extensive peripheral vasoconstriction, all tending to make it difficult to palpate a peripheral pulse or hear a heart beat. Muscles are extremely flaccid and the pupils are widely dilated. Even cardiac standstill demonstrated electrocardiographically is insufficient evidence of death since the literature contains documented cases of cardiac arrest of durations up to one hour in hypothermic victims who were subsequently revived. Therefore, it is imperative that resuscitative efforts begin at once.

Figure 7: Symptoms and signs in acute hypothermia
Treatment. The goal of therapy is to restore to normal the core temperature of accidental hypothermic victims as expeditiously and safely as possible. The treatment of immersion hypothermia may be divided into preventive, first aid, and definitive segments. The most effective treatment is undoubtedly prevention, and the issuance of protective clothing and personal survival gear to marines together with instructions in their proper use may well reduce the incidence of immersion hypothermia cases. In spite of these precautions, personnel aboard ship may be swept over the side; or adequately clothed and outfitted downed crew members may exceed designed time limitations for clothing protection in cold water, and, once rescued, treatment may be required.

Treatment will depend on the conditions under which the rescue was executed and the immediate facilities available. Survivors who are rational and possess motor function, although shivering dramatically, usually do not require treatment beyond safeguarding against further heat loss. If facilities are available for rewarming, they should be utilized. But, if the survivor is well insulated and protected from further heat loss, metabolic heat produced by their own shivering will rewarm them in time. Hot, sweet drinks, rest, and avoidance of an overheated environment are recommended.

The following paragraphs discuss the treatment of the semiconscious, unconscious, or apparently dead victim.

First-Aid Treatment. The primary goals of the first-aid treatment are to prevent further loss of heat and to maintain life until the survivor can be transported to a definitive treatment center. If the distance to be traveled requires more than 20 to 30 minutes, attempts should be made to provide definitive treatment on the spot.

1. Immediately remove the victim from the hypothermia-inducing environment.

2. Handle the patient gently in order to minimize the likelihood of the development of ventricular fibrillation in the sensitized myocardium. Do not attempt closed-chest cardiac resuscitation. Do not attempt to undress the patient because the manipulative efforts required to remove the wet clothing may be detrimental. In mild cases, wet clothing may be removed and replaced with dry clothes or blankets.
3. Maintain a clear airway. The patient should be placed in a slightly head-down position to combat hypotension. Semiconscious patients should be transported in the same manner in anticipation of unconsciousness which may develop as a result of the "afterdrop."

4. Further heat loss should be prevented by wrapping the patient in blankets, enclosing the body in a large polyethylene bag, and insulating them from the ground. No attempt should be made at this time to rewarm the patient actively by using hot water bottles, catalytic warmers, or any other means.

5. Administer oxygen by oronasal mask.

The risk of developing ventricular fibrillation in the cold-sensitized myocardium must be emphasized. Profound hypothermia mimics cardiac arrest, and attempts to restore cardiac activity by closed-chest cardiac resuscitation are likely to produce ventricular fibrillation. Furthermore, should the heart be in arrest, any effort to restore normal rhythm is unlikely to succeed until the myocardium begins to rewarm, when a spontaneous reversion to normal rhythm may occur. The patient isn't dead until they are warm and dead.

**Definitive Treatment.** Restoration of core temperature to normal levels may be accomplished by using either of two approaches, broadly categorized as external. In the external method, the surface is rewarmed in advance of the core, and heat is transferred from the shell to the inner body by direct conduction and by convection of the circulating blood. External rewarming may be an active process in which heat is applied to the body by immersion in 104°F (40°C) water, heating pads, hot water bottles, or heat cradles. Or, external rewarming may be passive in which case no heat is applied to the patient. Blankets may or may not be used to cover the patient who is allowed to achieve, without active assistance, an equilibrium state with ambient room temperature. In contrast to external rewarming, internal rewarming refers to raising the core temperature in advance of the shell. Although case reports are few in number, success has been reported by using such internal techniques as peritoneal lavage with solutions warmed to 104°F (40°C), placing the patient on a heart-lung machine, femoral artery bypass, and inhalation of warmed, water-saturated gases. Advantages cited for the internal rewarming method of treatment are the rapidity with which normothermia can be achieved, the more rapid return of cardiac output and the electrocardiogram toward normal, and the avoidance of "rewarming shock" or further drop in core temperature during early rewarming.

The method of rewarming selected as therapy by the attending medical officer will depend in part on facilities available, circumstances surrounding the immersion, condition of the patient, and the physician's familiarity with the various treatment modalities. In spite of the promise inherent in the new internal rewarming therapeutic approaches, rapid, active external rewarming probably remains the most successful and most easily performed treatment for the victim of immersion hypothermia. The following regimen is suggested for the operational physician with the understanding that it may differ from that available, or even desirable, in a fixed medical treatment
facility:

1. Handle the patient gently. If clothing must be removed, cut it off.

2. Rapidly rewarm the patient in a 106°F (41°C) water bath in which the water is agitated. A whirlpool bath is suitable for use when available. For multiple cases or where a whirlpool is not available, a small life raft inflated near a source of hot water is a satisfactory substitute. An unconscious, unclothed patient may be exposed to water temperatures up to 112°F (44.5°C). Initially, the arms and legs should be kept out of the bath to delay the revival of the circulation in the tissue mass of this portion of the cold shell. This may reduce the work load on the cold myocardium and possibly reduce the magnitude of the "afterdrop."

3. Maintain a clear airway and administer 95 percent oxygen and five percent carbon dioxide via an oronasal mask to overcome the oxygen debt that occurs on rewarming. If at all possible, intubation should be avoided as it can produce ventricular fibrillation.

4. Closely monitor core temperature, vital signs, and electrocardiogram.

5. Start a central venous pressure monitor and draw blood for an immediate reading pH, PCO₂, PO₂, and electrolytes, and then follow these parameters as an indication of treatment progress.

6. Be prepared to administer intravenous sodium bicarbonate warmed to 98.4°F (37°C) before transfusion.

7. Do not give antiarrhythmic drugs since they have little value at lowered body temperatures and may even precipitate ventricular fibrillation. Should ventricular fibrillation develop, it should be remembered that defibrillation is of little value when the cardiac temperature is below 82.4°F (28°C). If on rewarming sinus rhythm has not returned when deep body temperature reaches 86°F (30°C), then defibrillation should be attempted.

8. Leave the patient in the hot bath until they are subjectively warm. Then remove them and place them in a warmed bed. The patient should not be left in the hot bath until their temperature returns to normal levels because there is a tendency for the temperature to "overshoot" upon removal from the bath, and a subsequent difficulty in readjusting may be encountered.

Recovery from acute hypothermia is usually dramatically swift following rapid rewarming. Nevertheless, even after the patient revives, they should be treated as a total emergency and constantly monitored until their temperature returns to normal and they are ambulatory. Catastrophic arrhythmias have occasionally developed after it appears that the acid base balance and cardiovascular performance have returned to normal. The hypokalemia which is commonly seen in hypothermia, despite the usually associated acidosis, is probably secondary to the
intracellular migration of potassium and should not be interpreted as requiring vigorous replacement.

The most commonly seen late complications of acute accidental immersion hypothermia are pneumonia, renal failure, and pancreatitis.

