III. HEAT BALANCE AND HEAT EXCHANGE

An essential requirement for continued normal body function is that the deep body core temperature be maintained within the acceptable range of about 37°C (98.6°F) ± 1°C (1.8°F). To achieve this body temperature equilibrium requires a constant exchange of heat between the body and the environment. The rate and amount of the heat exchange are governed by the fundamental laws of thermodynamics of heat exchange between objects. The amount of heat that must be exchanged is a function of (1) the total heat produced by the body (metabolic heat), which may range from about 1 kcal per kilogram (kg) of body weight per hour (1.16 watts) at rest to 5 kcal/kg body weight/h (7 watts) for moderately hard industrial work; and (2) the heat gained, if any, from the environment. The rate of heat exchange with the environment is a function of air temperature and humidity, skin temperature, air velocity, evaporation of sweat, radiant temperature, and type, amount, and characteristics of the clothing worn. Respiratory heat loss is generally of minor consequence except during hard work in very dry environments.

A. Heat Balance Equation

The basic heat balance equation is:

$$\Delta S = (M-W) \pm C \pm R - E$$

where:

ΔS = change in body heat content
$(M-W)$ = total metabolism - external work performed
C = convective heat exchange
R = radiative heat exchange
E = evaporative heat loss

To solve the equation, measurement of metabolic heat production, air temperature, air water-vapor pressure, wind velocity, and mean radiant temperature are required [2,7,11,12,13,14,15,16,17,18,19,20,21].

B. Modes of Heat Exchange

The major modes of heat exchange between man and the environment are convection, radiation, and evaporation. Other than for brief periods of body contact with hot tools, equipment, floors, etc., which may cause burns, conduction plays a minor role in industrial heat stress.

The equations for calculating heat exchange by convection, radiation, and evaporation are available in Standard International (SI) units, metric units, and English units. In SI units heat exchange is in watts per square meter of body surface (W/m^2). The heat-exchange equations are available in both metric and English units for both the seminude individual and the worker wearing conventional long-sleeved workshirt and trousers. The values are in kcal/h or British thermal units per hour (Btu/h) for the "standard worker" defined as one who weighs 70 kg (154 lbs) and has a body surface area of 1.8 m^2 (19.4 ft^2). For workers who are smaller or larger than

the standard worker, appropriate correction factors must be applied [13]. The equations utilizing the SI units for heat exchange by C, R, and E are presented in Appendix B.

For these as well as other versions of heat-balance equations, computer programs of different complexities have been developed. Some of them are commercially available.

1. Convection (C)

The rate of convective heat exchange between the skin of a person and the ambient air immediately surrounding the skin is a function of the difference in temperature between the ambient air (t_a) and the mean weighted skin temperature ($\bar{t}_{sk}$) and the rate of air movement over the skin (V_a). This relationship is stated algebraically for the "standard worker" wearing the customary one-layer work clothing ensemble as [13]:

$$C = 7.0\ V_a^{\ 0.6}\ (t_a - \bar{t}_{sk})$$

where: C = convective heat exchange, kcal/h

V_a = air velocity in meters per second (m/sec)

t_a = air temperature °C

$\bar{t}_{sk}$ = mean weighted skin temperature usually assumed to be 35°C (95°F)

when t_a >35°C, there will be a gain in body heat from the ambient air by convection;

when t_a <35°C, heat will be lost from the body to the ambient air by convection.

This basic convective heat-exchange equation in English units has been revised for the "standard man" wearing the customary one-layer work clothing ensemble as:

$$C = 0.65\ V_a^{\ 0.6}\ (t_a - \bar{t}_{sk})$$

where: C = convective heat exchange in Btu/h

V_a = air velocity in feet per minute (fpm)

t_a = air temperature °F

$\bar{t}_{sk}$ = mean weighted skin temperature usually assumed to be 95°F (35°C)

2. Radiation (R)

The radiative heat exchange is primarily a function of the temperature gradient between the mean radiant temperature of the surroundings ($\bar{t}_w$) and the mean weighted skin temperature ($\bar{t}_{sk}$). Radiant heat exchange is a function of the fourth power of the absolute temperature of the solid surroundings less the skin temperature $(T_w - T_{sk})^4$ but an acceptable approximation for the customary one-layer clothed individual is [13]:

$$R = 6.6\ (\bar{t}_w - \bar{t}_{sk})$$

R = radiant heat exchange kcal/h

$\bar{t}_w$= mean radiant temperature of the solid surrounding surface °C

$\bar{t}_{sk}$ = mean weighted skin temperature

For the customary one-layer clothed individual and English units, the equation becomes:

$$R = 15.0\ (\bar{t}_w - \bar{t}_{sk})$$

R = radiant heat exchange Btu/h

$\bar{t}_w$ = mean radiant temperature °F

$\bar{t}_{sk}$ = mean weighted skin temperature

3. Evaporation (E)

The evaporation of water (sweat) from the skin surface results in a heat loss from the body. The maximum evaporative capacity (and heat loss) is a function of air motion (V_a) and the water vapor pressure difference between the ambient air (p_a) and the wetted skin at skin temperature (p_{sk}). The equation for this relationship is for the customary one-layer clothed worker [13]:

$$E = 14V_a^{0.6}\ (p_{sk} - p_a)$$

E = Evaporative heat loss kcal/h

V_a = air speed, m/sec

p_a = water vapor pressure of ambient air, mmHg

p_{sk} = vapor pressure of water on skin assumed to be 42 mmHg at a 35°C skin temperature

This translates in English units for the customary one-layer clothed worker into:

$$E = 2.4V_a^{0.6}\ (p_{sk} - p_a)$$

E = Evaporative heat loss Btu/h

V_a = air velocity, fpm

p_a = water vapor pressure air, mmHg

p_{sk} = water vapor pressure on the skin assumed to be 42 mmHg at a 95°F skin temperature

C. Effects of Clothing on Heat Exchange

Clothing serves as a barrier between the skin and the environment to protect against hazardous chemical, physical, and biologic agents. A clothing system will also alter the rate and amount of heat exchange between the skin and the ambient air by convection, radiation, and evaporation. When calculating heat exchange by each or all of these routes, it is, therefore, necessary to apply correction factors that reflect the type, amount, and characteristics of the clothing being worn when the clothing differs substantially (i.e., more than one-layer and/or greater air and vapor impermeability) from the customary one-layer work clothing. This clothing efficiency factor (F_{cl}) for dry heat exchange is nondimensional [22,23,24]. In general, the thicker and greater the air and vapor impermeability of the clothing barrier layer or layers, the greater is its interference with convective, radiative, and evaporative heat exchange.

Calculating heat exchange, when it must be modified by the F_{cl}, is a time consuming and complex task that requires the use of a hand held programmable calculator [133]. Corrections of the REL and RAL to reflect the F_{cl} based on heat transfer calculation for a variety of environmental and metabolic heat loads and three clothing ensembles have been suggested [168]. The customary one layer clothing ensemble was used as the basis for comparisons with the other clothing ensembles. When a two layer clothing system is worn, the REL and RAL should be lowered by 2°C (3.8°F). When a partially air and/or vapor impermeable ensemble or heat reflective or protective aprons, leggings, gauntlets, etc. are worn, the REL and RAL should be lowered 4°C (7.2°F). These suggested corrections of the REL or RAL are scientific judgments that have not been substantiated by controlled laboratory studies or long term industrial experience.

In those workplaces where a vapor and air impermeable encapsulating ensemble must be worn, the WBGT is not the appropriate measurement of environmental heat stress. In these instances, the adjusted air temperature (t_{adb}) must be measured and used instead of the WBGT. Where the t_{adb} exceeds approximately 20°C (68°F) physiologic monitoring (oral temperature and/or pulse rate) is required. This physiologic monitoring must be conducted on a time schedule based upon metabolic heat production and t_{adb}. The suggested frequency of physiologic monitoring for moderate work varies from once every two hours at t_{adb} of 24°C (75°F) to every 15 minutes for moderate work at t_{adb} of 32°C (90°F) [169].

