CHAPTER 30

PHYSIOLOGY OF HEAT STRESS

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INTRODUCTION

Industrial heat exposure often exceeds that encountered in the hottest natural climate. Because hot industrial jobs usually require heavy work, the added burden of metabolic heat production may exceed the worker's physiologic capacity to regulate his body temperature, leading to impaired performance or clinical signs of heat illness. The physiologist's aim is to determine the duration and intensity of work (internal heat load) in combination with heat exposure (external heat load), which can be tolerated without excessive heat strain on thermoregulatory systems. By reason of physical fitness, work capacity, age, health status, living habits, and level of acclimatization, men vary in ability to tolerate heat stress. The purpose of this chapter will be to discuss a) homeostatic control of body temperature by balancing heat loss and heat gain, b) physiological indices of heat strain, c) acclimatization and other factors affecting heat tolerance, and d) clinical illnesses resulting when adaptations fail.

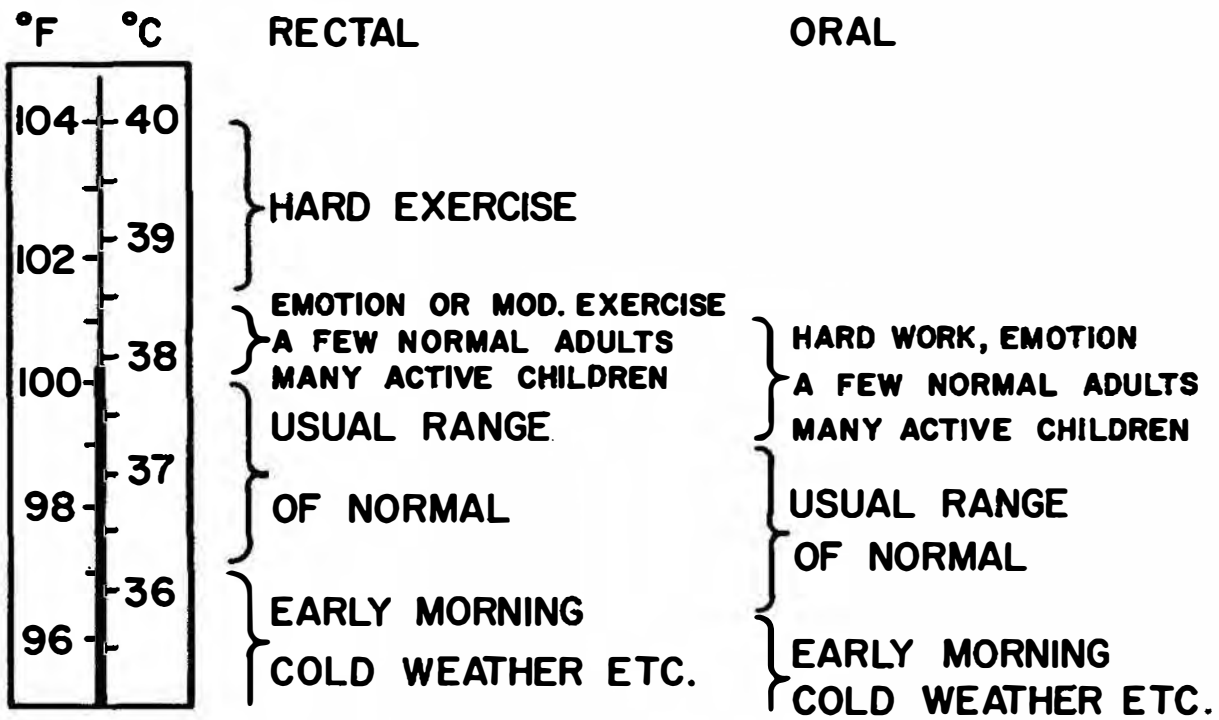
BODY TEMPERATURE REGULATION

Thermal Homeostasis

Man, and other homeotherms, regulate internal body temperature within narrow limits by physiologic control of blood flow from sites of heat production in muscles and deep tissues to the cooler body surface where heat is dissipated through physical channels of radiation, convection and evaporation to the environment. When heat loss is in balance with heat production, internal temperature is maintained at the regulated level. Homeostasis thus maintains a favorable and uniform internal temperature despite fluctuations in the thermal environment.

Normal Range

Figure 30-1[1] indicates the usual range in body temperature (rectal, oral) in normal persons as well as extreme upper and lower limits of normal. There are other sites in the lower esophagus and



DuBois, E. F.: Fever and the Regulation of Body Temperature. Springfield, Illinois, Charles C. Thomas, 1948.

Figure 30-1. Range of Normal Rectal and Oral Temperatures [from DuBois (1)]

in the ear canal for measuring internal temperature which reflect more promptly the responses to transient heating or cooling of the body. For present purposes central or "core" temperature, wherever measured, will be designated T_c.

Body Shell

This term refers to the cooler superficial tissues (skin, subcutaneous tissues, extremities) surrounding the warm core. Temperature of the shell tissues, particularly the distal extremities, varies more widely than the core under ambient heat and cold, as reflected in changes of mean temperature of the skin surface, $\overline{T}_s$, which is a weighted average taken at up to ten skin sites. The shell acts as a thermal buffer between the core and thermal environment. Under comfortable ambient conditions T_c is 37° and $\overline{T}_s$ is normally 33-34°C, but may approach to within a degree or two of T_c under heat stress and decline to as much as 10-15°C below T_c in the cold.

These changes in core to surface gradient are accompanied by alterations in rate of blood flowing from the warm core to the cool surface to meet changing needs in heat conductance, which is defined as units of heat transferred through the skin per unit time to the environment per degree of temperature gradient. Under heat stress $\overline{T}_s$ rises, and core to skin gradient narrows. 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 circulation, for which conductance is a useful index.

Heat Production

Energy required to sustain all body functions at rest and during work is derived by enzymatically controlled oxidative combustion of fuel substrates (carbohydrate, fat, protein), with CO_2, water and nitrogen wastes forming end products. These reactions are exothermic, the heat produced in the body being essentially equal to that measured when the same quantities of food substrates are oxidized at high temperature outside the body.

This is the basis of *indirect calorimetry* in which heat produced metabolically can be measured by the rate of oxygen uptake during rest or activity, one liter of O_2 being closely equivalent to a heat output of 5 kcal. Laboratory or field measurements of metabolic heat production are relatively simple, involving a system for measuring the volume of air breathed each minute and an instrument for measuring the difference in O_2 concentration between inspired and expired air.

Resting O_2 uptake in an average man (70 kg body weight; 1.8m² surface area) is about 0.3 1/min, equivalent to heat production at a rate of 1.5 kcal/min or 90 kcal/hr. In terms of surface area this is 50 kcal/m² hr, a unit referred to as *1 met*, the metabolic rate of a man sitting at rest in a comfortable environment.

There is little individual variation in resting metabolism when expressed in heat production per unit area. In terms of maximum capacity to perform work, however, there are wide differences, depending mainly on body size, muscular development, physical fitness and age. An important measure of work capacity is the maximum rate at which a man can take up oxygen during brief strenuous work effort. Maximum oxygen uptake (VO_2 max) among healthy workers ranges between about 2.0 and 4.0 1/min.