Local Hypothermia

Local hypothermic injury may be represented as a collection of traumatic conditions whose severity is characterized by a continuum of tissue damage ranging from the most mild, chilblains, to the most extensive, deep tissue freezing. The type of injury produced is a function of the temperature to which the body or its parts are exposed, the duration of exposure, and other concurrent environmental factors.

It is an accepted convention to divide injuries into "freezing" and "nonfreezing" types. Nonfreezing cold injuries comprise chilblains and trench or immersion foot. Chilblains result from intermittent exposure to temperatures above freezing accompanied by high humidity. Chilblain is the only cold injury which is not militarily significant.

Trench Foot. Trench foot and immersion foot are indistinguishable with respect to cause, pathology, and treatment. Trench foot (immersion foot) results from prolonged exposure to wet, cold foot gear or outright immersion of the feet at temperatures usually below 50°F (10°C). At the upper range of temperatures, exposures of 12 hours or more will cause injury. Shorter durations of exposure at or near 32°F (0°C) will cause the same injury. Dependency or immobility of the feet aggravates and predisposes the development of the condition. Sailors in seawater or soldiers and marines with wet feet in trenches, rice paddies, or foxholes develop trench foot.

Warm water immersion injuries were described during military operations in Vietnam (Anderson, 1967). In these cases, the injury consisted of whitening and wrinkling of the skin and pain in the feet after two days or more of water exposure. Additional exposure resulted in erythema on the weight-bearing surfaces and edema. Complete recovery occurred following proper foot care. In a seven to ten-day military operation in which continuous wet foot exposure occurred, Anderson found that about 30 percent of the troops required evacuation for immersion foot injury. This could have been prevented had the troops been able to dry their feet for six to eight hours per day with boots and socks off, but such is rarely possible in combat situations.

Recently, Hawryluk (1977) described military operations in a temperate climate over a 13-day period when the minimum temperature ranged between 28 and 43°F with a chill factor between 8°F and 41°F. Cold injuries represented over one-third of the cases seen for treatment, and approximately one-third of these received diagnoses of trench or immersion foot.

Frostbite. Frostbite results from the formation of minuscule crystals of ice within the extracellular
fluid of the skin and adjacent tissues and is caused by exposure to temperatures below the freezing point. The severity and extent of injury are functions of the temperature and the duration of exposure. Human flesh freezes at cooling values of 1,300 to 1,500 calories per square meter per hour. With a wind velocity of 22 miles per hour, exposure at temperatures of 17.6°F (-8°C) for one hour, or -22°F (-30°C) for one minute, will cause human flesh to freeze. This emphasizes the importance of windchill as a causative agent in frostbite.

Pathogenesis. The multiple and complex mechanisms eventually resulting in tissue damage are not fully understood. Some of the mechanisms which have been implied are

1. Direct cold effect on certain cellular enzyme systems.

2. Intracellular hyperosmolarity secondary to cellular dehydration as the water from within the cell is drawn into the extracellular fluid to replace the water in the extracellular space, which has crystallized into ice.

3. Mechanical disruption of the cellular membrane and intracellular structures by ice crystals, particularly during slow thawing during which some refreezing of the melt occurs. The new ice crystals are actually larger than those formed during the original freezing.

4. Tissue hypoxia as a result of decreased perfusion due to irreversible damage to capillaries and small vessels.

Diagnosis. The development of frostbite is particularly insidious, and the victim frequently is unaware of its happening. Frostbite most commonly occurs on the face, hands, and feet. A sudden blanching of the skin of the nose, ear, or cheek signals its onset. This may be subjectively, noted by the experienced as a momentary tingling or "ping." Subjectively, the patient feels that his face muscles will not work. Telltale, yellow-white spots appear early, and their early observation by another person may minimize tissue damage. A frozen extremity appears white, yellow-white, or mottled blue-white and is hard, cold, and insensitive to touch. Even a very shallow or superficial frostbite injury may have the appearance of being frozen completely solid because of the dermal freezing alone. An elegant classification of cold injury by degree of severity is presented in NAVMED P-5052-29, but such designations are somewhat academic in the clinical situation since the definitive classification into degree of injury is often a retrospective diagnosis and the treatment is the same.

Once thawing has transpired, the injured extremity usually becomes edematous. Large, serum-filled blisters develop within an hour to several days after thawing is complete. Unless accidentally ruptured, the blebs remain intact until the fourth to tenth day post injury when resorption of the fluid begins. At this time, spontaneous rupture of the blebs may occur. As the blebs dry, a hard eschar begins to separate spontaneously, and the delicate healthy tissue beneath
becomes visible.

Should the extent of the tissue damage be so severe as to preclude tissue healing, blebs do not develop, and the skin remains cyanotic and cold. This is most commonly seen in distal phalanges, and evidence of beginning mummification may be observed, often within a few days. Mummification becomes more pronounced over a period of days, weeks, or months, and the demarcation between healthy and dead tissue becomes more obvious. The viable tissues separate and retract from the mummified until spontaneous amputation of the soft tissue is essentially complete.

The foregoing description is based on a clinical pattern uncomplicated by infection or premature surgical intervention. Infection or unwarranted early debridement may result in excessive tissue loss, osteomyelitis with need for successively higher amputations, extensive skin grafting, and prolonged hospitalization.

**Treatment.** Treatment of frostbite is directed towards the preservation of the maximum amount of viable tissue and the restoration of maximum function. These goals are achieved by rigid adherence to the following principles: gentleness in handling the frozen parts to prevent additional mechanical trauma, rapid thawing of the frostbitten tissue, prevention of infection, early institution of active motion of the injured part, and avoidance of premature surgical intervention. Mills (1973, 1976) has reported good anatomical and functional results by using such a regimen of treatment.

When the patient is at a distance from a medical facility capable of providing definitive care, the management of a frostbitten extremity is dependent on the amount of time necessary to reach that facility. If the time is only several hours, it is best that the frozen part be kept in the frozen state until arrival. The patient should be transported with the extremity carefully padded or splinted to avoid mechanical trauma, and the affected part should be either isolated from the heater of the transport vehicle or even placed on ice. Because there appears to be a direct relationship between the amount of time that tissue is frozen and the amount of residual tissue damage, the frozen extremity of a patient who is more than several hours away from definitive care should be thawed by rapid rewarming in an environment where refreezing cannot possibly occur. Then, the thawed extremity must be protected from pressure or mechanical injury. Thawing of a frostbitten part should not be undertaken when there is any danger of refreezing; the danger of thawing and subsequent refreezing is greater than the danger of remaining frozen.

Upon arrival of the patient, operational physicians should perform a thorough physical examination to rule out general hypothermia, concomitant injuries, and cardiorespiratory problems. Should general hypothermia be present, or should there be generalized or local tissue anoxia secondary to blood loss or trauma, they must be prepared to perform intubation, cardiac defibrillation, or other resuscitative procedures which may be indicated. Once the examination and any emergency procedures have been completed, the affected part should be rapidly thawed. A whirlpool bath at 100°F to 108°F (38°C to 42°C) is the method of choice. This temperature
range is warm enough to dissolve the ice in the tissue rapidly, but not so warm that tissue damage might result from excessive heat. The part should be gently handled to prevent mechanical trauma. Although the thawing process is relatively quick, it is usually quite painful, and morphine or meperidine may be required for relief of pain.