1. Clothing Insulation and Nonevaporative Heat Loss

Even without any clothing, there is a thin layer of still air (the boundary layer) trapped next to the skin. This external still air film acts as a layer of insulation against heat exchange between the skin and the ambient environment. Typically, without body or air motion this air layer (I_a) provides about 0.8 clo units of insulation. One clo unit of clothing insulation is defined as allowing 5.55 kcal/m^2/h of heat exchange by radiation and convection (H_{R+C}) for each °C of temperature difference between the skin (at a mean skin temperature $\bar{t}_{sk}$) and adjusted dry bulb temperature $t_{adb}= (t_a+\bar{t}_r)/2$. For the average man with 1.8 m^2 of surface area, the hourly heat exchange by radiation and convection (H_{R+C}) can be estimated as:

$$H_{R+C}=(10/clo)(\bar{t}_{sk}-t_{adb})$$

Thus, the 0.8 clo still air layer limits the heat exchange by radiation and convection for the nude standard individual to about 12.5 kcal/h (i.e., 10/0.8) for each °C of difference between skin temperature and air temperature. A resting individual in still air producing 90 kcal/h of metabolic heat will lose about 11 kcal/h (12%) by respiration and about the same by evaporation of the body water diffusing through the skin. The worker will then have to begin to sweat and lose heat by evaporation to eliminate some of the remaining 68 kcal/h of metabolic heat if the t_{adb} is less than 5.5°C below t_{sk} [14].

The still air layer is reduced by increasing air motion, reaching a minimal value of approximately 0.2 clo at air speeds above 4.5 m/sec (890 fpm or 10 mph). At this wind speed, 68 kcal/h can be eliminated from the skin without sweating at an air temperature only 1.4°C below skin temperature, i.e., 68/(10/0.2)=1.36°C.

Studies of clothing materials over a number of years have led to the conclusion that the insulation provided by clothing is generally a linear function of its thickness. Differences in fibers or fabric weave, unless these directly affect the thickness or the vapor or air permeability of the fabric, have only very minor effects on insulation.

The function of the fibers is to maintain a given thickness of still air in the fabric and block heat exchange. The fibers are more conductive than insulating; increasing fiber density (as when trying to fit two socks into a boot which has been sized to fit properly with one sock) can actually reduce the insulation provided [14].

The typical value for clothing insulation is 1.57 clo per centimeter of thickness (4 clo per inch). It is difficult to extend this generalization to very thin fabric layers or to fabrics which, like underwear, may simply occupy an existing still air layer of not more than 0.5 cm thickness. These thin layers show little contribution to the intrinsic insulation of the clothing unless there is (a) "pumping action" of the clothing layers by body motion (circulation of air through and between layers of clothing due to body movement); (b) compression of the clothing by pressure from other clothing, by

objects in contact with the body, or by external wind; or (c) penetration of some of the wind (as a function of the air permeability of the outer covering fabric) into the trapped air layer [14,24,25]. Table III-1 presents a listing for the intrinsic insulation contributed by adding each of the listed items of civilian clothing.

The total intrinsic insulation is not the sum of the individual items, but 80% of their total insulation value; this allows for an average loss of 20% of the sum of the individual items to account for the compression of one layer on the next. This average 20% reduction is a rough approximation which is highly dependent on such factors as nature of the fiber, the weave, the weight of the fabric, the use of foam or other nonfibrous layers, and the clothing fit and cut.

TABLE III-1.--Clo insulation units for individual items of clothing and formula for obtaining total insulation (I_{cl})

Clothing	Men	Clothing	Women
Underwear			
Sleeveless	0.06	Bra and Panties	0.05
T-shirt	0.09	Half slip	0.13
Underpants	0.05	Full slip	0.19
Torso			
Shirt		Blouse	
Light, short sleeve	0.14	Light	0.20[a]
long sleeve	0.22	Heavy	0.29[a]
Heavy, short sleeve	0.25	Dress	
long sleeve	0.29	Light	0.22[a,b]
(Plus 5% for tie or turtleneck)		Heavy	0.90[a,b]
Vest		Skirt	
Light	0.15	Light	0.10[b]
Heavy	0.29	Heavy	0.22[b]
Trousers		Slacks	
Light	0.26	Light	0.26
Heavy	0.32	Heavy	0.44
Sweater		Sweater	
Light	0.20[a]	Light	0.17[a]
Heavy	0.37[a]	Heavy	0.37[a]
Jacket		Jacket	
Light	0.22	Light	0.17
Heavy	0.49	Heavy	0.37
Footwear			
Socks		Stockings	
Ankle length	0.04	Any length	0.01
Knee-high	0.10	Panty hose	0.01
Shoes		Shoes	
Sandals	0.02	Sandals	0.02
Oxfords	0.04	Pumps	0.04
Boots	0.08	Boots	0.08

Total I_{cl} = 0.80 (individual items) plus the external air layer of 0.8

[a]Less 10% if short sleeve or sleeveless
[b]Plus 5% if below knee length, less 5% if above

Adapted from Reference 25.

In summary, insulation is generally a function of the thickness of the clothing ensemble, and this, in turn, is usually a function of the number of clothing layers. Thus, each added layer of clothing, if not compressed, will increase the total insulation. That is why most two-layer protective clothing ensembles exhibit quite similar insulation characteristics; most three-layer systems are comparable, regardless of some rather major differences in fiber or fabric type [14].

2. Clothing Permeability and Evaporative Heat Loss

Evaporative heat transfer through clothing tends to be affected linearly by the thickness of the ensemble. The moisture permeability index (i_m) is a dimensionless unit, with a theoretical lower limit value of 0 for a vapor- and air-impermeable layer and an upper value of 1 if all the moisture that the ambient environment can take up (as a function of the ambient air vapor pressure and fabric permeability) can pass through the fabric. Since moisture vapor transfer is a diffusion process limited by the characteristic value for diffusion of moisture through still air, values of i_m approaching 1 should be found only with high wind and thin clothing. A typical i_m value for most clothing materials in still air is less than 0.5 (e.g., i_m will range from 0.45 to 0.48). Water repellent treatment, very tight weaves, and chemical protective impregnations can reduce the i_m value significantly. However, even impermeable layers seldom reduce the i_m value to zero since an internal evaporation-condensation cycle is set up between the skin surface and the inner surface of the impermeable layer which effectively transfers some heat from the skin to the vapor barrier; this shunting, by passing heat across the intervening insulation layers, can be reflected as an i_m value of perhaps 0.08 even for a totally impermeable overgarment.

Very few fiber treatments have been found to improve the i_m index value of fabric layers; surfactants which increase the number of free hydroxyl (OH) radicals on the fiber surface or which somehow improve wicking appear to have improved the i_m value of a fabric. However, the ultimate evaporative heat transferred from the skin through the clothing and external air layers to the environment is <u>not</u> simply a function of the i_m, but is a function of the permeability index-insulation ratio (i_m/clo). The maximum evaporative heat exchange with the environment can be estimated for the H_{R+C} of a man with 1.8 m^2 of surface area, as:

$$HE_{max}=10 i_m/clo \times 2.2(p_{sk}-p_a)$$

The constant 2.2 is the Lewis number; p_{sk} is the water vapor pressure of sweat (water) at skin temperature (t_{sk}); and p_a is the water vapor pressure of the ambient air at air temperature, t_a. Thus, the maximum evaporative transfer tends to be a linear, inverse function of insulation if not further degraded by various protective treatments which range from total impermeability to water repellent treatments [14,20,26].