Table 30-1[2] lists examples of work activity, the O_2 requirement, the heat equivalent, and the

TABLE 30-1
Oxygen Uptake, Body Heat Production, and Relative Energy Cost of Work in 70 kg Men

Activity	Oxygen* Uptake (1/min)	Body Heat** Production (M) (kcal/hr)	Maximum Oxygen Uptake (1/min)		
			Low 2.5	Medium 3.0	High 3.5
			Percent VO_2 max Required		
Rest (seated)	0.3	90	12	10	8.5
Light Machine Work	0.66	200	26	22	19
Walking (3.5 mph on level)	1.0	300	40	33	28
Forging	1.3	390	52	43	37
Shoveling (depends on rate, load and lift)	1.5-2.0	450-600	60-80	50-66	43-58
Slag Removal	2.3	700	92	77	66

*From Passmore and Durnin (See Figure 30-2).

**In lifting, pushing, or carrying loads, cranking, etc. the heat equivalent of the external work (W) is subtracted from the total energy output (O_2 uptake) to obtain heat produced in the body (M). Net efficiency of W, i.e., $\frac{W}{O_2 \text{ uptake (work)} - O_2 \text{ uptake (rest)}}$ is 20% or less for most work. Skill acquired by practice increases efficiency of work performance and thus reduces heat load of M.

TABLE 30-2
Heart Rate, Core Temperature, and Endurance Time Corresponding to Relative Energy Cost of Work

		Percent VO_2 max				
	At Rest	25	33⅓	50	75	100
Heart Rate (min)	60-80	90-100	105-110	120-130	150-160	180-190
Core Temperature (°C) at Equilibrium	37	37.4	37.8	38.2	38.8 (Unstable)	Continuous Rise
Endurance Time for Continuous Work	—	>8 hr	8 hr	1 hr	15-20 min	4-6 min

percent of VO_2 max required for this work in men of low (2.5 1/min), intermediate (3.0 1/min) and high (3.5 1/min) work capacity.

A comparative index for estimating maximum work capacity among men is the heart rate (HR) attained during steady work at less than the maximal effort (Table 30-2) which also indicates that the rise in T_c is proportional to % VO_2 max.

At work rates at 50% VO_2 max and above the oxygen supply to the muscle fails to meet the 0_2 demand, thus limiting endurance time. Successive increments in energy requirement for work are supported by progressively greater proportions of the energy being supplied by anaerobic (i.e.,

TABLE 30-3
Symbols and Their Meaning for Physical Factors in the Thermal Environment and Physiological Factors in Heat Exchange

Physical Factors		Physiological Factors	
Symbol	Meaning	Symbol	Meaning
T_a	*Air temperature using dry bulb thermometer.*	$\bar{T}_s$	*Mean skin temperature.*
$\bar{T}_r$	*Mean temperature of surrounding surfaces* (wall temperature). In presence of radiant heat, $T_r > T_a$.	T_c	*"Core" or central temperature* (measured in the rectum, esophagus, or near the tympanic membrane.
V	*Air velocity (fpm or m/s).*	P_{ws}	*Water vapor pressure of wetted skin at skin temperature.*
T_g	*Temperature of the 6" black globe.* T_g exceeds T_a when $T_r > T_a$. Elevation of T_g in equilibrium with radiant heat varies inversely with convective cooling by V. With appropriate coefficients T_g represents R + C.	A	*Total surface area of the body* (m^2).
		s	*Area of wetted surface.* $\frac{s}{A} \times 100 =$ % of wetted body surface.
P_{wa}	*Water vapor pressure of ambient air.*	M	*Metabolic rate of body heat production* (*kcal/hr*).
T_{wb}	*Temperature of the wet bulb thermometer.* Evaporative cooling under forced convection depresses reading of T_{wb} below T_a, the degree varying inversely with P_{wa}. In air fully saturated with water vapor (100% RH) $T_{wb} = T_a$.	*met*	*Unit of M per m^2/hr.* Resting M = 1 *met* or 50 kcal/m^2 hr
		VO_2 max	*Maximum oxygen uptake.* Also called maximum aerobic work capacity.
T_{ET}	*Effective Temperature Scale.* An empirical index combining T_a (or T_g), T_{wb}, and V into a single value based on sensory effect.*	SR	*Sweat rate (kg/hr).*
		E	*Body heat loss by evaporation (kcal/hr).*
		BF_s	*Blood flow to the skin (1/m^2 min).*
$°C_{ET}$	*Effective Temperature in degrees Centigrade.*	C	*Conductance* = $\frac{M/A}{T_c - T_s}$ [kcal/m^2 hr per degree of gradient]

*ET Scales in the form of nomograms (Basic Scale for men stripped to the waist and Normal Scale for men lightly clothed) were derived from tests on men moving between two climate chambers, a test chamber with T_a, T_{wb}, and V fixed in various combinations, and a reference chamber with still air fully saturated held at temperatures ranging in different tests from 0 to 43°C. All combinations of T_a, T_{wb}, and V producing immediate thermal sensations which were equivalent to those experienced in the reference chamber were assigned the same Effective Temperature, namely that of saturated still air at that temperature.

without O_2) splitting of muscle glycogen, the carbohydrate energy storehouse, into lactic acid, which accumulates in the muscle, impairs contraction and results in fatigue. During the rest period, the "oxygen debt" incurred during work is paid off, as indicated by O_2 uptake remaining elevated and declining exponentially to the resting level as the accumulated lactic acid is oxidized or resynthesized into glycogen. Under heat stress, the recovery period is longer to eliminate heat stored in the body during work.

Heat Loss

Under comfortable ambient conditions 25 percent of heat produced by metabolism (M) at rest is transferred from the skin surface to the cooler air by convection (C), 50 percent by radiative transfer to cooler surfaces in the surroundings (R), and the remaining 25 percent by warming inspired air, and by evaporation of 20 to 30 g/hr of moisture diffusing through the non-sweating skin. Respiratory heat loss (8-10% of resting M) plays little role in temperature regulation and only heat loss through the skin will be considered here.

Symbols and their meanings to designate the environmental and physiological variables used in this chapter are listed in Table 30-3.

The foregoing sections may be summarized in the heat balance equation as expressed for temperature equilibrium below and in Table 30-4.

$$M \pm R \pm C - E = O,$$

in which R and C are rates of radiative and convective heat transfer. M and E are defined in Table 30-3. Table 30-4 indicates how the equation applies under three different conditions of the temperature and vapor pressure gradient between skin and environment. It should be noted that when $\bar{T}_s < T_g$ and P_{ws} approaches or equals P_{ws}, equilibrium is not possible either at rest or during work.