As tissue thawing proceeds, a superficial pink flushing will be seen to progress distally along the extremity. Immersion in the whirlpool bath should be continued until the distal tip of the thawed part flushes, is warm to the touch, and remains flushed when removed from the bath. Occasionally, the flush may not be pink, but rather burgundy or purple, colors which are usually indicative of ischemia and retention of venous blood. In frostbite, however, the color change seems to be directly related to the bath water temperature. The color change is usually transient, but persistent cyanosis or ischemia, despite rapid thawing, may indicate increasing pressure within the fascial compartment due to an associated fracture, sprain, soft tissue injury, or disease, and a fasciotomy may be necessary.

With rapid thawing, the change is dramatic and the patient quickly becomes responsive and even alert. So rapid, however, is this method of thawing that the rapid entry into the circulation of liberated acid end products of metabolism may precipitate metabolic acidosis and subsequent death as a result of ventricular fibrillation within one to three hours. Consequently, initial care must also be directed toward the avoidance of acidosis. Electrolytes, pH, PCO₂, and PO₂ should be serially monitored as fluids and sodium bicarbonate are administered. Electrocardiographic monitoring should be simultaneously performed.

Sensation often returns to the affected part with rapid thawing, but this is a transitory phenomenon. The sensation disappears once the blebs develop and separate the epidermis from the dermis or the dermis from underlying tissue. Sensation does not fully return again until healing is complete.

Once thawing is complete, the injury is treated in an "open" fashion, and a modified isolation technique is employed. If the lower limbs were frozen, the patient is placed on absolute bed rest, and the legs are elevated and kept on sterile sheets. A protective cradle covered with sterile drapes is placed over the legs to prevent injury or pressure, and sterile cotton pledgets are put between the toes. When the injury is to the hands and forearms, they may be comfortably positioned on sterile sheets placed on the patient's chest and abdomen. Attendants should wear clean gowns and masks, and when changing linens or manipulating the injured part, they should wear sterile gloves. Extreme care should be exercised to prevent further injury during nursing procedures requiring manipulation of the affected part.

The primary goals during this phase of therapy are to maintain joint motion and prevent infection. At the core of the treatment regimen is use of the whirlpool bath at least twice daily. The water temperature is maintained at 95°F to 98.6°F (35°C to 37°C), and hexachlorophene is added to the water for its bacteriostatic effect. The agitated water of the whirlpool bath cleanses...
gently and promotes physiological debridement while aiding circulation. This treatment is extremely effective in minimizing infection, and as a result, antibiotics are seldom necessary unless there is a very deep infection. A tetanus toxoid booster is routinely administered.

Active exercises are the other indispensable part of this phase of treatment, and they are begun as soon as possible. Hourly digital movements are demanded of the patient. Most patients find the movement easier to perform while in the whirlpool bath due to the softening of the hard eschar by the warm water. When there is lower extremity involvement, Buerger's exercises are performed for 20 to 30 minutes at least four times daily as follows:

1. Patient supine, legs elevated at 30° angle for two minutes.

2. Patient sits on edge of bed, feet dangling:
   a. Flexes and extends ankle . . . One minute, then
   b. Rotates lower leg . . . . . Slowly and deliberately for one minute, then
   c. Spreads and closes toes . . Three minutes.

3. Patient then lies flat in bed with legs under blanket for five minutes.

4. Repeat above cycle three to six times per session.

The importance of active physiotherapy during the first six weeks for the prevention of flexion contractures and joint ankylosis cannot be overemphasized.

Because of the prolonged nature of treatment and the danger of addiction, narcotics are used for control of pain only during the initial thawing process. After rapid rewarming, the patient should be switched to aspirin, propoxyphene, or diazepam for the long recovery period. Use of tobacco is prohibited because of the vasoconstrictive effect. Alcohol is generally not permitted because of its variable effect on peripheral blood flow.

The blebs which appear shortly after the initial rewarming process should be left intact. The fluid contained in the blebs is usually sterile, as is the underlying tissue. The blebs generally rupture on about the third to seventh day, and as they dry, hard, dark, and often circumferential eschars develop which may inhibit motion of the digits, particularly the interphalangeal and metacarpal phalangeal joints. Should this occur, the eschars should be carefully split along the dorsum or lateral borders to avoid injury of the underlying neurovascular bundles. The eschar should not be removed. The whirlpool bath will perform that function at a physiological rate and minimize scarring which could occur from cutting into granulating membranes during surgical debridement.

Generally by the 10th to 14th day, and almost always by the 21st day, the eschar has begun to slough into the whirlpool bath and healing is readily apparent. At that point, isolation technique and sterile precautions may be terminated. However, the physiotherapy and whirlpool should be
Despite adherence to the foregoing treatment regimen, some digits or distal portions of an extremity may remain black and cold and appear nonviable. Surgical intervention should be withheld until spontaneous amputation of the soft tissue is nearly complete. This may require from three weeks to four months. At that time, the mummified portion may be surgically removed without danger of retraction of distal tissue or a higher amputation than necessary. The rationale underlying this phase of therapy is to permit the injured part every opportunity to demonstrate its viability. The physician must resist the urge to do something surgical as well as the exhortations of the depressed patient to amputate the injured part prematurely and rid him of the black foot or finger.

The single exception to this policy of nonintervention is when a thawed extremity, which was frozen for a relatively long period, exhibits a clinical picture similar to anterior tibial compartment syndrome. This includes pain, severe edema, restricted joint motion, evidence of ischemia, and a marked increase in compartment pressures, which is obviously compromising the blood supply. In these patients, a fasciotomy or a sympathectomy should be considered. The vascular response is almost immediate following a fasciotomy, and a sympathectomy, according to Mills (1976), appears to promote a much more rapid resolution of any infection which may be present, as well as rapid diminution of edema and a significant lessening of pain. It has also been observed that the combination of the two procedures seems to hasten the demarcation between healthy and nonviable tissue in the affected part.

The whole therapeutic approach for deep freezing injuries is by necessity lengthy, and it requires a great deal of care and patience. During the first several months, pleasant environment, frequent visits, encouragement, and occupational therapy are mandatory. The patient should be involved in his own treatment by explanations of tissue changes that are taking place and discussions regarding his progress. The patient's observations of joint motility in the whirlpool should be reinforced by the physician in order to substantiate that, despite the grim appearance of the injury, function is being preserved. As in any tissue-destroying illness, a high caloric, high protein diet with vitamin supplements is beneficial.

Other modalities of therapy have been proposed from time to time, and still others are under investigation. The beneficial effects ascribed to steroids and antihistamines in the treatment of cold injury have not been clinically demonstrated. The use of vasodilators has been disappointing. In studies of microcirculation of experimental animals following freezing and thawing, Mundth (1964) reported that immediately following thawing the circulation was apparently unimpaired, but within a few minutes, evidence of obstruction could be seen in the venules. The obstruction apparently began with aggregations of platelets, followed by piling up of erythrocytes behind them with stasis extending back through the capillary bed to the arterioles. Within two hours or less, the stasis had become irreversible, and the vessels were totally filled with a structureless,
hyaline-like material. There was tissue edema and evidence of extravasation of hemoglobin into the perivascular spaces. Mundth investigated the use of low molecular weight Dextran in rabbits following freezing and thawing. Examination of the microcirculation demonstrated that the low molecular weight Dextran had alleviated the post-thawing obstruction and was effective in reducing tissue loss in frozen rabbit feet if it was infused as late as two hours following thawing. Clinical experience with this method of treatment is still limited.