3. Physiologic Problems of Clothing

The percent of sweat-wetted skin surface area ($\underline{w}$) that will be required to eliminate the required amount of heat from the body by evaporation can be estimated simply as the ratio of the required evaporative cooling (E_{req}) and the maximum water vapor uptake capacity of the ambient air (E_{max}). A totally wetted skin = 100%.

$$\underline{w} = E_{req}/E_{max}$$

Some sweat-wetted skin is not uncomfortable; in fact, some sweating during exercise in heat increases comfort. As the extent of skin wetted with sweat approaches 20%, the sensation of discomfort begins to be noted. Discomfort is marked with between 20% and 40% wetting of the body surface, and performance decrements can appear; they become increasingly noted as $\underline{w}$ approaches 60%. Sweat begins to be wasted, dripping rather than evaporating at 70%; physiologic strain becomes marked between 60% and 80% $\underline{w}$. Increases of $\underline{w}$ above 80% result in limited tolerance even for physically fit, heat-acclimatized young workers. The above arguments indicate that any protective work clothing will pose some limitations on tolerance since, with I_a plus I_{clo} rarely below 2.5 clo, their i_m/clo ratios are rarely above 0.20 [20].

The physiologic problem with clothing, heat transfer, and work can be estimated from equations which describe the competition for the blood pumped by the heart. The cardiac output (CO) is the stroke volume (SV) (or volume of blood pumped per beat) times heart rate (HR) in beats per minute (b/min) (CO=SVxHR). The cardiac output increases essentially linearly with increasing work; the rate limiting process for metabolism is the maximum rate of delivery of oxygen to the working muscle via the blood supply. The blood supply (or cardiac output) is a function of HR times SV (HRxSV). It is expressed in liters per minute (L/min). In heat stress this total blood supply must be divided between the working muscles and the skin where the heat exchange occurs.

Stroke volume rapidly reaches a constant value for a given intensity of work. Thus, the work intensity, i.e., the rate of oxygen delivered to the working muscles, is essentially indicated by heart rate; the individual worker's maximum heart rate limits the ability to continue work. Conditions that impair the return of blood from the peripheral circulation to fill the heart between beats will affect work capacity.

The maximum achievable heart rate is a function of age and can be roughly estimated by the relationship: 220 b/min minus age in years [27,28]. Given equivalent HR at rest (e.g., 60 b/min), a 20-year-old worker's HR has the capacity to increase by 140 b/min, i.e., (220-20)-60 while a 60-year-old worker can increase his HR only 100 b/min, i.e., (220-60)-60. Since the demands of a specific task will be roughly the same for 20- and 60-year-old individuals who weigh the same and do the same amount of physical work, the decrease in HR increase capacity with age increases both the perceived and the actual relative physiologic strain of work on the older worker.

The ability to transfer the heat produced by muscle activity from the core of the body to the skin also is a function of the cardiac output. Blood passing through core body tissues is warmed by heat from metabolism during rest and work. The basic requirement is that skin temperature (t_{sk}) must be maintained at least 1°C (1.8°F) below deep body temperature (t_{re}) if blood that reaches the skin is to be cooled before returning to the body core. The heat transferred to the skin is limited, ultimately, by the cardiac output and by the extent to which t_{sk} can be maintained below t_{re}.

A worker's t_{re} is a function of metabolic heat production (M) (t_{re} = 36.7+0.004M) as long as there are no restrictions on evaporative and convective heat loss by clothing, high ambient vapor pressures, or very low air motion; e.g., at rest, if M = 105 watts, t_{re} is about 37.1°C (98.8°F). Normally, under the same conditions of unlimited evaporation, skin temperatures are below t_{re} by about 3.3°C + (0.006M); thus, at rest, when t_{re} is 37°C, the corresponding t_{sk} is about 33°C, i.e., 37-(3.3+0.6). This 3°-4°C difference between t_{re} and t_{sk} indicates that at rest each liter of blood flowing from the deep body to the skin can transfer approximately 4.6 watts or 4 kcal of heat to the skin. Since t_{re} increases and t_{sk} decreases due to the evaporation of sweat with increasing M, it normally becomes easier to eliminate body heat with increasing work since the difference between t_{re} and t_{sk} increases by about 1°C (1.8°F) per 100 watts (86 kcal) of increase in M (i.e., t_{re} up 0.4°C (0.7°F), and t_{sk} down 0.6°C (1.1°F) per 100 watts of M). Thus, at sustainable hard work (M=500 watts or 430 kcal/h), each liter of blood flowing from core to skin can transfer 9 kcal to the skin, which is 2.5 times that at rest [20,26].

Work under a heat-stress condition sets up a competition for cardiac output, particularly as the blood vessels in the skin dilate to their maximum and less blood is returned to the central circulation. Gradually, less blood is available in the venous return to fully fill the heart between beats, causing the stroke volume to decrease; therefore, heart rate must increase to maintain the same cardiac output.

For a fit, young workforce, the average work heart rate should be limited to about 110 b/min if an 8-hour workshift is to be completed; an average heart rate of 140 b/min for a maximum work time of 4 hours or less, and 160 b/min should not be maintained for more than 2 hours [29]. If the intensity of work results in a heart rate in excess of these values, the intensity of work should be reduced. Thus, heat added to the demands of work rapidly results in problems even in a healthy, young workforce. These problems are amplified if circulating blood volume is reduced as a result of inadequate water intake to replace sweat losses, which can average one liter an hour over an 8-hour workshift (or by vomiting, diarrhea, or diuresis).

The crisis point, heat exhaustion and collapse, is a manifestation of the inadequate blood supply to the brain; this occurs when cardiac output becomes inadequate, because of insufficient return of blood from

the periphery to fill the heart for each beat, or because of inadequate time between beats to fill the heart as heart rates approach their maxima.

Unfortunately, clothing interferes with heat loss from the skin, and skin temperature rises predictably with increased clothing. Because of the insulation induced rise in t_{sk} and the resultant limited ability to dissipate heat that has been transferred from the core to the skin, core temperature (t_{re}) also rises when clothing is worn. Another type of interference with heat loss from the skin arises when sweat evaporation is required for body cooling (i.e., when $M+H_{R+C}>0$), but is limited either by high ambient water vapor pressure, low wind, or low clothing permeability index (i_m/clo).

As E_{req} approaches E_{max}, skin temperature increases dramatically and deep body temperature begins to increase rapidly. Deep body temperatures above 38.0°C (100.4°F) are considered undesirable for an average industrial workforce. The risk of heat-exhaustion collapse is about 25% at a deep body temperature of 39.2°C (102.6°F) associated with a skin temperature of 38°C (100.4°F) (i.e., t_{sk} converging toward t_{re} and approaching the 1°C (1.8°F) limiting difference where one liter of blood can transfer only 1 or 2 kcal to the skin). At a similarly elevated t_{sk} where t_{re} is 39.5°C (103.1°F), there is an even greater risk of heat-exhaustion collapse, and as t_{re} approaches 40°C (104°F), with elevated skin temperatures, most individuals are in imminent danger of heat-induced illness. Finally, t_{re} levels above 41°C (105.8°F) are associated with heatstroke, a life-threatening major medical emergency. The competition for cardiac output is sorely exacerbated by hypohydration (limited stroke volume), by age (limited maximum heart rate), and by reduced physical fitness (compromised cardiac output). These work-limiting and potentially serious deep body temperatures are reached more rapidly when combinations of these three factors are involved.

As indicated in the above statements, maximum work output may be seriously degraded by almost any protective clothing worn during either heavy work in moderately cool environments or low work intensities in hot conditions, because of the clothing interfering with heat elimination. The heat-stress problem is also likely to be increased with any two-layer protective ensembles or any effective single-layer vapor barrier system for protection against toxic products, unless some form of auxiliary cooling is provided [20,26].