TABLE 30-4
Heat Balance under Different External Temperature Gradients and Factors Limiting Endurance Time for Work

External Gradient	Example	Heat Balance	Endurance Time Limited by:	Representative Environments
$\bar{T}_s > T_g$ $P_{ws} > > P_{wa}$	$T_g = 25°C$	$M = R + C + E$	Work Rate	Temperate climate. Also thermally neutral work places.
$\bar{T}_s = T_g$ $P_{ws} > P_{wa}$	$T_g = 35°C$	$M = E$	Work rate and elevated P_{wa} and/or low V (Restricted evaporation)	Tropical climate. Also canning, textiles, laundries, deep metal mines.
$\bar{T}_s < T_g$ $P_{ws} > > P_{wa}$	$T_g = 45°C$	$M + R + C = E$	Work rate and maximum capacity to sweat (Free evaporation)	Hot desert climate. Also manufacturing of primary metals, glass, chemicals, etc.

Hypothalamic Regulation of Body Temperature

There is convincing evidence based on animal experiments that the temperature regulating center in man lies in a region at the base of the brain called the hypothalamus. The anterior portion contains the "heat loss" center which responds to increases in its own temperature, as well as to incoming (afferent) nerve impulses from warm receptors in the skin. It activates heat loss through increased blood flow to the skin and sweating (man) or panting (other mammals).

A model of the thermoregulatory system for control of body temperature under heat stress is represented in Figure 30-2* as an analog of an engineering control system known as a proportional controller using negative feedback. Feedback is negative because the error signal is the *difference* between the set point of the thermostat (input) and T_c and/or T_s (output). It is a *proportional* controller because the central drive and effector responses (BFs and SR) are proportional to the error signal. In the absence of a heat load, central drive is zero, output and input being equal. The model predicts that when equilibrium is reached under a given heat load, the output of the system (T_c, $\bar{T}_s$) will stabilize at a level above the set point by an amount also proportional to the load. This deviation from the set point is known as the "load error." In the presence of a load, a proportional controller does not restore the error signal to zero. These characteristics of the model are also seen in thermoregulatory control under heat stress in man. Finally, effectiveness of the controller in temperature regulation depends on its *gain*, or sensitivity to an error signal. The gain factor is high in individuals with high heat tolerance, and increases in acclimatization.

Subjective and Behavioral Responses to Heat Stress

Subjective sensations of heat, perceived as neutral, warm, or hot, depend primarily on skin temperature. *Heat discomfort*, however, is the subjective evaluation of the thermal environment in terms of unpleasantness and depends not only on sensations of heat but also on the level of physiological strain (SR, BF_s, T_c). Thermal comfort scales (e.g., Effective Temperature Scale) define limits of ambient temperatures, activity levels, and clothing under which heat balance can be maintained without thermal strain.

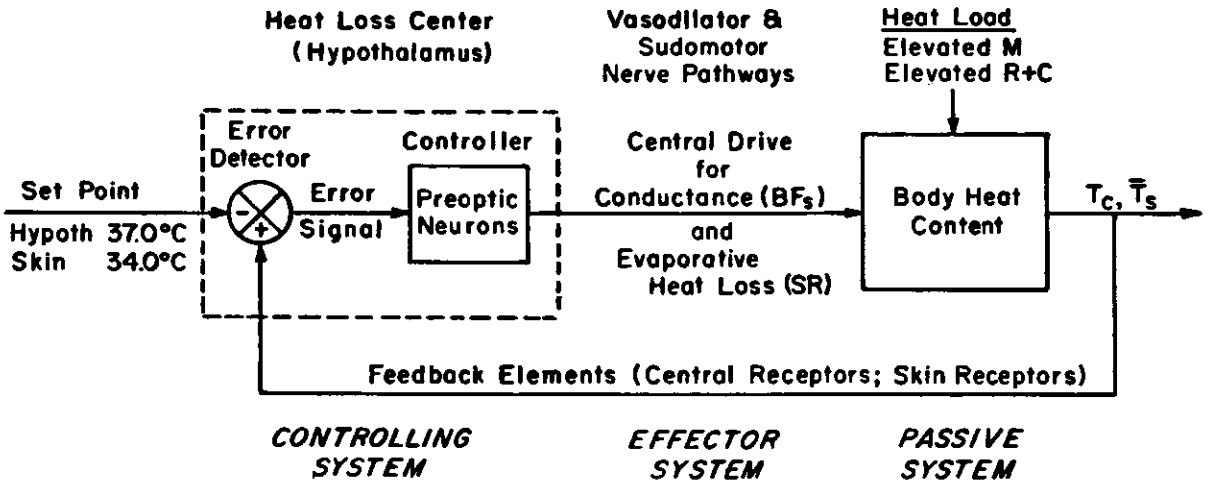


Figure 30-2. Model of the System for Thermoregulatory Control of Body Temperature.

*The model shown in Figure 30-2 is adapted from recent studies (Nadel *et al.*[3]) indicating that the central drive for sweating is the summation of effects from the elevation of T_c and $\bar{T}_s$ above their corresponding set points (37° and 34°C). The input from elevated T_c, however, is 10x greater in effect than that from elevated T_s. Moreover, local heating or cooling of the skin augments or suppresses local sweating from a temperature effect on the neuroglandular junction with a Q_{10} of 2.7 to 3.0. The summation model with the multiplicative factor for local skin temperature, T_{s_l}, is given in the form

$$SR = [\alpha (T_c - 37) + \beta (\bar{T}_s - 34)]\, e^{(T_{sl} - 34)/10}$$

where $\frac{\alpha}{\beta}$ is about 10, and e is the base of natural logarithms.

These authors point out that SR is also inversely related to skin wetness and directly to degree of acclimatization.

Immediate sensations of heat or the subsequent discomfort from strain may lead to behavioral responses such as slowing or stopping work (reduced M), modifying clothing, or withdrawing to a cooler environment (reduced R+C). Such adaptive behavior, based on instinct or experience, serves as man's first line of defense against severe or incapacitating strain.

INDICES OF HEAT STRAIN

Sweat Rate

Under heat loads resulting in an error signal, effector outflow from the hypothalamus is transmitted via nerves to sweat glands of the skin which are activated by release of acetylcholine at the neuroglandular junction. Rate of secretion of individual glands, and the number of active glands recruited determines the total sweat rate. Under maximum central drive, the estimated 2½ million eccrine glands can secrete sweat at peak rates of more than 3 kg/hr for up to an hour in highly acclimatized men, and can maintain rates of 1 to 1.5 kg/hr for several hours.

When sweat can evaporate freely from the skin (i.e., SR=E), evaporative cooling is regulated under steady state conditions of work and heat exposure to balance the heat load (M+R+C) up to the maximum rate of sweating (1 kg/hr). SR follows $\bar{T}_s$ (Figure 30-3) which varies linearly with ambient temperature. Over a wide range of ambient temperatures from cool to moderately hot, Nielsen[4] found that T_c is constant under steady state conditions of work, the elevation of T_c above 37°C depending solely on M. On the other hand, under constant ambient conditions SR varies with M, to which the elevation of T_c is proportional. The central drive for sweating is thus determined by work rate, M, but the actual sweat output is modulated by skin temperature to meet evaporative requirements under conditions from cool to hot up to the limits for sweating capacity.