**HYPERTERMIA**

When heat production or exchange of heat from the environment to the body exceeds heat loss, a state of hyperthermia may develop. Circumstances in which the potential exists for the development of hyperthermic conditions are ubiquitous. Some examples are personnel inspections during hot and humid weather, recruit training, field maneuvers in tropical or subtropical climates, strenuous physical activity by individuals with sunburn, duties in ships' engineering spaces, the "greenhouse" effect in poorly ventilated and unairconditioned aircraft, and extravehicular activity (EVA) by astronauts. Seabees are at particular risk because of the intense physical labor associated with construction projects in extremes of climate and temperature. In his roles as industrial medical officer and general medical officer, the operational physician is frequently involved not only in the treatment of such cases, but also in the assessment of environmental risks, evaluation of equipment, and training of personnel. Crews must be monitored very closely to prevent heat casualties.

**Physiology of Hyperthermia**

In a discussion of the interaction of environmental physical parameters and physiological processes, it is customary to adopt the engineering usage of the terms "stress" and "strain," particularly since the body's final physiological state reflects both the independent and integrated results of all those factors which exert an influence on heat exchange and the body's regulatory mechanisms. The term "stress" denotes the force or load acting upon the biological system, and the term "strain" is used to designate any resulting distortion of the biological system. Conventionally, stress factors are heat, cold, humidity, radiation, air movement, and surface temperature. Thermal strain, as a response to the imposed thermal stress, may manifest itself in specific cardiovascular, thermoregulatory, respiratory, renal, endocrine, and other reactions which differ in type or degree from accepted norms. A thermal stress is categorized as acceptable when humans are able to compensate without undue strain, but it is considered unacceptable when humans are able to compensate but incur severe strain, or when they are unable to compensate and incur excessive strain. Thermal strains are categorized as those interfering with work performance and safety, and those with more overt manifestations of physiological decompensation, such as heat rash, heat cramps, heat exhaustion, heat stroke, or cold injuries.

**Thermoregulation.** Although thermoregulatory mechanisms in humans have not been fully explicated, animal experiments have provided convincing evidence that the temperature-regulating center in humans lies in the hypothalamus. The anterior portion appears to contain the "heat loss"
center which responds to increases in its own temperature as well as to afferent nerve impulses from receptors in the skin. The center mediates heat loss through increased blood flow to the skin and sweating (humans) or panting (other mammals).

Sweat Rate. In response to the stimulus of a heat load resulting in an error signal, skin sweat glands are activated. The number of glands recruited and the rate of secretion of each gland determine the total sweat rate. The estimated 2.5 million eccrine glands can secrete sweat at peak rates of more than 3 kg/hr for up to an hour in highly acclimatized humans, and they can maintain rates of 1 to 1.5 kg/hr for several hours. When there are no restrictions to evaporation of sweat from the skin and under steady state conditions of work and heat exposure, evaporative cooling is regulated to balance the heat load up to the maximum rate of sweating of 1 kg/hr. Under steady state conditions of work, core temperature \((T_c)\) is constant despite ambient temperatures widely varying from cool to moderately hot, and any elevation of \(T_c\) above 37°C depends solely on the metabolic work rate. The central drive for sweating is determined by work rate but the actual sweat output is modulated by skin temperature to meet evaporative requirements under conditions from cool to hot, up to the limits of the sweating mechanism.

Sweat Evaporation. The efficiency of body cooling by sweat is a function of the rate of evaporation. When evaporation of sweat is restricted by a reduced vapor pressure gradient due to high ambient humidity, more sweat glands are recruited in order to increase the area of wetted body surface. If the evaporative cooling is sufficient to balance the heat load under these conditions, core temperature remains essentially unchanged. When the body surface is 100 percent wetted, any further increase in sweat production does not contribute to cooling, and the sweat drips off the body and is wasted.

Cardiovascular System. Under comfortable ambient conditions, \(T_{skin}\) is normally 33°C to 34°C, but under heat stress, it may approach to within a degree or two of \(T_c\), or it may decline to as much as 10°C to 15°C below \(T_c\) in the cold. Changes in core to surface gradient are accompanied by alterations in the rate of blood flowing from the core to the surface to meet changing needs in heat conductance. Heat conductance is defined as units of heat transferred to the environment through the skin per unit time per degree of temperature gradient. Under conditions of heat stress, \(T_{skin}\) rises and the core to skin gradient narrows. Consequently, a greater volume of blood must flow through the skin each minute to achieve the same rate of heat exchange as in a neutral environment. This is the basic cause for heat strain on the cardiovascular system.

In a thermally neutral environment, \(T_{skin}\) is lower during work than at rest because of redistribution of blood from the skin blood vessels to those of active muscles. The return of adequate venous blood to the heart is maintained by the reduced capacity and increased resistance of skin and visceral vessels together with the pumping action of the muscles. This facilitates increased cardiac output which is proportional to the percentage of maximum oxygen uptake.
required by skeletal muscle work. When there are external heat loads concurrent with the performed work, however, both the central drive for increased conductance and the rise in local skin temperature cause dilation of skin vessels, thereby increasing their blood capacity and reducing their resistance. To meet the oxygen demand of the working muscles, cardiac output can be maintained only by further constriction of splanchnic vessels and increased heart rate.

Heat Tolerance

Acclimatization. Any well-motivated young person in good physical condition who works for the first time under conditions of heat stress will exhibit signs of heat strain evidenced by increased heart rate, high body temperature, and other signs of heat intolerance. But on each succeeding day of heat exposure, their ability to work improves and signs of strain and discomfort diminish. In other words, they adapt to the thermal stress, and as a result of working in a hot environment, they acquire the enhanced tolerance to environmental heat stress called heat acclimatization.

The acclimatization process begins with the first exposure to heat and is achieved most safely and expeditiously over a period of one to two weeks by progressive degrees of heat exposure and physical exertion. In order to achieve maximum acclimatization, the work level should be in the 200 to 300 kcal/hr range. Sedentary work levels will not result in adaptation. A factor apparently essential in inducing acclimatization is the sustained elevation of \( T_c \) and \( T_s \) above levels for the same work in a cool environment. Acclimatization ordinarily cannot occur when heat stress levels exceed a certain level, and personnel working in areas of unusual stress, such as fire rooms and engine rooms, should not be expected to adapt physiologically to their environment if the parameters outlines in Table 2 are exceeded.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Lower Work Level Inside Space</th>
<th>Upper Work Level Inside Space</th>
</tr>
</thead>
</table>

Table 2: Heat stress design conditions*
DryBulb Temperature (°F) 97 105
Wet-Bulb Temperature (°F) 83 84
Globe Temperature (°F) 117 125
Effective Air Velocity At Man (fpm) 25.3 24.6
Relative Humidity (%) 91.2 94.3
Ambient Vapor Pressure (mm Hg) 155.4 162.0
Wet-Bulb Globe Temperature Index (°F)
Mean Radiant Temperature (°F)

- All of the above figures apply to normal work sites with a time-weighted-mean metabolic rate of 76 kcal/(m²-hr) for the lower work level; data were based upon no cumulative fatigue in personnel (NAVMED P-5013-3, 1974)

Once acclimatization has occurred, there is a significant increase in sweat output which is produced at a lower Tₛ in comparison to both sweat rates and skin temperatures earlier in the adaptive process. Within the environmental restrictions discussed above, the increase in evaporative cooling with a steeper core to skin gradient is sufficient to compensate fully for the heat load. This is demonstrated by restoration of the core temperature to levels observed while performing the same work in a cool environment. Although BF₆ remains elevated following acclimatization, the circulatory load is diminished as evidenced by a reduction in heart rate.