IV. BIOLOGIC EFFECTS OF HEAT

A. Physiologic Responses to Heat

1. The Central Nervous System

The central nervous system is responsible for the integrated organization of thermoregulation. The hypothalamus of the brain is considered to be the central nervous system structure which acts as the primary seat of control. In general terms, the anterior hypothalamus operates as a integrator and "thermostat" while the posterior hypothalamus provides a "set point" of the core or deep-body temperature and initiates the appropriate physiologic responses to keep the body temperature at the "set point" if the core temperature changes.

The anterior hypothalamus is the area which receives the information from receptors sensitive to changes in temperature in the skin, muscle, stomach, other central nervous system tissues, and elsewhere. In addition, the anterior hypothalamus itself contains neurons which are responsive to changes in temperature of the arterial blood serving the region. The neurons responsible for the transmission of the temperature information use monoamines among other neurotransmitters; this has been demonstrated in animals [30]. These monoamine transmitters are important in the passage of appropriate information to the posterior hypothalamus. Another neuronal transmitter is acetylcholine. It is known that the "set point" in the posterior hypothalamus is regulated by ionic exchanges.

The ratio of sodium to calcium ions is also important in thermoregulation. The sodium ion concentration in the blood and other tissues can be readily altered by exercise and by exposure to heat. However, the "set-point" hypothesis has recently generated considerable controversy [31].

When a train of neural traffic is activated from the anterior to the posterior hypothalamus, it is reasonable to suppose that once a "hot" pathway is activated, it will inhibit the function of the "cold" pathway and vice versa. However, there is a multiplicity of neural inputs at all levels in the central nervous system and many complicated neural "loops" undoubtedly exist.

The posterior hypothalamus, besides determining the "set-point," is also responsible for mobilizing the physiologic mechanisms required to maintain that temperature. In situations where the "set-point" temperature is exceeded, the circulation is controlled on a regional basis through the sympathetic nervous system to dilate the cutaneous vascular bed and thereby increase skin blood flow, and if necessary, the sweating mechanism is invoked. These mechanisms are designed to dissipate heat in an attempt to return the "set-point" to its original level.

A question that must be addressed is the difference between a physiologically raised body temperature and a fever During a fever, it

is considered that the "set-point" is elevated as determined by the posterior hypothalamus. At the onset of a fever, the body invokes heat-conservation mechanisms (such as shivering and cutaneous vasoconstriction) in order to raise the body temperature to its new "set-point" [30]. In contrast, during exercise in heat, which may result in an increase in body temperature, there is no change in "set-point" temperature, and only heat-dissipation mechanisms are invoked. Once a fever is induced, the elevated body temperature appears to be normally controlled by the usual physiologic processes around its new and higher "set-point."

2. Muscular Activity and Work Capacity

The muscles are by far the largest single group of tissues in the body, representing some 45% of the body weight. The bony skeleton, on which the muscles operate to generate their forces, represents a further 15% of the body weight. The bony skeleton is relatively inert in terms of metabolic heat production. However, even at rest, the muscles produce about 20-25% of the body's total heat production. The amount of metabolic heat produced at rest is quite similar for all individuals when it is expressed per unit of surface area or of lean or fat-free body weight. On the other hand, the heat produced by the muscles during exercise can be much higher, all of which must be dissipated if a heat balance is to be maintained. The heat load from metabolism is, therefore, widely variable, and it is during work in hot environments (which imposes its own heat load or restricts heat dissipation) that the greatest challenge to normal thermoregulation exists.

The proportion of maximal aerobic capacity ($\dot{V}O_2max$) needed to do a specific job is important for several reasons. First, the cardiovascular system must respond with an increased cardiac output which at levels of work of up to about 40% $\dot{V}O_2max$ is brought about by an increase in both stroke volume and heart rate. When maximum stroke volume is reached, additional increases in cardiac output can be achieved solely by increased heart rate (which itself has a maximum value). Further complexities arise when high work intensities are sustained for long periods, particularly when work is carried out in hot surroundings. Second, muscular activity is associated with an increase in muscle temperature, which then is associated with an increase in core temperature, with attendant influences on the thermoregulatory controls. Third, at high levels of exercise even in a temperate environment, the oxygen supply to the tissues may be insufficient to meet the oxygen needs of the working muscles completely.

In warmer conditions, an adequate supply of oxygen to the tissues may become a problem even at moderate work intensities because of competition for blood distribution between the working muscle and the skin. Because of the lack of oxygen, the working muscles must then begin to draw on their anaerobic reserves, deriving energy from the oxidation of glycogen in the muscles. That event leads to the accumulation of lactic acid which may be associated with the development of muscular fatigue. As the proportion of $\dot{V}O_2max$ used increases further, anaerobic metabolism assumes a relatively greater proportion of

the total muscular metabolism. An oxygen "debt" occurs when oxygen is required to metabolize the lactic acid that accumulates in the muscles. This "debt" must be repaid during the rest period. In hot environments, the recovery period is prolonged as the elimination of both the heat and the lactic acid stored in the body has to occur and water loss must be replenished. These occurrences may take 24 hours or longer [31,32].

It is well established that, in a wide range of cool to warm environments, 5°-29°C (41°-84.2°F), the deep body temperature rises during exercise to a similar equilibrium value in subjects working at the same proportion of $\dot{V}O_2max$ [18,33]. However, two individuals doing the same job and working at the same absolute load level and who have widely different $\dot{V}O_2max$ values will have quite different core temperatures. Currently, recommendations for an acceptable proportion of $\dot{V}O_2max$ for daily industrial work vary from 30-40% of the $\dot{V}O_2max$, which in comfortably cool surroundings [34] is associated with rectal temperatures of, respectively, 37.4° and 37.7°C (99.3°-99.9°F), while work at 50% $\dot{V}O_2max$ yields a rectal temperature of 38°C (100.4°F) in the absence of heat stress.

In addition to sex- and age-related variability, the interindividual variability of $\dot{V}O_2max$ is high; therefore, the range of $\dot{V}O_2max$ to include 95 of every 100 individuals will be $\pm$20% of the mean $\dot{V}O_2max$ value. Differences in body weight (particularly the muscle mass) can account for about half that variability when $\dot{V}O_2max$ is expressed as mL O_2/kg/min, but the source of the remaining variation has not been precisely identified. Age is associated with a reduction in $\dot{V}O_2max$ after peaking at about 20 years of age, and falling in healthy individuals by nearly 10% each decade after age 30. The decrease in $\dot{V}O_2max$ with age is less in individuals who have maintained a higher degree of physical fitness. Women have levels of $\dot{V}O_2max$ which average about 70% of that for men in the same age group due to lower absolute muscle mass [34]. There are many factors to consider with respect to the deep body temperature when the same job is done by both men and women of varying body weights, ages, and work capacities.

Other sources of variability when individuals work in hot environments are differences in circulatory system capacity, in sweat production, and in the ability to regulate electrolyte balance, each of which may be large.

Previously, work performance was comprehensively reviewed [35,36], and little or no new data have been published. Work capacity is reduced to a limited extent in hot surroundings if body temperature is elevated. That reduction becomes greater as the body temperature is increased. The $\dot{V}O_2max$ is not reduced by hypohydration itself (except for severe hypohydration) so that its reduction in hot environments seems to be principally a function of body temperature. Core temperature must be above 38°C (100.4°F) before a reduction is noticeable; however, a rectal temperature of about 39°C (102.2°F) may result in some reduction of $\dot{V}O_2max$.