Sweat Evaporation

The evaporation of 1 g of sweat from the skin eliminates 0.58 kcal of body heat. Efficiency of body cooling by sweat, however, depends on the rate of evaporation, which is determined by the gradient between vapor pressure of wetted skin (P_{ws}) and ambient air (P_{wa}) multiplied by a root function of effective air velocity at the skin surface ($V^{0.6}$) and s, the fraction of body surface, A, that is wetted.

When evaporation of sweat is restricted, $\bar{T}_s$ rises above that observed under less humid conditions at the same T_a. The heat loss center responds by recruiting more sweat glands, thus increasing the extent of wetted body surface, s.*

If cooling needed to balance M+R+C under these conditions is thereby met, core temperature remains essentially unchanged. At higher levels

*As a fraction of the total area of body surface, s cannot be measured directly. It is estimated from the ratio of the rate of evaporation required to balance M+R+C (E_{req}) to the maximum rate at which sweat evaporation can occur (E_{max}) at a given $\bar{T}_s$, P_{wa}, and air velocity.

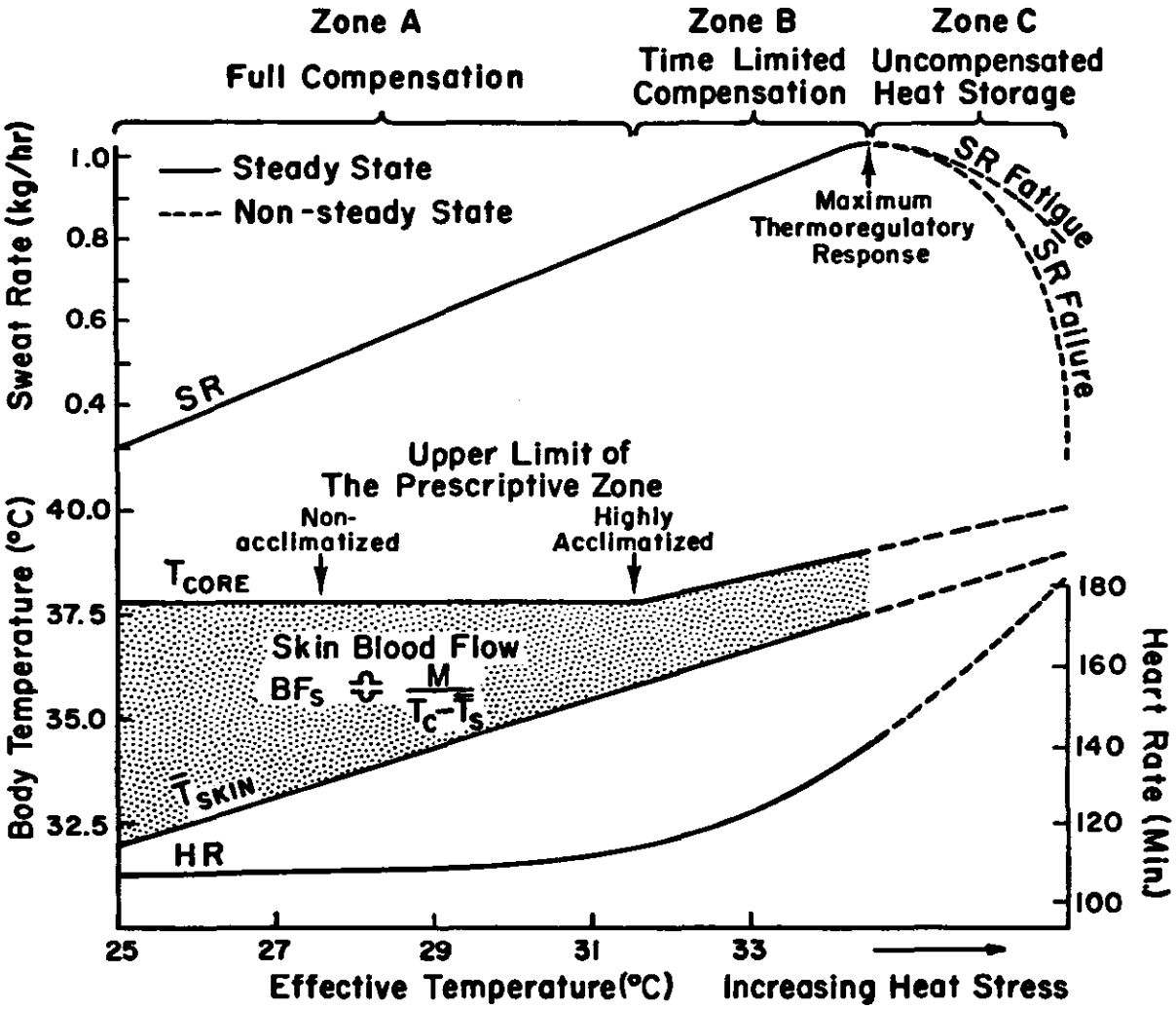


Figure 30-3. Thermoregulatory Responses to Heat Stress in Zone A (Full Compensation), B (Time Limited Compensation) and C (Uncompensated Heat Storage). Graph illustrates the effector responses (SR, BF_s), circulatory strain (HR) and the controlled variables (T_c, T_s) in a highly acclimatized man working at one-third VO_2 max (M = 300 kcal/hr) at levels of heat stress up to his limits of tolerance. Responses under steady state conditions are linear with Effective Temperature in Zones A and B. In Zone C, the steady state is impossible. Dashed lines indicate continuous heat storage and show trends only of T_c, T_s, SR and HR with increasing heat stress. (Semi-schematic representation based on data from refs. 7, 8, 9, 10, 11, 12, and 13.)

of P_{w_s} or lower air velocities, s approaches A (the total body surface area) and at the point when s = A, the body surface is 100% wetted. Any further increase in sweat production does not contribute to cooling, but drips off the body and is wasted. Under higher levels of P_{w_s} with further restriction on E, body heat will be stored, raising both $\overline{T}_s$ and T_c. The response is a greater central drive for sweating. But as $\overline{T}_s$ rises, P_{w_s} and evaporative rate increase also. As a result, a new steady state may be established but at a cost of increased thermoregulatory strain, as reflected in further elevation of SR, BF_s and HR.

As seen above, sweat rate in the zone of free evaporation varies linearly with heat load, and SR is proportional to M + R + C. In the zone of restricted evaporation when s/A approaches 1.0, SR > E and is proportional to the increase in T_c and $\overline{T}_s$.

Sweat rate is, therefore, an index of heat stress over the entire range of compensation. It is also an index of heat strain in the zone of time-limited compensation, where its rise parallels T_c and HR.

Sweat rate serves as a time-weighted average of heat stress. It is measured by the difference in body weight over a given time period corrected for weight gain by water and food intake and weight loss by urine and feces.

A constraint on the use of SR as an index of heat stress (or strain) is that the level of sweating tends to decline with time of heat exposure, particularly under restricted evaporation when skin is extensively wetted.

As long as SR > E, the decline in sweat rate does not interfere with heat loss, and might be regarded as an adaptive mechanism to conserve body water and electrolytes under conditions in which more sweat is produced than is useful.