Acclimatization to wet heat increases tolerance to dry heat and vice versa. The reason why tolerance to wet heat is increased is not clear, nor are the underlying changes in the thermoregulatory axis which control the adaptive process itself. It is well known that people who work at hot industrial tasks acquire levels of acclimatization commensurate with their average heat exposure, but increased work demands or increased environmental stress may overload their thermoregulatory capacity and lead to signs of overstrain. Heat acclimatization requires periodic reinforcement, such as occurs daily during the workweek. A partial loss of acclimatization may be demonstrated after return to work following the weekend, and should the absence be longer, such as a vacation of several weeks, the loss of acclimatization may be substantial. A summary of physiological indices of advanced heat acclimatization is provided in Table 3.
Physical Fitness. A state of good physical fitness alone does not confer heat acclimatization, but physical training even without heat exposure does improve heat tolerance, as indicated by somewhat lower heart rates and core temperatures in people exposed to heat after conditioning as compared with before conditioning. However, sweat rates do not increase and skin temperature remains high. Therefore, it may be said that physical conditioning enhances heat tolerance by increasing the functional capacity of the cardiovascular system. This results from an increase in the number of capillary blood vessels in muscle, thus providing a larger interface between circulating blood and muscle for exchange of oxygen and metabolic waste products. Small veins in tissues other than muscle also develop increased tone and are able to reduce their capacity during exercise, thus promoting an increase in pressure in the large central veins returning blood to the heart. Together, these factors allow an increased maximum oxygen uptake in the physically conditioned individual, permitting them to withstand a greater circulatory strain of work under heat stress.

### Table 3: Physiological indices of heat acclimatization

<table>
<thead>
<tr>
<th>Physiologic Parameter</th>
<th>Progressive Adaptive Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cardiac output (estimate by noninvasive indirect technique)</td>
<td>1. Increased on day one; decreasing toward normal with progressive exposure</td>
</tr>
<tr>
<td>2. Cardiovascular reserve (measured in terms of peripheral vascular resistance with peripheral vascular collapse theoretic end-point)</td>
<td>2. Gradual increasing from first day of exposure</td>
</tr>
<tr>
<td>3. Body temperatures (rectal, deep esophageal, tympanic, skin)</td>
<td>3. Markedly increased on day one; gradual normalizing thereafter. After adaptation to tropical environmental temperatures a thermal equilibrium was achieved within 30 minutes in acclimatized subjects doing constant moderate work.</td>
</tr>
<tr>
<td>4. Sweat rate</td>
<td>4. Low on day one, increasing daily thereafter until day 8-10, after which time it again decreased toward normal.</td>
</tr>
<tr>
<td>5. Urine Osmolality</td>
<td>5. Slight increase on day one with more rapid increments thereafter</td>
</tr>
</tbody>
</table>
6. Serum Osmolality

Until day 8-10. Osmolality then decreases each day toward normal (response is similar to that of sweat rate).

6. Studies are incomplete, but suggest that changes are minimal during acclimatization. Investigation suggests that other adaptations tend to maintain the osmolar integrity of the serum.

**Surface Area to Weight Ratio.** Heat loss is a function of body surface area, A, and heat production is a function of body weight, Wt. Therefore, a low A/Wt ratio is a handicap for individuals performing sustained work under conditions of thermal stress. In obese individuals, as well as those with compact or stocky builds, the A/Wt ratio is relatively low. If lacking in acclimatization, physically unfit and obese people are at greater risk of succumbing to heat stroke.

**Age and Disease.** A healthy worker over 45 years of age may perform well on hot jobs when they are allowed to work at their own pace. However, under demands for sustained work output in the heat, they are at a physiological disadvantage compared to the younger worker. Between ages 30 and 65, the maximum oxygen uptake declines 20 to 30 percent, and the older worker has less cardiovascular reserve capacity. In addition, at levels of heat stress above the prescriptive zone, the older worker compensates less effectively for the heat loads, as demonstrated by higher core temperature and peripheral blood flow for the same work output. This has been attributed to a delay in onset of sweating as well as to a lower rate of sweating, contributing to greater heat storage and a longer recovery time. The presence of any degenerative diseases of the heart and blood vessels intensifies the age effect by limiting the circulatory capacity to transport heat from the core to the surface.

Illness other than degenerative disease may predispose to the development of heat injury under mild environmental conditions which would ordinarily not be considered to represent a significant risk. Bartley (1977) lists the following heat factors which have been reported in the literature as significant risks for development of heat stroke: acute febrile illness, fatigue, recent immunizations with subsequent reactions, acute and convalescent infections, medications, past history of heat injury, chronic disease such as diabetes or cardiovascular disease, following certain surgical procedures, and lesions of the hypothalamus, brainstem, and cervical part of the spinal cord.

**Water and Salt Balance.** Successful adaptation to heat and the maintenance of effective work performance are dependent on the replenishment of the body water and salt lost in sweat. Sweat volume may amount to 12 liters in a 24-hour period (Dasler, 1971), leading to a contracted extracellular fluid space. A fully acclimatized worker weighing 70 kg can secrete six to eight kilograms of sweat per eight-hour shift. Failure to replace water lost in sweat, despite continued sweating, may result in severe dehydration due to loss of intracellular as well as extracellular fluid. Although water deficits of one kilogram (1.4 percent of body weight of a standard human) can be tolerated without serious effect, deficits of 1.5 kg or more during work in the heat deplete
circulating blood volume. Even an acclimatized human will exhibit signs and symptoms of increasing heat strain (elevated T<sub>c</sub> and HR, thirst, and severe heat discomfort) resembling those seen in an unacclimatized individual. With water deficits of two to four kilograms (three to six percent of body weight), work performance is impaired, and continued work will lead to signs of incipient heat exhaustion.

A factor in the development of acclimatization is the successful attempt by the body to conserve salt. Already hypotonic sweat is produced in increased quantities with an even lower salt content, and the rate of renal sodium (Na<sup>+</sup>) reabsorption is increased. Among the mechanisms proposed to explain the ability to retain Na<sup>+</sup> is an increased plasma renin activity concomitant with the incremented aldosterone secretion which occurs during acclimatization. Studies (Francesconi, Maher, Bynum, & Mason, 1977) using small numbers of experimental subjects suggest that heat acclimatization may reduce the increase in plasma potassium (K<sup>+</sup>) induced by mild exercise at high ambient temperatures. No significant differences were observed in urinary potassium excreted by exercising and sedentary subjects. No significant differences in plasma Na<sup>+</sup> levels were demonstrated between exercising and sedentary men, but there was a significant reduction in urinary Na<sup>+</sup> excretion in the exercising men.