The capacity for prolonged exercise of moderate intensity in hot environments is adversely affected by hypohydration which may be associated with a reduction of sweat production and a concomitant rise in rectal temperature and heart rate. If the total heat load is high and the sweat rate is high, it is increasingly more difficult to replace the water lost in the sweat (750-1,000 mL/h). The thirst mechanism is usually not strong enough to drive one to drink the large quantities of water needed to replace the water lost in the sweat. Existing evidence supports the concept that as the body temperature increases in a hot working environment, the endurance for physical work is decreased. Similarly, as the environmental heat stress increases, many of the psychomotor, vigilance, and other experimental psychologic tasks show decrements in performance [37,38,39,40,41]. The decrement in performance may be at least partly related to increases in core temperature and hypohydration. When the rectal temperature is raised to 38.5°-39.0°C (101.3°-102.2°F), associated with heat exhaustion, there are many indications of disorganized central nervous system activity, including poor motor function, confusion, increased irritability, blurring of vision, and changes in personality, prompting the unproven suggestion that cerebral anoxia (reduced oxygen supply to the brain) may be responsible [4,39,42].

3. Circulatory Regulation

The circulatory system is the transport mechanism responsible for delivering oxygen and foodstuffs to all tissues and for transporting unwanted metabolites and heat from the tissues. However, the heart cannot provide enough cardiac output to meet both the peak needs of all of the body's organ systems and the need for dissipation of body heat. The autonomic nervous system and endocrine system control the allocation of blood flow among competing organ systems.

During exercise, there is widespread, sympathetic circulatory vasoconstriction initially throughout the body, even in the cutaneous bed. The increase in blood supply to the active muscles is assured by the action of locally produced vasodilator substances which also inhibit (in the blood vessels supplying the active muscles) the increased sympathetic vasoconstrictor activity. In inactive vascular beds, there is a progressive vasoconstriction with the severity of the exercise. This is particularly important in the large vascular bed in the digestive organs, where venoconstriction also permits the return of blood sequestered in its large venous bed, allowing up to 1 liter of blood to be added to the circulating volume [36].

If the need to dissipate heat arises, the autonomic nervous system reduces the vasoconstrictor tone of the cutaneous vascular bed, followed by "active" dilation which occurs by a mechanism which is, at present, unclear. The sweating mechanism and an unknown critical factor that causes the importantly large dilation of the peripheral blood vessels in the skin are mutually responsible for man's remarkable thermoregulatory capacity in the heat.

When individuals are exposed to continuous work at high proportions of VO_2max or to continuous work at lower intensities in hot surroundings, the cardiac filling pressure remains relatively constant, but the central venous blood volume decreases as the cutaneous vessels dilate. The stroke volume falls gradually, and the heart rate must increase to maintain the cardiac output. The effective circulatory volume also decreases, partly due to hypohydration as water is lost in the sweat and partly as the thermoregulatory system tries to maintain an adequate circulation to meet the needs of the exercising muscles as well as the circulation to the skin [36].

4. The Sweating Mechanism

The sweat glands are found in abundance in the outer layers of the skin. They are stimulated by cholinergic sympathetic nerves and secrete a hypotonic watery solution onto the surface of the skin. Sweat production at rates of about 1 L/h has been recorded frequently in industrial work and represents a large potential source of cooling if all the sweat is evaporated; each liter of sweat evaporated from the skin surface represents a loss of approximately 580 kcal (2320 Btu or 675 W) of heat to the environment. Large losses of water by sweat also pose a potential threat to successful thermoregulation, because a progressive depletion of body water content occurs if the water lost is not replaced; hypohydration by itself affects thermoregulation and results in a rise of core temperature.

An important constituent of sweat is salt or sodium chloride. In most circumstances, a salt deficit does not readily occur, because our normal diet provides 8-14 g/d. However, the salt content of sweat in unacclimatized individuals may be as high as 4 g/L, while for the acclimatized individual it will be reduced to 1 g/L or less. It is possible for a heat-unacclimatized individual who consumes a restricted salt diet to develop a negative salt balance. In theory, a prolonged negative salt balance with a large fluid intake could result in a need for moderate supplementation of dietary salt. If there is a continuing negative salt balance, acclimatization to heat is diminished. However, salt supplementation of the normal diet is rarely required except possibly for heat-unacclimatized individuals during the first 2 or 3 days of heat exposure [32]. By the end of the third day of heat exposure, a significant amount of heat acclimatization will have occurred with a resulting decrease in salt loss in the sweat and urine and a decrease in salt requirement. In view of the high incidence of elevated blood pressure in the U.S. worker population and the relatively high salt content of the average U.S. diet, even in those who watch salt intake, recommending increased salt intake is probably not warranted. Salt tablets can irritate the stomach and should not be used [43]. Heavier use of salt at meals has been suggested for the heat-unacclimatized individual during the first 2-3 days of heat exposure (if not on a restricted salt diet by physician's orders). Carefully induced heat acclimatization will reduce or eliminate the need for salt supplementation of the normal diet.

Because potassium is lost in sweat, there can be a serious depletion of potassium when workers, who are unacclimatized, suddenly have to work hard in hot climates; marked depletion of potassium can lead to serious physiologic consequences including the development of heatstroke [4]. A high table salt intake may increase potassium loss. However, potassium loss is usually not a problem, except for individuals taking diuretics, because potassium is present in most foods, particularly meats and fruits [32]. Since diuretics cause potassium loss, workers taking such medication while working in a hot environment may require special medical supervision.

The rate of evaporation of sweat is controlled by the difference in water vapor pressure on the sweat-wetted skin surface and the air layer next to the skin and by the velocity of air movement over the skin. As a consequence, hot environments with increasing humidity limit the amount of sweat that can be evaporated. Sweat that is not evaporated drips from the skin and it does not result in any heat loss from the body. It is deleterious, because it does represent a loss of water and salt from the body.

a. Water and Electrolyte Balance and the Influence of Endocrines

It is imperative to replace the water lost in the sweat. It is not uncommon for workers to lose 6-8 quarts of sweat during a working shift in hot industries. If the lost water is not replaced, there will be a progressive decrease of body water with a shrinkage not only of the extracellular space and interstitial and plasma volumes but also of water in the cells. There is clear evidence that the amount of sweat production depends on the state of hydration [4,32,35] so that progressive hypohydration results in a lower sweat production and a corresponding increase in body temperature, which is a dangerous situation.

Sweat lost in such quantities is often difficult to replace completely as the day's work proceeds, and it is not uncommon for individuals to register a water deficit of 2-3% or greater of the body weight. During exercise in either cool or hot environments, a correlation has been reported between the elevation of rectal temperature and the percentage of water deficit in excess of 3% of body weight [44]. Because the normal thirst mechanism is not sensitive enough to ensure a sufficient water intake [32], every effort should be made to encourage individuals to drink water or low-sodium noncarbonated beverages. The fluid should be as palatable as possible at 10°-15°C or 50°-60°F. Small quantities taken at frequent intervals, about 150-200 mL or 5-7 ozs every 15-20 minutes, is a more effective regimen for practical fluid replacement than the intake of 750 mL or more once an hour. Communal drinking containers should be prohibited. Individuals are seldom aware of just how much sweat they produce or how much water is needed to replace that lost in the sweat; 1 L/h is not an uncommon rate of water loss. With suitable instruction concerning the problems of not drinking enough to replace water lost in sweat, most individuals

will comply. Those who do not replace water loss while at work, will at least diminish the amount of water deficit they generate and will usually replenish that deficit in off-duty hours.

Two hormones are important in thermoregulation, the antidiuretic hormone (ADH) and aldosterone. A variety of stimuli encourages the synthesis and release of those hormones, such as changes in plasma volume, plasma concentration of sodium chloride, etc. ADH is released by the pituitary gland, which has direct neural connections with the hypothalamus but may receive neural input from other sources. Its function is to reduce water loss by the kidney, but it has no effect on the water loss through sweat glands. Aldosterone is released from the adrenal glands and reduces salt lost both in the kidney and in the sweat glands.

b. Dietary Factors

There is no reason to believe that a well-balanced diet for work in temperate environments should not suffice for hot climates.