Circulatory Strain

Thermal conductance (C), referred to earlier as an index of BF_s, is defined as

$$C = \frac{M/A}{T_c - \bar{T}_s} \text{ [Kcal/m}^2\text{-hr per degree of gradient]}$$

The narrower the gradient for a given M, or the higher the M for a given gradient, the greater is the BF_s required to transfer metabolic heat from core to the environment (Figure 30-3).

In a thermally neutral environment, $\bar{T}_s$ is lower during work than at rest, reflecting a redistribution of blood from skin blood vessels to those of active muscles. The reduced capacity and increased resistance of skin vessels and also of vessels in abdominal organs together with the pumping action of muscles maintain the return of venous blood to the heart whose output increases in proportion to the % VO_2 max required by work.

Under external heat loads both the central drive for increased conductance and the rise in local skin temperature dilate skin vessels, thereby increasing their blood capacity and reducing their resistance. BF_s increases but at the cost of reducing venous return of blood to the heart, resulting in less output per beat. To meet the oxygen requirements of working muscles, cardiac output can then be maintained only by further vasoconstriction in abdominal organs and an increase in heart rate (Figure 30-3).

Thermoregulatory requirements for BF_s thus compete for available cardiac output with energy requirements of active muscles. BF_s, as estimated by C, increases from a quarter of a liter per minute at rest in a neutral environment to over two liters per minute in men working at 3-4 *met* under heat stress.

Heart Rate as An Index of Circulatory Strain

Heart rate is responsive both to the increased cardiac output required by working muscles as well as the added circulatory strain imposed by heat exposure, and is a useful index of total heat load.

Measured either by counting the pulse at the wrist or by using electronic devices for monitoring, HR is a valuable guide in assessing hazards to health of workers exposed to heat stress. Brouha[5] has used the term *cardiac cost of work* to indicate the total heart beats above the resting level during work, and the term *cardiac cost of recovery,* or "cardiac debt," to denote the total number of beats above the resting level during the recovery period following work.

The detrimental effect of heat stress on work performance is indicated by an increase in cardiac cost both of work and recovery. Reducing work load, increasing time of recovery or providing cool rest areas are alternative measures which management may elect to prevent excessive heat strain on workers.

To ensure that men performing intermittent work in the heat will remain in thermal balance for the full shift without cumulative effects of strain, Brouha proposed a simple guide. *Pulse rate is counted for the last 30 seconds of the first three minutes after rest begins. If the first recovery pulse, i.e., from 30 to 60 seconds, is maintained at 110/min or below and deceleration between the first and third minute is at least 10 beats/min, no increasing strain occurs as the work day progresses.* Extensive testing to validate this guide in the management of health problems of industrial heat stress seems highly warranted.

The mean HR level observed during an entire work shift reflects sustained elevations and peak rates as well as recovery and resting rates and thus can also serve as a guide in assessing circulatory strain. Electronic devices for integrating total count or continuously recording individual heart beats are now generally available. Both methods were employed in a recent study of heart rate responses in steelworkers in a Pittsburgh steel mill.[6] There was variance in the mean HR in five workers on the same shift depending on work capacity (VO_2 max) of the individual and in the same worker on different shifts depending on total heat load. HR in all workers ranged from 99 to 136/min. The two workers with mean HR's exceeding 120/min showed evidence of excessive strain as indicated by a high resting HR and impaired work performance during a standard exercise after the shift.

The heart rate of men working at one-third VO_2 max will be 105-110/min (Table 30-2). On the basis that the combined effects of heat stress and work should not impose a greater circulatory demand than from work alone, an upper value of 110/min as the mean HR for an 8-hour shift would appear to be a reasonable limit for work involving heat exposure. For men or women of lower work capacity (i.e., VO_2 max less than 3 1/min) the energy expenditure at this heart rate would be less, but the strain proportionally the same.

In order to adopt the limit of 110/min. expressed in Table 30-2 as a standard, extensive testing and validation in industry would be required. The aim would be to compare circulatory strain in workers performing jobs in which heat exposure and work rates vary in proportion, and in workers on jobs involving peak loads with those exposed to more uniform work stresses. Finally, it would be necessary to determine whether the level of 110/min is safe or should be lowered for male or female workers whose work capacity and heat tolerance is limited because of age, physical fitness, acclimatization, or general health status.

Core Temperature

In Zone A (Figure 30-3) SR and BF_s increase proportionally with the total heat load (M+R+C). T_c is maintained at a uniform level which is determined only by M, and is independent of

ambient temperatures at lower levels of external heat stress.[4] Lind[7] terms this the *prescriptive zone* to indicate the range of thermal environments in which men can work without strain on homeostatic control of core temperature.

The upper limits of this zone are lower at high work rates because T_c is higher (Table 30-2). By the same token, the limit would be similar for men differing in physical fitness but expending the same percent of their VO_2 max (Table 30-1).

From data of Robinson[8] and others[9, 10, 11, 12] the upper limit of the prescriptive zone in highly acclimatized men working at 300 kcal/hr is 31-32°C_{ET}.

For non-acclimatized men varying in physical fitness Lind[13] recommends 27.5°C_{ET} (Figure 30-3) as a realistic limit for this level of work. The wide latitude of heat stress between these limits clearly indicates the perplexing nature of the problem of setting rational standards for industrial heat stress which will both protect workers of low heat tolerance and not restrict unduly work performance of those with higher heat tolerance.

In a man performing steady work at 300 kcal/hr, T_c is higher in Zone B than in the prescriptive zone in proportion to heat stress up to a limiting value of 39°C (Figure 30-3). This represents the highest core temperature at which highly acclimatized men can attain a steady state of thermal balance, and then for only two hours or less. The upward inflection of T_c in Zone B serves to maintain the core to surface gradient as $\overline{T}_s$ attains higher levels, but at a cost of thermoregulatory strain on T_c. In terms of the model (Figure 30-2), the load error of the control system as reflected in T_c increases in proportion to the load.

The maximum tolerable level of heat stress corresponding to the T_c limit of 39°C was found by Robinson and others to be 34 to 35°C_{ET}. Men less fit or less well-acclimatized for work at 300 kcal/hr would reach limiting levels for thermal balance at lower core temperatures and at correspondingly lower levels of external heat stress. As a practical guide, the average core temperature of men should not exceed 38°C for a work shift. Transient increases to 39°C should be permitted but only briefly, and with ample time for recovery in cooler areas.

The border between Zones B and C marks the upper limit of man's capacity to sweat, thus representing the maximum effector response of the thermoregulatory center. Hence, in Zone C, rates of heat loss fail to match rates of heat gain, leading to heat storage with T_c and $\overline{T}_s$, rising continuously at rates proportional to the heat load. Storage may be further accelerated by fatigue or failure of sweating. No steady state during continued work is possible. This is indicated by broken lines in Zone C which imply trends only and not the transient state. Under extreme heat (e.g., 40 to 45°C_{ET}) the core to surface gradient will be reversed, the blood returning from the skin heating the body core instead of cooling it. The rising body temperature accelerates metabolic processes (Q_{10} effect), further increasing the rate of body temperature rise. Unless the man stops working and seeks relief, heat exposure in Zone C leads inevitably to his collapsing from circulatory failure or heat stroke. Voluntary tolerance time for work in Zone C ranges from a maximum of one hour to less than 20 minutes.[14].