The estimated average diet of the general population of the United States contains about 15 gms/day of sodium chloride, including salt shaker supplementation at the table. This would meet the needs of an acclimatized worker producing six to eight kilograms of sweat containing one to two grams of salt during a single shift. During the period of acclimatization, a worker with no previous heat exposure might require supplemental salt because, although maximal sweat rates in unacclimatized workers are lower (four to six kilograms per shift), salt concentrations are higher (three to five grams per kilogram of sweat) than after acclimatization. Despite the difficulty of trying to equate time spent in physical conditioning, drills, or field exercises with shifts, military personnel may experience a similar magnitude of loss. A salt deficit of 15 to 25 grams may occur during the first several days of increased thermal stress. If field rations are consumed as meals, supplemental salt is not required because each ration, including the accompanying salt packet, contains 31 grams of salt, and daily dietary intake of salt may reach 93 grams.

An individual's greatest need for salt would occur during the simultaneous stresses of initial physical conditioning and heat acclimatization in a hot, humid environment without water restriction. Although salt tablets, which are 10 grains (0.648 gms) each (0.255 gms of sodium and 0.393 gms of chloride), are available, both experimental and clinical data suggest that unrestricted use of supplementary sodium chloride or salt tablets is contraindicated under most conditions of heat stress. Costill, Cote, Miller, Miller, and Wynder (1975) found minimal physiological benefit in supplementing drinking water with electrolytes when sufficient quantities of those ions were available in the daily diet, and subjects were permitted to ingest food and drink ad libitum. A physiological plasma sodium chloride level can be achieved by providing adequate water, a normal diet, and a salt shaker on the table for conservative use, with no more than the equivalent of two grams of supplementary salt (preferably not as salt tablets) per day.
Progressive dehydration may occur if water is replaced without concurrent replacement of salt because homeostatic controls are designed to maintain a balance between the electrolyte concentrations of the extracellular and intracellular fluid compartments. Deficient salt intake with continued intake of water tends to cause hypo-osmolality of the plasma which suppresses pituitary antidiuretic hormone (ADH). The renal tubules then fail to reabsorb water, and dilute urine containing little salt is excreted. Thus, electrolyte concentration of the body fluids is homeostatically maintained but at the cost of depleting body water and ensuing dehydration. Under continued heat stress, symptoms of heat exhaustion develop similar to those resulting from water restriction but with more severe signs of circulatory insufficiency and notably little thirst.

BUMEDINST 6260.2 series provides current statements on salt and water requirements.

Alcohol. Many authors have reported an excessive intake of alcohol by patients within hours or a day or two prior to onset of heat stroke. Striking reductions in workers’ heat tolerance on the day following alcoholic excesses have been described. Suppression of ADH by alcohol has been described, and the losses of body water in the urine and resultant dehydration have been postulated to be a primary mechanism.

Hyperthermic Illness

The classification of hyperthermic illness used in this chapter is the one agreed upon jointly by committees representing the United Kingdom and United States in 1964. A summary of the etiology, signs and symptoms, treatment, and prevention of heat illness is presented in Table 4.

Heat Stroke. Heat stroke is a bona fide medical emergency, and if treatment is not instituted immediately, the mortality rate is high. It occurs when the thermoregulatory mechanisms fail for reasons as yet undetermined. The central drive for sweating becomes inoperative, and cooling by evaporation is lost. There is an uncontrolled accelerating rise in Tc due to uncompensated heat storage. Predisposing factors include any of those which adversely affect tolerance to heat. Prodromal symptoms are headache, malaise, discomfort perceived as excessive warmth, or even those symptoms associated with heat exhaustion. The onset is usually abrupt with sudden loss of consciousness, convulsions, or delirium. Typically, sweating is absent, and the patient may have noted this prior to the onset of other symptoms. Since the patient may continue to ingest water in the absence of sweating, over hydration rather than dehydration may occur. Should diuresis occur as a result of this, it should be interpreted as an additional sign of the critical condition of the patient. During the early stages of heat stroke, the patient may experience a febrile euphoria once sweating has ceased and Tc has risen. Physical signs are a flushed, hot, dry skin; in severe cases, there may be petechiae present secondary to direct thermal injury of vascular endothelium which initiates platelet aggregation. Tc is high, frequently in excess of 105°F (40.5°C). A rectal temperature of 108°F (42°C) is not uncommon. As Tc continues to rise, loss of consciousness, convulsions, and coma may ensue. The patient's pulse is full and rapid, while the systolic blood
pressure may be normal or elevated, and the diastolic pressure may readily become depressed. Respirations are rapid and deep and simulate Kussmaul breathing. As the patient's condition worsens, peripheral vascular collapse may occur manifested by a rapid pulse, hypotension, and cyanosis. Breathing becomes shallow and irregular. Pulmonary edema and renal failure may develop. If the patient survives until the second day, recovery often occurs, but relapses may occur in the first few days after the temperature has been reduced from the critical level.

Treatment is directed toward rapid restoration of normal temperature. Immediate attempts should be made to lower body temperature to safe levels, $T_c$ of 100°F to 101°F (37.5°C to 38°C). The longer hyperpyrexia continues, the greater is the threat to life. In the field, the patient's clothes should be removed, and if there is a source of cool water nearby, they should be immersed in it. Otherwise, water should be sprinkled over the patient and its evaporation hastened by fanning. In addition to these cooling measures, attendants should rub the victim's extremities and trunk briskly to increase circulation to the skin. The patient should be transported as soon as possible to a facility properly equipped to perform definitive treatment. During transportation, cooling efforts should be continued by permitting passage of air currents through the open door of the field ambulance or helicopter. Once the patient reaches the hospital, they should be placed immediately into a tub of water and ice. Their extremities should be continuously massaged as noted above. During immersion, rectal temperature should be closely monitored, and when it drops to between 100°F and 101°F, the patient may be removed to a hospital bed. Rectal temperature should continue to be monitored every ten minutes until stable. During the first several days, the patient is susceptible to hypothermia as well as relapses of hyperpyrexia. It is therefore desirable to maintain rectal temperature between 100°F and 101°F. Rapidly increasing temperatures can usually be managed with ice water sponge baths and fanning; precipitous drops in temperature may require judicious use of warm blankets. Shivering is associated with increased involuntary muscular activity which is undesirable because it accentuates tissue hypoxia and lactic acid acidosis. If simple warming measures fail to control shivering, the physician may administer small intravenous doses of diazepam (10 mg).

Hypotensive shock usually responds to the cautious administration of balanced intravenous fluids, but plasma volume expanders may be useful if the patient is normothermic. Vasopressors are to be avoided. Central venous pressure, serum electrolytes, and hourly urinary output must be carefully monitored to avoid hyperhydration. Replacement fluids should be sufficient to repair imbalance of serum electrolytes and to restore acid-base balance, but care must be exercised to detect early signs of pulmonary congestion, rising venous pressure, or renal failure. Administration of oxygen by face mask or nasal catheter may be useful to combat tissue anoxia. Convulsions may be controlled by intravenous use of diazepam or short-acting barbiturates (sodium pentothal). Long-acting barbiturates and narcotics are contraindicated.