A very high protein diet might increase the obligatory urine output for nitrogen removal, and thus increase water intake requirements [31,32]. The importance of water and salt balance has been emphasized above, and the possibility that it might be desirable to supplement the diet with potassium has also been considered. In some countries where the normal diet is low or deficient in Vitamin C, supplementation may enhance heat acclimatization and thermoregulatory function [45].

5. Acclimatization to Heat

When workers are unexpectedly exposed to hot work environments, they readily show signs of distress and discomfort; develop increased core temperatures and heart rates; complaints of headache, giddiness, or nausea; and suffer other symptoms of incipient heat exhaustion [4,8,39,44,46,47,48]. On first exposure, they may faint. On repeated exposure there is a marked adaptation in which the principal physiologic benefit appears to result from an increased sweating efficiency (earlier onset, greater sweat production, and lower electrolyte concentration) and a concomitant stabilization of the circulation, so that after daily heat exposure for 7-10 days, the individuals perform the work with a much lower core temperature and heart rate and higher sweat rate (i.e., a reduced thermoregulatory strain) and with none of the distressing symptoms that were experienced at first. During that period there is at first a rapid expansion of plasma volume, so that even though there is a hemoconcentration throughout the exposure to heat, the plasma volume at the end of the heat exposure in the acclimatized state is often equal to or in excess of the value before the first day of heat exposure. Acclimatization to heat is an unsurpassed example of physiologic adaptation which is well demonstrated in laboratory experiments and field experience [48,49]. However, acclimatization does not necessarily mean that the individuals can work above the Prescriptive Zone as effectively as below it (see Appendix A) [18].

Full heat acclimatization occurs with relatively brief daily exposures to working in the heat. It does not require exposure to heat at work and rest for the entire 24 h/d; in fact, such excessive exposures may be deleterious, because it is hard for individuals without heat acclimatization experience to replace all of the water lost in sweat. The minimum exposure time for achieving heat acclimatization is a continuous exposure of about 100 minutes daily [4,49]. Some daily period of relief from exposure to heat, in air-conditioned surroundings, is beneficial to the well-being of the individuals if for no other reason, they find it hard to rest effectively in hot surroundings [44].

The level of acclimatization is relative to the initial level of individual physical fitness and the total heat stress experienced by the individual. Thus, a worker who does only light work indoors in a hot climate will not achieve the level of acclimatization needed to work outdoors with the additional heat load from the sun or to do harder physical work in the same hot environment indoors.

Failure to replace the water lost in sweat will retard or even prevent the development of the physiologic adaptations described. In spite of the fact that acclimatization will be reasonably well maintained for a few days of nonheat exposure, absence from work in the heat for a week or more results in a significant loss in the beneficial adaptations. However, usually heat acclimatization can be regained in 2 to 3 days upon return to a hot job [47,49]. Heat acclimatization appears to be better maintained by individuals who are physically fit [50].

The total sweat production increases with acclimatization, and sweating begins at a lower skin temperature. The cutaneous circulation and circulatory conductance decreases with acclimatization, reflecting the reduction in the proportion of cardiac output that must be allocated for thermoregulation, because of the more efficient sweating mechanism. There is still no clear explanation of how these events are brought about and what the underlying mechanisms are that alter the cardiovascular and thermoregulatory responses so dramatically. It is clear, however, that during exercise in heat, the production of aldosterone is increased to conserve salt from both the kidney and the sweat glands, while an increase in antidiuretic hormone conserves the amount of water lost through the kidneys.

It is obvious from the foregoing description that sudden seasonal shifts in environmental temperature may result in thermoregulatory difficulties for exposed workers. At such times, cases of heat disorder may occur, even for acclimatized workers, if the outside environment becomes very hot.

Acclimatization to work in hot, humid environments provides adaptive benefits which also apply in hot, desert environments, and vice versa; the qualifying factor appears to be the total heat load experienced by the individual.

6. Other Related Factors

a. Age

The aging process results in a more sluggish response of the sweat glands, which leads to a less effective control of body temperature. Aging also results in a curiously increased high level of skin blood flow associated with exposure to heat. The cause of this remains undetermined, but implies an impaired thermoregulatory mechanism possibly related to a reduced efficiency of the sympathetic nervous system [18,27,28]. For women, it has been found that the skin temperature increases with age in moderate and high heat loads, but not in low heat loads [27,28]. When two groups of male coal miners of average age 47 and 27 years, respectively, worked in several comfortable or cool environments, they showed little difference in their responses to heat near the REL with light work, but in hotter environments the older men showed a substantially greater thermoregulatory strain than their younger counterparts; the older men also had lower aerobic work capacities [51]. In analyzing the distribution of 5 years' accumulation of data on heatstroke in South African gold mines, Strydom [52] found a marked increase in heatstroke with increasing age of the workers. Thus, men over 40 years of age represented less than 10% of the mining population, but they accounted for 50% of the fatal and 25% of the nonfatal cases of heatstroke. The incidence of cases per 100,000 workers was 10 or more times greater for men over 40 years than for men under 25 years of age. In all the experimental and epidemiologic studies described above, the workers had been medically examined and were considered free of disease. Total body water decreases with age which may be a factor in the observed higher incidence of fatal and nonfatal heatstroke in the older group.

b. Gender

Purely on the basis of a lower aerobic capacity, the average woman, similar to a small man, is at a disadvantage when she has to perform the same job as the average-sized man. While all aspects of heat tolerance in women have not been fully examined, their thermoregulatory capacities have been. When they work at similar proportions of their VO_2max, the women perform either similarly or only slightly less well than men [53,54,55,56]. There seems to be little change in thermoregulatory capacities at different times during their menstrual cycles [57].

c. Body Fat

It is well established that obesity predisposes individuals to heat disorders [4]. The acquisition of fat means that additional weight must be carried, thereby calling for a greater expenditure of energy to perform a given task and use of a greater proportion of the VO_2max. In addition, the body surface to body weight ratio (m^2 to kg) becomes less favorable for heat dissipation. Probably more important is the lower physical fitness and decreased maximum

work capacity and cardiovascular capacity frequently associated with obesity. The increased layer of subcutaneous fat provides an insulative barrier between the skin and the deep-lying tissues. The fat layer theoretically would reduce the direct transfer of heat from the muscles to the skin [58].

d. Drugs

(1) Alcohol

Alcohol has been commonly associated with the occurrence of heatstroke [4]. It is a drug which interferes with central and peripheral nervous function and is associated with hypohydration by suppressing ADH production. The ingestion of alcohol prior to or during work in the heat should not be permitted, because it reduces heat tolerance and increases the risk of heat illnesses.

(2) Therapeutic Drugs

Many drugs prescribed for therapeutic purposes can interfere with thermoregulation [59]. Some of these drugs are anticholinergic in nature or involve inhibition of monoamine oxidative reactions, but almost any drug that affects central nervous system (CNS) activity, cardiovascular reserve, or body hydration could potentially affect heat tolerance. Thus, a worker who requires therapeutic medications should be under the supervision of a physician who understands the potential ramifications of drugs on heat tolerance. In such instances, a worker taking therapeutic medications who is exposed only intermittently or occasionally to a hot environment should seek the guidance of a physician.