Under intense radiant heat loads, skin temperature rises rapidly to the pain threshold (45°C). Under these conditions pain becomes the limiting factor in tolerance time rather than heat storage in deeper tissues.

FACTORS IN HEAT TOLERANCE

Heat Acclimatization

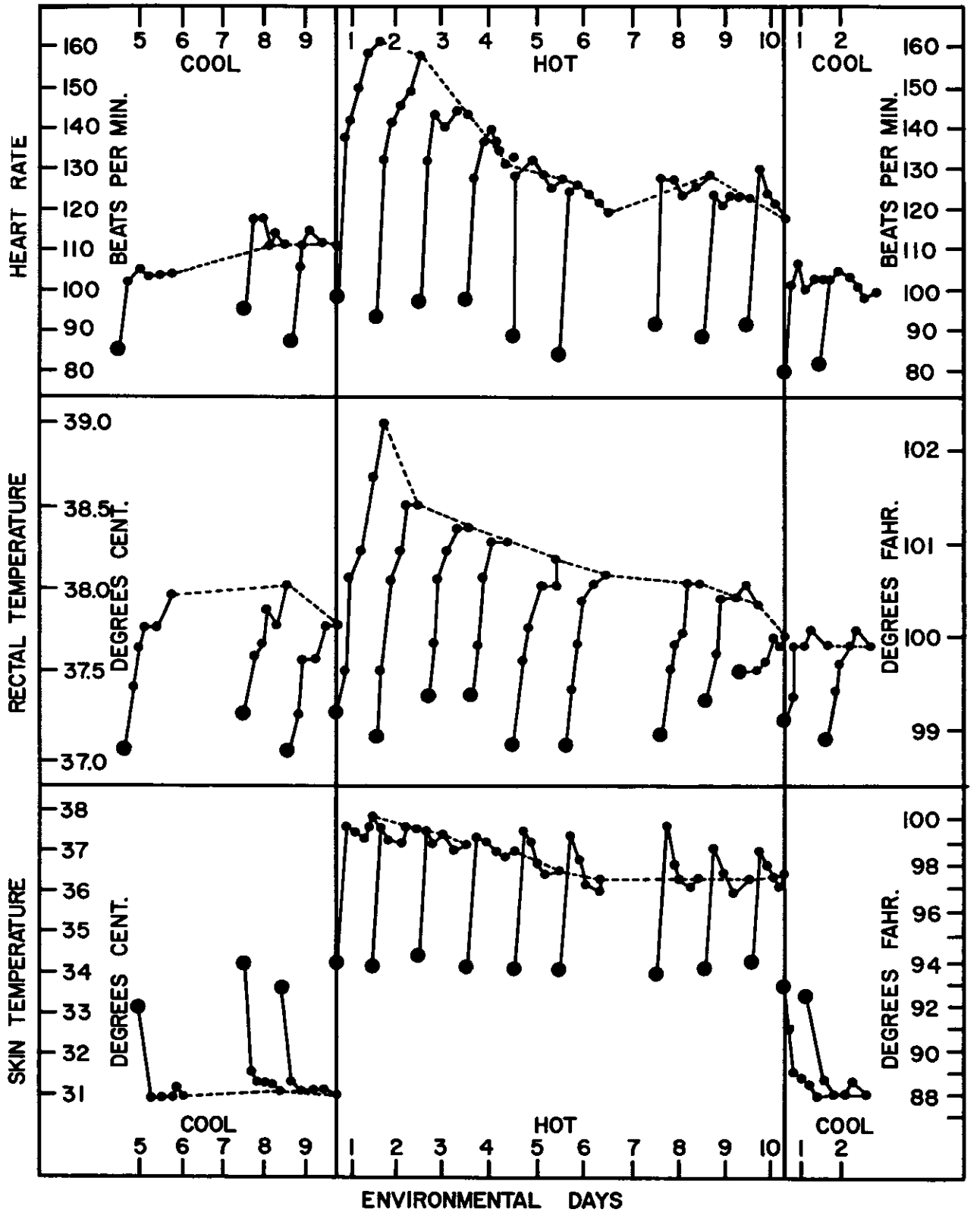
Any man, however healthy, well-conditioned and motivated, who works for the first time under heat stress will develop signs of severe strain with abnormally high body temperature, pounding heart, and other signs of heat intolerance. On each succeeding day of heat exposure his ability to work improves as signs of strain and discomfort diminish. After a week or two he can work without difficulty. The enhanced tolerance to heat acquired by working in a hot environment is called heat acclimatization.

Figure 30-4 from Eichna *et al.*[10] illustrates the principal physiological adjustments in thermal balance which occurred in three highly conditioned young men with no previous heat exposure working for one hour/day for ten days at 300 kcal/hr (3 *met)* in dry heat (T_a, 50.5°C; T_{wb}, 26.5°C; V, 450 fpm). T_c, $\overline{T}_s$, and HR at rest in a cool environment (large dots) and at the end of each 10 min period of work in the heat (small dots) are shown, with control tests at rest and working under cool conditions before and after ten days of work in the heat. Table 30-5 summarizes the authors' data, including measurements of sweat rates and BF_s calculated from conductance.

The most significant change was a 10% increase in sweat output which was produced *at a lower* $\overline{T}_s$ on hot day 10 compared with hot day 1. This increase in evaporative cooling with a steeper core to skin gradient was sufficient to compensate fully for the heat load, as indicated by the fact that core temperature was restored to within 0.1°C of that observed on cool day 1. In other words, conditions which had initially been nearly intolerable now fell within the "prescriptive zone" of full compensation, allowing the men to complete the work with no more difficulty than in the cool environment. Although BF_s remained elevated on hot day 10, acclimatization reduced the circulatory load by 32%.

Essential factors inducing acclimatization appear to be sustained elevations of T_c and $\overline{T}_s$, above levels for the same work in cool environments for an hour, or preferably more, per day for one or two weeks.

It seems well-established that acclimatization to wet heat increases tolerance to dry heat and vice versa. The reason why tolerance to wet heat is increased is not clear, because the increased sweat output, which may nearly double, is largely wasted. Heat conduction through the skin is enhanced,



Eichna, L. W., Park, C. R., Nelson, N., et al: Thermal regulations in a hot dry (dessert type) environment. Am. J. Physiol. 163:585, 1950.

Figure 30-4. Thermoregulatory Responses to Heat Stress in Zone A (Full Compensation), B (Time Limited Compensation), and C (Uncompensated Heat Storage).