Serious physiological damage and altered response to heat stress may persist long after recovery from heat stroke. There is evidence to suggest that heat stroke victims may be more susceptible to recurrent episodes of heat illness under less intense environmental conditions. Therefore, they
should never be returned to heat stress similar to that which precipitated their illness without an evaluation and appearance before an appropriate Medical Board. A "Heat Casualty Report" (NAVMED 6500/1) should be submitted to Chief, Bureau of Medicine and Surgery, Department of the Navy, Washington, DC  20372.

**Heat Hyperpyrexia.** Heat hyperpyrexia is a milder form of the same illness in which there is partial, rather than complete failure of the central drive for sweating. In heat hyperpyrexia, the core temperature is lower, less than 105°F (40.5°C), and some degree of sweating is present. Central nervous symptoms and signs are less severe. Treatment is directed towards lowering core temperature as in heat stroke, but due to the less severe nature of the illness, less drastic measures may be adequate.
### Table 4: Classification of heat illness.

<table>
<thead>
<tr>
<th>Category</th>
<th>Clinical Features</th>
<th>Predisposing Factors</th>
<th>Predisposing Factors</th>
<th>Prevent</th>
<th>Prevent</th>
<th>Prevention</th>
</tr>
</thead>
</table>

(Leithed & Lind, 1964, published by perrCo.)
parade formation in hot outdoor climates. It is unrelated to salt or water deficiency or to excessive physical activity. Lack of acclimatization may be a predisposing factor together with the enforced immobility of standing in parade formation. The syncope results from a pooling of blood in dilated vessels of skin and lower parts of the body. Vagotonia may be a contributing factor. The momentary cerebral ischemia is relieved promptly once the patient's posture becomes horizontal as a result of the faint, and recovery is complete once the patient is moved to a cooler area.

Salt and Water Depletion. Disorders of salt and water depletion include the clinical entities of heat exhaustion and heat cramps. These conditions are most commonly seen in unacclimatized personnel who have sweated profusely during exertion under conditions of thermal stress. The sweating mechanism remains functional under adequate central drive, but the patients have mismanaged the water replacement of water and salt lost in sweating.

1. Heat Exhaustion. Patients with heat exhaustion suffer peripheral vascular collapse as a result of dehydration and depletion of circulating blood volume. The depletion of the circulatory blood volume may be absolute and secondary to failure to replace water and salt lost in sweat, or relative due to circulatory strain from competing demands for blood flow to skin and large skeletal muscles. The condition is characterized by profuse sweating, headache, tingling sensation in the extremities, dyspnea, giddiness, palpitations, and gastrointestinal symptoms of anorexia, nausea, or even vomiting. The patient may complain of neuromuscular disturbances with trembling, weakness, and incoordination. They may also experience cerebral signs ranging from slight clouding of the sensorium to actual loss of consciousness. Physical examination reveals a patient in mild to severe circulatory collapse with a pale, moist, even clammy skin and a rapid (120 to 200 beats per minute at rest), thready pulse. Systolic blood pressure may be normal at the time of examination, but prior to the onset of the illness and while at work, it may have been quite elevated (180 mm, Hg or higher) and then may have fallen precipitously while work continued. The pulse pressure, however, generally remains decreased at the time of examination. Oral temperature may be normal or only slightly elevated, but rectal temperature usually is found to be elevated in the range of 100°F to 102°F (37.5°C to 38.9°C). It may be even higher depending on the type and duration of physical activity prior to the onset of illness. In the water restriction type of illness, urine volume is small and the urine is highly concentrated. In the salt restriction type, urine is much less concentrated with the chloride level being less than three grams per liter.

Patients with heat exhaustion generally respond rapidly once they have been removed to a cool place and have rested. Their water deficit should be restored. If strenuous exertion preceded the onset of illness, and if examination of their urine indicates a salt deficiency, cautious administration of 0.1 percent salt solution, orally, or physiological saline, intravenously, may accelerate recovery. Patients should remain at rest until urine volume and salt content indicate that salt and water balances have been restored. Immediate return to duty is inadvisable except in the mildest cases. A 24 to 48-hour period of limited duty for personnel recovering from an
episode of severe heat exhaustion is recommended.

2. Heat Cramps. Heat cramps are painful spasms of muscles used during work (arms, legs, or abdominal) with their onset being observed during exertion or after work hours. They may occur in conjunction with heat exhaustion, or they may occur as an isolated illness and with normal body temperature. The patient has usually sweated heavily during hot work, experienced thirst, and drunk copious quantities of water without replacing salt loss. The patient presents with cool, moist skin, and their temperature is normal or only slightly elevated. Muscular soreness must be differentiated from that occurring in association rhabdomyolysis, which is accompanied by muscle necrosis often resulting in tea-colored urine. Once the diagnosis of heat cramps is confirmed by serum/urine chemistries, treatment consists of medication for pain and restoration of the salt deficit by 0.1 percent salt solution, orally, or physiological saline, intravenously.

Skin Eruptions and Behavioral Disorders. Skin eruptions and behavioral disorders are summarized in Table 4.

The operational physician is encouraged to refer to NAVMED P-5052-5, "The Etiology, Prevention, Diagnosis, and Treatment of Adverse Effects of Heat" and NAVMED P-5010-3, Manual of Naval Preventive Medicine, Chapter 3: "Ventilation and Thermal Stress Ashore and Afloat" for a more detailed discussion of these topics.

Indices of Thermal Stress
In an effort to maintain maximum productivity of personnel while minimizing their likelihood of developing adverse reactions from exposure to thermal stress, attempts have been made throughout the last several decades to integrate the several environmental, physiological, and behavioral variables affecting heat transfer to humans from the environment into a simple index. Such attempts have not met with unqualified success. Such an index must take into consideration dry bulb, globe, and wet bulb temperature readings as well as air velocities at both ventilation duct opening and work location. In addition, workers' body temperatures, work loads, heart rates, and pre- and post-exposure body weights must be considered. Ideally, the resultant index should assess the individual worker's cardiovascular reserve under different degrees of heat stress.

Chapter 3, "Ventilation and Thermal Stress Ashore and Afloat," in the Manual of Naval Preventive Medicine (NAVMED P-5010-3), concisely describes various indices of thermal stress and summarizes their advantages and disadvantages. The Wet Bulb Globe Temperature Index (WBGT) and the Physiological Heat Exposure Limits (PHEL) are the most widely used thermal indices in the Navy.