(3) Social Drugs

It is hard to separate drugs used therapeutically from those which are used socially. Nevertheless, there are many drugs other than alcohol which are used on social occasions. Some of those have been implicated in cases of heat disorder, sometimes leading to death [59].

e. Nonheat Disorders

It has long been recognized that individuals suffering from degenerative diseases of the cardiovascular system and other diseases such as diabetes or simple malnutrition are in extra jeopardy when they are exposed to heat, and when a stress is imposed on the cardiovascular system. The outcome is readily seen during sudden or prolonged heat waves in urban areas where there is a sudden increase in mortality especially among older individuals who supposedly have age-related reduced physiologic reserves [4,60,61,62]. In prolonged heat waves, the mortality is higher in the early phase of the heat wave [60,61]. While acclimatization may

play a part in the decrease in mortality during the later part of a prolonged heat wave, the increased death rate in the early days of a heat wave may reflect an "accelerated mortality," with the most vulnerable more likely to succumb at that time rather than more gradually as a result of degenerative diseases.

f. Individual Variation

In all experimental studies of the responses of humans to hot environmental conditions, a wide variation in responses has been observed. These variations are seen not only between different individuals but also to some extent in the same individual exposed to high stress on different occasions. Such variations are not totally understood. It has been shown [47] that the influence of body size and its relationship to aerobic capacity in tolerance to heat could account for about half of the variability, leaving the remainder to be accounted for. Possibly, changes in hydration and salt balance might be responsible for some of the remaining variability [63]. However, the degree of variability in tolerance to hot environments remains a vexing problem.

7. Heat-Related Health and Safety Effects

The incidence of work-related heat illness in the United States is not documented by an occupational injury/illness surveillance system. However, the Supplementary Data System (SDS) maintained by the U.S. Bureau of Labor Statistics (BLS) contains coded information from participating states about workers' heat illness compensation claims.

Those workers' compensation cases which were coded to indicate that the disorder was a heat illness which occurred during 1979 have been analyzed by Jensen [64]. The results indicate that the industries with the highest rate of reported compensation cases for heat illness per 100,000 workers are agriculture (9.16 cases/100,000 workers), construction (6.36/100,000), and mining (5.01/100,000). The other industrial divisions had case rates of fewer than 3 per 100,000 workers. Dinman et al. [65] reported an incidence rate of 6.2 per 1,000,000 man-hours in a study of three aluminum plants during a May-September observation period. Minard reported 1 per 1,000 workers had heat-related illnesses during a 5-month period in three aluminum and two steel plants (presumably the same plants reported by Dinman et al.) [66]. Janous, Horvath, and Horvath and Colwell also reported an increased incidence of heat illnesses in the iron and steel industry [67,68,69].

In 1979, the total U.S. incidence of work-related heat illnesses for which the worker lost at least one day of work following the day of onset (lost-workday cases) was estimated to be 1,432 cases [64]. The estimation is based on the assumption that the proportion of cases of a particular kind of injury in the SDS data base is equivalent to the proportion of cases for that kind of injury or lost-workday cases nationwide. It has been shown that when the thermal environmental conditions of the workplace exceed temperatures which are typically

preferred by most people, the safety-related behavior of workers is detrimentally affected and increased exponentially with increasing heat load [70,71].

In an analysis by cause (chemical and physical agent) of the occupational illnesses and injuries reported in the 1973 State of California, Division of Labor Statistics and Research, Dukes-Dobos [71] found that 422 cases resulting in some lost time were the result of "heat and humidity," which was the most frequent physical agent cause. Forty-seven of these cases were hospitalized and three died. Chlorine was the most frequently cited chemical hazard with 529 lost-time cases; 48 were hospitalized, and there were no deaths. Other chemical and physical agents such as ammonia, trichloroethylene, noise, benzene, lead, and chromium were less frequently involved than heat. Janous [67] reported increased accidents in heat-exposed steelworkers.

B. Acute Heat Disorders

A variety of heat disorders can be distinguished clinically when individuals are exposed to excessive heat [4,18,72,73,74,75]. These disorders range from simple postural heat syncope (fainting) to the complexities of heatstroke. The heat disorders are interrelated and seldom occur as discrete entities. A common feature in all the heat-related disorders (except simple postural heat syncope) is some degree of elevated body temperature which may then be complicated by deficits of body water. The prognosis depends on the absolute level of the elevated body temperature, the promptness of treatment to lower the body temperature, and the extent of deficiency or imbalance of fluids or electrolytes. A summary of classification, clinical features, prevention, and treatment of heat illnesses is presented in Table IV-1. Recently, a new scheme for differential diagnosis of heat-induced illnesses has been published [72].

TABLE IV-1.--Classification, medical aspects, and prevention of heat illness

Category and clinical features	Predisposing factors	Underlying physiologic disturbance	Treatment	Prevention
1. Temperature Regulation Heatstroke				
Heatstroke: (1) Hot dry skin usually red, mottled or cyanotic; (2) t_{re}, 40.5°C (104°F) and over; (3) confusion, loss of consciousness, convulsions, t_{re} continues to rise; fatal if treatment delayed	(1) Sustained exertion in heat by unacclimatized workers; (2) Lack of physical fitness and obesity; (3) Recent alcohol intake; (4) Dehydration; (5) Individual susceptibility; and (6) Chronic cardiovascular disease	Failure of the central drive for sweating (cause unknown) leading to loss of evaporative cooling and an uncontrolled accelerating rise in t_{re}, there may be partial rather than complete failure of sweating	Immediate and rapid cooling by immersion in chilled water with massage or by wrapping in wet sheet with vigorous fanning with cool dry air, avoid overcooling, treat shock if present	Medical screening of workers, selection based on health and physical fitness, acclimatization for 5-7 days by graded work and heat exposure, monitoring workers during sustained work in severe heat
2. Circulatory Hypostasis Heat Syncope				
Fainting while standing erect and immobile in heat	Lack of acclimatization	Pooling of blood in dilated vessels of skin and lower parts of body	Remove to cooler area, rest recumbent position, recovery prompt and complete	Acclimatization, intermittent activity to assist venous return to heart
3. Water and/or Salt Depletion				
(a) Heat Exhaustion				
(1) Fatigue, nausea, headache, giddiness; (2) Skin clammy and moist; complexion pale, muddy, or hectic	(1) Sustained exertion in heat; (2) Lack of acclimatization; and (3) Failure to replace water lost in sweat	(1) Dehydration from deficiency of water; (2) Depletion of circulating blood volume; (3) Circulatory strain from	Remove to cooler environment, rest recumbent position, administer fluids by mouth, keep at rest	Acclimatize workers using a breaking-in schedule for 5 to 7 days, supplement dietary salt only

(continued)

TABLE IV-1.--Classification, medical aspects, and prevention of heat illness

Category and clinical features	Predisposing factors	Underlying physiologic disturbance	Treatment	Prevention
flush; (3) May faint on standing with rapid thready pulse and low blood pressure; (4) Oral temperature normal or low but rectal temperature usually elevated (37.5°-38.5°C) (99.5°-101.3°F); water restriction type: urine volume small, highly concentrated; salt restriction type: urine less concentrated, chlorides less than 3 g/L		competing demands for blood flow to skin and to active muscles	until urine volume indicates that water balances have been restored	during acclimatization, ample drinking water to be available at all times and to be taken frequently during work day
(b) Heat Cramps				
Painful spasms of muscles used during work (arms, legs, or abdominal); onset during or after work hours	(1) Heavy sweating during hot work; (2) Drinking large volumes of water without replacing salt loss	Loss of body salt in sweat, water intake dilutes electrolytes, water enters muscles, causing spasm	Salted liquids by mouth, or more prompt relief by I-V infusion	Adequate salt intake with meals; in unacclimatized workers supplement salt intake at meals
4. Skin Eruptions				
(a) Heat Rash (miliaria rubra; "prickly heat")				
Profuse tiny raised red vesicles (blister-like) on affected areas, pricking sensations during heat exposure	Unrelieved exposure to humid heat with skin continuously wet with unevaporated sweat	Plugging of sweat gland ducts with retention of sweat and inflammatory reaction	Mild drying lotions, skin cleanliness to prevent infection	Cool sleeping quarters to allow skin to dry between heat exposures