TABLE 30-5
Changes in Thermoregulatory Responses with Acclimatization (Eichna *et al.*[10])

Condition	HR (min)	SR (kg/m^2hr)	T_c °C	$\bar{T}_s$ °C	$T_c - \bar{T}_s$	BF_s ($1/m^2 \cdot_{min}$)
Initial cool day	111	0.079	37.8	30.9	6.9	0.35
Hot day 1	162	0.621	39.0	37.8	1.2	2.58
Hot day 10	118	0.692	37.9	36.4	1.5	1.76
Final cool day	103	0.083	37.7	31.1	6.6	0.37

Reprinted from American Journal Physiology, 163:585, (1950).

however, which suggests a change in distribution of blood to the skin.[15]

Whether the underlying change in acclimatization is greater sensitivity of the center to thermal inputs from skin or central receptors, a lower threshold of skin receptors to heat, an enhanced response of the sweat glands to the central drive, or some combination of these factors is still the basic problem of thermal physiology which remains under active study.

Men who work at hot industrial tasks acquire levels of acclimatization commensurate with their average heat exposure. Unusual demands for work effort or sudden spells of hot weather may, however, overload their thermoregulatory capacity, leading to signs of overstrain. Heat acclimatization needs periodic reinforcement, such as occurs daily during the work week. Men may show some loss of acclimatization on the first day of the new shift after being idle for two days or over a weekend. After vacations of two weeks or longer, the loss of acclimatization is substantial, several days at work elapsing before heat tolerance is fully restored. Some traces of acclimatization may be evident, however, as long as eight weeks following the last heat exposure.

Seasonal changes in outside weather are reflected in heat tolerance of workers,[16] the lower level during cooler seasons owing largely to milder heat stress on the job, which parallels changes in outside weather temperature.

Physiologic adjustments in acclimatization also include changes in sweat composition. Sweat is a dilute solution of electrolytes, principally sodium chloride. In unacclimatized subjects, sodium chloride concentration in sweat (3 to 5 g/kg) is about half the concentration in blood plasma. In acclimatized subjects, sweat is not only more abundant but more dilute, the salt concentration falling to levels of 1 to 2 g/kg, reflecting an adaptive change in hormonal balance through secretion of aldosterone which acts to conserve body salt both by the kidneys and sweat glands.

Surface Area to Weight Ratio

In obese individuals as well as in those with stocky build the body surface area (A) to body weight (Wt) ratio is relatively low. Because heat loss is a function of A and heat production a function of Wt, a low A/Wt is a handicap for men performing sustained work in the heat. If lacking in acclimatization, physically unfit and obese men are at greater risk of succumbing to heat stroke.

Age and Degenerative Diseases

The healthy older worker (40-65 yrs) performs well on hot jobs if allowed to work at his own pace. Under demands for sustained work output in the heat, he is at a double disadvantage compared with younger men. First, VO_2 max declines 20-30% between ages 30 and 65, leaving the older worker with less cardiocirculatory reserve capacity and second, under levels of heat stress above the prescriptive zone, the older worker compensates for the heat loads less effectively than the younger man, as indicated by his higher core temperature and peripheral blood flow for the same work output.[17] This has been attributed to a delay in onset of sweating, and a lower sweat rate in older men, resulting in greater heat storage during work and longer time for recovery.

Degenerative diseases of the heart and blood vessels intensify the age effect on heat tolerance by limiting the circulatory capacity to transport heat from body core to surface. Elderly men and women with chronic diseases of aging account for much of the excess in mortality reported in large northern cities during sustained heat waves.[18]

Men with long work experience in hot industries, on the other hand, seem to be less at risk of dying from cardiovascular and other diseases than workers of similar age without a work history of heat exposure. In a recent unpublished biostatistical study of 12,946 open hearth steel workers,[19] the mortality rate for arteriosclerotic heart disease, and respiratory disease, as well as overall mortality rate were significantly less in this group than in the entire population of 58,829 steel workers. A self-selection process which eliminates those of low physical fitness and heat tolerance from jobs on the open hearth could not be ruled out.

Water Balance

Effective work performance in the heat depends on replenishing body water and salt lost in sweat. A fully acclimatized worker weighing 70 kg can secrete 6-8 kg of sweat per 8-hr shift. If water lost in sweat is not replaced by drinking, continued sweating ultimately draws on water both from tissue spaces and body cells as well, leading to the picture of shriveled skin, dry mouth and tongue, and sunken eyes recognized as extreme dehydration.

Sweat loss of 1 kg of body water (1.4% of body weight) can be tolerated without serious effect. Water deficits of 1.5 kg or more during work in the heat deplete the volume of circulating blood, resulting in signs and symptoms of in-

creasing heat strain (elevated HR and T_e; thirst and severe heat discomfort), resembling those seen in unacclimatized men. With water deficits of 2 to 4 kg (3 to 6% of body weight) work performance is impaired. Continued work leads to incipient signs of *heat exhaustion*. Therefore, to avoid excessive depletion of body water, sweating men should drink frequently at intervals of 30 min or less.

Unacclimatized men should be encouraged to drink somewhat more than thirst dictates to avoid "voluntary" dehydration. Well-acclimatized men succeed much better in balancing water losses, even when sweating at high rates. Dehydration of more than 1 to 2% of body weight in acclimatized workers during a shift may signify greater heat loads than usual, or lack of access to water.

Salt Balance

Salt (NaCl) losses in sweat during work can usually be replaced at mealtime. The average American diet, which contains 10 to 15 g/day of salt, would meet the needs of an acclimatized worker producing 6-8 kg of sweat containing 1 to 2 g of salt per kg during a single shift. For the period of acclimatization supplementary salt during hot work might be needed by workers with no previous heat exposure. Although maximal sweating rates in unacclimatized men are lower (4-6 kg/shift), salt concentrations are higher (3 to 5 g/kg sweat) than after acclimatization. At the higher sweat rate, an unacclimatized man may lose 18 to 30 grams of salt. Supplements in the form of 0.65-g salt tablets (preferably impregnated to avoid gastric irritation) may be taken if ample water is available. Better practice is to use salted water (0.1% or 1 tsp/gal) or to advise increased salt on food at mealtime.

Salt supplements should be reduced or discontinued after several days of heat exposure because salt loading suppresses normal hormonal mechanisms regulating salt and water metabolism under heat stress.

Depletion of body salt may occur in unacclimatized men exposed to heat who replace water losses without adequate salt intake in food. This leads to progressive dehydration because homeostatic controls are geared to maintain a balance between electrolyte concentration in tissue fluids with that in the cells. Deficient salt intake with continued intake of water tends to dilute tissue fluid, which suppresses the antidiuretic hormone (ADH) of the pituitary gland. The kidney then fails to reabsorb water and excretes dilute urine containing little salt.

Thus homeostasis maintains the electrolyte concentration of body fluids but at the cost of depleting body water with 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. Absence of chloride in the urine (< 3 g/l) is diagnostic of salt deficiency.

On a short-term basis, sweating men drinking large volumes of unsalted water may develop *heat cramps* which are excruciatingly painful spasms of those muscles used while working (arms, legs, or abdominal). Dilution of tissue fluid around the working muscle results in transfer of water into muscle fibers, causing the spasms.