Wet Bulb Globe Temperature Index
This index was developed in the late 1950's to provide a convenient method to assess, quickly and with minimum operator skills, conditions which imposed intolerable levels of thermal stress on military personnel. Fundamentally, the WBGT Index is an algebraic approximation of the Effective Temperature concept.
The WBGT Index is computed from readings of (1) a stationary wet bulb thermometer exposed to the sun and to the prevailing wind, (2) a black globe thermometer similarly exposed, and (3) a dry bulb thermometer shielded from the direct rays of the sun. It is important that the readings be taken in an area representative of the conditions to which the personnel will be exposed. Construction and assembly details for a WBGT Index field apparatus can be found in NAVMED P-5052-5. The temperature of the wet bulb is depressed compared to the dry bulb reading for the air temperature because of evaporation resulting from the natural motion of the ambient air. Therefore, a principal advantage in using the WBGT Index is that wind velocity does not have to be measured. Of the six formulae which have been developed to obtain the WBGT Index, only two are in common use:

\[
\text{WBGT Index} = (0.7 \times \text{WB}) + (0.2 \times \text{G}) + (0.1 \times \text{DB})
\]

\[
\text{WBGT Index} = (0.7 \times \text{WB}) + (0.3 \times \text{G})
\]

where

\[
\text{WB} = \text{wet-bulb temperature}
\]

\[
\text{G} = \text{globe temperature}
\]

\[
\text{DB} = \text{dry-bulb temperature}.
\]

### Table 5: WGBT Index guide.

<table>
<thead>
<tr>
<th>WBGT Index (°F)</th>
<th>Intensity of Physical Exertion</th>
</tr>
</thead>
<tbody>
<tr>
<td>78-81.9</td>
<td>Extremely intense physical exertion may precipitate heat exhaustion or heat stroke, therefore, caution should be taken.</td>
</tr>
<tr>
<td>82-84.9</td>
<td>Discretion required in planning heavy exercise for unseasoned personnel. This is a marginal limit of environmental heat stress.</td>
</tr>
<tr>
<td>85-87.9</td>
<td>Strenuous exercise and activity (i.e., close order drill) should be curtailed for new and unseasoned personnel during the first three weeks of heat exposure.</td>
</tr>
<tr>
<td>88-89.9</td>
<td>Strenuous exercise curtailed for all personnel with less than 12 weeks training in hot weather.</td>
</tr>
<tr>
<td>90 and above</td>
<td>Physical training and strenuous exercise suspended for all personnel (excludes operational commitment not for training)</td>
</tr>
</tbody>
</table>
The first formula was originally applied to outdoor environments and the second to indoor spaces. It has since been demonstrated that the first formula is reliable in both situations. Incidence of heat casualties has been reduced during Marine Corps recruit training by applying the WBGT Index. Table 5 outlines the recommendations for different levels of physical activities at several WBGT ranges. It applies especially to personnel during training and recreational exercises in hot weather. It is not a substitute for the Physiological Heat Exposure Limits (PHEL) curves (discussed below) whose application is more suitable for an industrial type exposure, nor is it necessarily useful in operational settings.

The development of a WBGT Meter, which is a compact electronic instrument that independently measures the dry-bulb, wet-bulb, and globe temperatures and air velocity, and translates these variables directly into the WBGT Index, has provided the operational physician with a portable means to assess thermal stress in any space aboard ship.

**Physiological Heat Exposure Limits (PHEL)**

Integration of human metabolic heat production with the WBGT Index has allowed for the refinement and redefinition of heat tolerance limits. These newer indices have been designated PHEL curves. The PHEL curves recognize that under conditions of maximum work and heat stress the heat strain, although readily apparent, will be reversible. These curves represent the results of Navy research on personnel whose ages ranged from 18 to 40 years. The end point designated as an acceptable tolerance limit was a rectal temperature of 102°F (38.9°C) or a rapid rate of temperature change. Time-weighted means were calculated for both metabolic heat production and WBGT exposure in order to allow for the subsequent development of a set of time maximum heat exposure limits. The experimental data from which the PHEL curves were derived allowed for the development of a related series of rest/work ratios for different degrees of physical activity.

The Physiological Heat Exposure Limits are maximum allowable standards, and they should be applied only in cases of short-term work exposures of up to eight-hours duration. The limits presume that no prior heat injury is present and that no cumulative heat fatigue exists prior to re-exposure. Whether or not these assumptions are valid has not as yet been demonstrated.

In the application of heat stress standards, sound health, physical conditioning for the specific task, and adequate rest and nutrition are essential in order to minimize the effects of heat stress. Drinking water should be unrestricted and readily available. Threshold WBGT values for the hottest two-hour period of the work shift should be determined based on the workload of personnel. Table 6 presents the WBGT threshold values useful in identifying heat stress levels at which sound preventive measures should be instituted. They should not be confused with PHEL curves which deal with maximum time-weighted-mean limitations on an individual's work capacity.
in hot environments.

**Table 6 Threshold WBGT values.**

<table>
<thead>
<tr>
<th>Work Load</th>
<th>WBGT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light work (time weighted mean metabolic rate of 152 kcal/hr)</td>
<td>86</td>
</tr>
<tr>
<td>Moderate work (time weighted mean metabolic rate of 192 kcal/hr)</td>
<td>82</td>
</tr>
<tr>
<td>Heavy work (time weighted mean metabolic rate of 232 kcal/hr)</td>
<td>77</td>
</tr>
</tbody>
</table>

Figure 9 illustrates a PHEL chart for practical use. Work levels corresponding to curves A, B, and C are given in Table 7. Table 8 combines the data presented in Figure 9 and Table 7 and describes Physiological Heat Exposure Limits as functions of WBGT Index and metabolic work rate. When conditions of thermal stress preclude the worker's completing an eight-hour shift, the PHEL for that particular WBGT Index is often referred to as "stay time." Adherence to exposure time limits permits adjustment of the work/rest cycle in order to allow the body to dissipate the heat stored during periods of activity. Cool rest areas should be provided to maximize the benefits of the rest period and to avoid cumulative heat fatigue. Environmental engineering can do much to modify the determinants of heat stress, but aboard ship, operational exigencies and economic constraints may mandate the use of the number and duration of exposures as the most practical and expedient measure for the prevention of heat illness.

**Table 7: Metabolic rates of PHEL curves.**

<table>
<thead>
<tr>
<th>PHEL Curves (t_{wm} metabolic rates)</th>
<th>Duties*</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;A&quot; (152 kcal/hr)</td>
<td><em>Water Level checkman</em> during other than heavy repair or casualty control activity.</td>
</tr>
<tr>
<td>&quot;B&quot; (192 kcal/hr)</td>
<td><em>Burnerman</em> during other than heavy repair or casualty control functions; <em>Messenger</em> during other than full power conditions or when continuous mobility is not required.</td>
</tr>
<tr>
<td>&quot;C&quot; (252 kcal/hr)</td>
<td><em>Messenger</em> during full power operations or other activities requiring continuous mobility; any personnel involved in heavy repair work requiring manual labor (e.g., pump disassembly); casualty control functions; laundry/scullery work assignments.</td>
</tr>
</tbody>
</table>
**Examples of assignments found aboard steam propulsion ships (NAVMED P-5010-3, 1974)**

Table 8: PHEL versus WBGT*.

<table>
<thead>
<tr>
<th>WBG T (°F)</th>
<th>Curve &quot;A&quot; (t_{wm} Metabolic Rate=152 kcal/hr)</th>
<th>Curve &quot;B&quot; (t_{wm} Metabolic Rate=192 kcal/hr)</th>
<th>Curve &quot;C&quot; (t_{wm} Metabolic Rate=252 kcal/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>84</td>
<td>8+</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>86</td>
<td>8+</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>88</td>
<td>7</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>90</td>
<td>6</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>92</td>
<td>5</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>94</td>
<td>4</td>
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<td>96</td>
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<tr>
<td>120</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>122</td>
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<td>0</td>
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</tr>
<tr>
<td>124</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

- Assumes no cumulative fatigue or predisposing illness prior to heat Stress exposure (NAVMED P-5010-3, 1974)
Figure 9: PHEL Curves.

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