(continued)

TABLE IV-1.--Classification, medical aspects, and prevention of heat illness

Category and clinical features	Predisposing factors	Underlying physiologic disturbance	Treatment	Prevention
(b) Anhidrotic Heat Exhaustion (miliaria profunda)				
Extensive areas of skin which do not sweat on heat exposure, but present gooseflesh appearance, which subsides with cool environments; associated with incapacitation in heat	Weeks or months of constant exposure to climatic heat with previous history of extensive heat rash and sunburn	Skin trauma (heat rash; sunburn) causes sweat retention deep in skin, reduced evaporative cooling causes heat intolerance	No effective treatment available for anhidrotic areas of skin, recovery of sweating occurs gradually on return to cooler climate	Treat heat rash and avoid further skin trauma by sunburn, periodic relief from sustained heat
5. Behavioral Disorders				
(a) Heat Fatigue--Transient				
Impaired performance of skilled sensorimotor, mental, or vigilance tasks, in heat	Performance decrement greater in unacclimatized and unskilled worker	Discomfort and physiologic strain	Not indicated unless accompanied by other heat illness	Acclimatization and training for work in the heat
(b) Heat Fatigue--Chronic				
Reduced performance capacity, lowering of self-imposed standards of social behavior (e.g., alcoholic overindulgence), inability to concentrate, etc.	Workers at risk come from temperate climates, for long residence in tropical latitudes	Psychosocial stresses probably as important as heat stress, may involve hormonal imbalance but no positive evidence	Medical treatment for serious cases, speedy relief of symptoms on returning home	Orientation on life in hot regions (customs, climate, living conditions, etc.)

Adapted from Reference 73.

1. Heatstroke

The classical description of heatstroke includes: (1) a major disruption of central nervous function (unconsciousness or convulsions); (2) a lack of sweating; and (3) a rectal temperature in excess of 41°C (105.8°F) [4,59,75,76]. The 41°C rectal temperature is an arbitrary value for hyperpyrexia, because the disorder has not been produced experimentally in humans so that observations are made only after the admission of patients to hospitals, which may vary in time from about 30 minutes to several hours after the event. In some heatstroke cases, sweating may be present [76]. The local circumstances of metabolic and environmental heat loads which give rise to the disorder are highly variable and are often difficult or impossible to reconstruct with accuracy. The period between the occurrence of the event and admission to a hospital may result in a quite different medical outcome from one patient to another depending on the knowledge, understanding, skill, and facilities available to those who render first aid in the intervening period. Recently, the sequence of biologic events in some fatal heatstroke cases have been described [77].

Heatstroke is a MEDICAL EMERGENCY, and any procedure from the moment of onset which will cool the patient improves the prognosis. Placing the patient in a shady area, removing outer clothing and wetting the skin, and increasing air movement to enhance evaporative cooling are all urgently needed until professional methods of cooling and assessment of the degree of the disorder are available. Frequently, by the time a patient is admitted to a hospital, the disorder has progressed to a multisystem lesion affecting virtually all tissues and organs [77]. In the typical clinical presentation, the central nervous system is disorganized, and there is commonly evidence of fragility of small blood vessels, possibly coupled with the loss of integrity of cellular membranes in many tissues. The blood-clotting mechanism is often severely disturbed, as are liver and kidney functions. It is not clear, however, whether these events are present at the onset of the disorder, or whether their development requires a combination of a given degree of elevated body temperature and a certain period for tissue or cellular damage to occur. Postmortem evaluation indicates there are few tissues which escape pathological involvement. Early recognition of the disorder or its impending onset, associated with appropriate treatment, considerably reduces the death rate and the extent of organ and tissue involvement. An ill worker should not be sent home or left unattended without a physician's specific order.

2. Heat Exhaustion

Heat exhaustion is a mild form of heat disorder which readily yields to prompt treatment. This disorder has been encountered frequently in experimental assessment of heat tolerance. Characteristically, it is sometimes but not always accompanied by a small increase in body temperature (38°-39°C or 100.4°-102.2°F). The symptoms of headache, nausea, vertigo, weakness, thirst, and giddiness are common to both heat exhaustion and the early stage of heatstroke. There is a wide interindividual variation in the ability to tolerate an increased body

temperature; some individuals cannot tolerate rectal temperatures of 38°-39°C, and others continue to perform well at even higher rectal temperatures [78].

There are, of course, many variants in the development of heat disorders. Failure to replace water may predispose the individual to one or more of the heat disorders and may complicate an already complex situation. Therefore, cases of hyperpyrexia can be precipitated by hypohydration. It is unlikely that there is only one cause of hyperpyrexia without some influence from another. Recent data suggest that cases of heat exhaustion can be expected to occur some 10 times more frequently than cases of heatstroke [59].

3. Heat Cramps

Heat cramps are not uncommon in individuals who work hard in the heat. They are attributable to a continued loss of salt in the sweat, accompanied by copious intake of water without appropriate replacement of salt. Other electrolytes such as Mg^{++}, Ca^{++}, and K^{+} may also be involved. Cramps often occur in the muscles principally used during work and can be readily alleviated by rest, the ingestion of water, and the correction of any body fluid electrolyte imbalance.

4. Heat Rashes

The most common heat rash is prickly heat (miliaria rubra), which appears as red papules, usually in areas where the clothing is restrictive, and gives rise to a prickling sensation, particularly as sweating increases. It occurs in skin that is persistently wetted by unevaporated sweat, apparently because the keratinous layers of the skin absorb water, swell, and mechanically obstruct the sweat ducts [21,79,80]. The papules may become infected unless they are treated.

Another skin disorder (miliaria crystallina) appears with the onset of sweating in skin previously injured at the surface, commonly in sunburned areas. The damage prevents the escape of sweat with the formation of small to large watery vesicles which rapidly subside once sweating stops, and the problem ceases to exist once the damaged skin is sloughed.

Miliaria profunda occurs when the blockage of sweat ducts is below the skin surface. This rash also occurs following sunburn injury, but has been reported to occur without clear evidence of previous skin injury. Discrete and pale elevations of the skin, resembling gooseflesh, are present.

In most cases, the rashes disappear when the individuals are returned to cool environments. It seems likely that none of the rashes occur (or if they do, certainly with greatly diminished frequency) when a substantial part of the day is spent in cool and/or dry areas so that the skin surface can dry.

Although these heat rashes are not dangerous in themselves, each of them carries the possibility of resulting patchy areas which are anhidrotic, and thereby adversely affects evaporative heat loss and thermoregulation. In experimentally induced miliaria rubra, sweating capacity recovers within 3-4 weeks [79,80]. Wet and/or damaged skin could absorb toxic chemicals more readily than dry unbroken skin.

C. Chronic Heat Disorders

Some long term effects from exposure to heat stress (based on anecdotal, historical, and some epidemiologic and experimental evidence) have been suggested. Recently, the evidence was reviewed by Dukes-Dobos who proposed a three-category classification of possible heat-related chronic health effects [77]. The three categories are Type I - those related to acute heat illnesses such as reduced heat tolerance following heatstroke or reduced sweating capacity; Type II - not clear clinical entities, but are similar to general stress reactions; and Type III - which includes anhidrotic heat exhaustion, tropical neurosthenia, and increased incidence of kidney stones. The primary references cited in the review are suggestive of some possible chronic heat effects. However, the available data do not contribute information of value in protecting workers from heat effects. Nevertheless, the concept of chronic health effects from heat exposure may merit further formal laboratory and hot industry investigations.