Treatment of the various clinical syndromes of water and salt depletion is similar; namely, replacement of depleted body water and/or salt by oral ingestion of salted liquids in mild cases or intravenous infusion of saline in more serious ones. An excess of salt or water over actual needs is readily controlled by kidney excretion. These and other clinical entities resulting from failure to adapt to heat stress are described in Table 30-6, which is based on a nomenclature prepared jointly by committees representing the U.K. and the U.S.[20] For further details on etiology, signs, symptoms, treatment and prevention of heat illnesses, the reader is referred to Leithead and Lind.[14] (See Table 30-6).

Alcoholic Habits

Many authors have noted an excessive alcohol intake by patients within hours or a day or two prior to onset of *heat stroke*. Others have described striking reductions in workers' heat tolerance on the day following an alcoholic "binge." It is known that alcohol suppresses ADH, leading to loss of body water in urine. Hence dehydration may be a primary factor.

Physical Fitness

Physical conditioning alone does not confer heat acclimatization. The subjects of Eichna *et al.*, (Figure 30-4) were all highly conditioned before the test. Physical training without heat exposure, however, does improve heat tolerance, as indicated by somewhat lower heart rates and core temperatures in men exposed to heat after conditioning as compared with before. Sweat rates do not increase and skin temperature remains high. Physical conditioning enhances heat tolerance by increasing functional capacity of the cardiocirculatory system. Two important changes are first, an increase in number of capillary blood vessels to muscle, thus providing a larger interface between blood and muscle for exchange of oxygen and waste products and second, increased tone of small veins from tissues other than muscle so as to reduce their capacity during exercise, thus increasing pressure in large central veins returning the blood to the heart. Cardiac output per minute during work can increase with less need to accelerate the heart. These factors combine to increase VO_2 max of the physically conditioned man, giving him a wider margin of safety in coping with the added circulatory strain of work under heat stress. The extent to which men might gain in heat tolerance by acclimatization is not easily predictable, but those with a high level of physical fitness have the advantage.

Selection and Periodic Examination of Workers

Past performance in heat is perhaps the only reliable criterion on which to predict effectiveness of a worker's future performance under heat stress. For new employees without previous heat exposure, screening procedures should include standard tests of physical fitness and heat tolerance. Heart rates attained during a stepping exercise at 332

TABLE 30-6.
Classification, Medical Aspects, and Prevention of Heat Illness

Category	Clinical Features	Predisposing Factors	Underlying Physiological Disturbance	Treatment	Prevention
1. Temperature Regulation					
Heat Stroke and Heat Hyperpyrexia	Heat Stroke: 1) *Hot dry skin*: red, mottled or cyanotic. 2) *High and rising* T_c, 40.5°C and over. 3) *Brain disorders*: mental confusion, loss of consciousness, convulsions, coma as T_c continues to rise. Fatal if treatment delayed. Heat Hyperpyrexia: milder form. T_c lower; less severe brain disorders, some sweating.	1) Sustained exertion in heat by unacclimatized workers. 2) Lack of physical fitness and obesity. 3) Recent alcohol intake. 4) Dehydration. 5) Individual susceptibility. 6) Chronic cardiovascular disease in the elderly.	*Heat Stroke*: Failure of the central drive for sweating (cause unknown) leading to loss of evaporative cooling and an uncontrolled accelerating rise in T_c. *Heat Hyperpyrexia*: Partial rather than complete failure of sweating.	*Heat Stroke*: 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. *Heat Hyperpyrexia*: Less drastic cooling required if sweating still present and $T_c < 40.5$.	Medical screening of workers. Selection based on health and physical fitness. Acclimatization for 8-14 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. Recovery prompt and complete.	Acclimatization. Intermittent activity to assist venous return to heart.
3. Salt and/or Water Depletion					
a) *Heat Exhaustion*	1) Fatigue, nausea, headache, giddiness. 2) Skin clammy and moist. Complexion pale, muddy or hectic 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). *Water restriction type*: Urine volume small, highly concentrated. *Salt restriction type*: Urine less concentrated, chlorides less than 3 g/1.	1) Sustained exertion in heat. 2) Lack of acclimatization. 3) Failure to replace water and/or salt lost in sweat.	1) Dehydration from deficiency of water and/or salt intake. 2) Depletion of circulating blood volume. 3) Circulatory strain from competing demands for blood flow to skin and to active muscles.	Remove to cooler environment. Administer salted fluids by mouth or give I-V infusions of normal saline (.9%) if unconscious or vomiting. Keep at rest until urine volume and salt content indicate that salt and water balances have been restored.	Acclimatize workers using a breaking-in schedule for 1 or 2 weeks. Supplement dietary salt only 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 men, provide salted (0.1%) drinking water.
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.	Cooled sleeping quarters to allow skin to dry between heat exposures.
b) *Anhidrotic Heat Exhaustion* (miliaria profunda)	Extensive areas of skin which do not sweat on heat exposure, but present goose flesh 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. Rarely seen except in troops in wartime.	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 men.	Discomfort and physiological 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 homes in 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 abroad (customs, climate, living conditions, etc.)

Reprinted from "Heat Stress and Heat Disorders", Leithead, C. S., Lind, A. R., (1964) published by F. A. Davies Co., Philadelphia, Pa.

kg.m/min are now being used in selecting men for further acclimatization before assigning them to mining ore under high heat stress in the deep gold mines of South Africa.[21] Those of low work capacity, as indicated by a heart rate of over 140/min, are eliminated for these tasks. Those with lower exercise heart rates, particularly if below 120/min, are considered the best candidates and undergo graded acclimatizing exercises for 4 hours/day for 8 days in hot rooms ($T_{wb} = 31.7°C$). A second screening at this stage eliminates those with oral temperatures persistently above 38.3°C. Through screening, acclimatization, and selective placement based on heat tolerance, together with careful supervision of workers, serious heat casualties from heat stroke among the 100,000 or more men recruited yearly in this industry have been greatly reduced and productivity substantially increased.

Operations of this magnitude do not exist in the United States, but the same principles are applicable. On pre-employment examination, the physician can readily assess physical fitness using stepping exercises, or a bicycle ergometer to estimate VO_2 max. In terms of O_2 uptake per kg, a VO_2 max of 28 ml/kg min or less should be disqualifying. A standard test for heat tolerance is desirable but requires special facilities.

Older workers, including both new applicants and those undergoing periodic evaluation, should be examined with particular attention to chronic impairments of the heart, circulation, and vascular system but also of the kidneys, liver, endocrines, lungs and skin. Significant disease of any of these systems should be disqualifying for new employment on jobs involving severe heat exposure, or for those previously employed in such jobs if the disease is progressive despite treatment. Careful inquiry should be made on use of drugs, particularly hypotensive agents, diuretics, antispasmodics, sedatives, tranquilizers, and anti-depressants, as well as the abuse of drugs, particularly amphetamines and alcohol. Many of these drugs impair normal physiological responses to heat stress, and others alter behavior, exposing the patient or fellow workers to safety hazards. Toxic agents in the work environment which reduce heat tolerance, notably carbon monoxide, must also be considered. History of repeated accidents on the job, poor work performance, emotional instability, or frequent sick absence should alert the physician to possible heat intolerance of the employee.

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