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The Achievement of Thermal Balance and Its Maintenance During Environmental Stress

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THE ACHIEVEMENT OF THERMAL BALANCE AND ITS MAINTENANCE DURING ENVIRONMENTAL STRESS

P. F. Iampietro
Thomas Adams

Approved by



J. ROBERT DILLE, M.D.
CHIEF, CIVIL AEROMEDICAL
INSTITUTE

Released by



P. V. SIEGEL, M.D.
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FOREWORD

Aviation activities are conducted in a wide variety of environments, and knowledge concerning the effects of these environments on man is necessary in order to insure adequate performance. Environmental temperature is one factor that may profoundly alter performance or personal well-being or both. This paper describes in some detail the means, both physical and physiological, available to man for maintenance of thermal balance. It is also concerned with some aspects of thermal balance and performance when work is performed in hot or cold environments and the enhancement of performance that thermal acclimatization imparts.

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THE ACHIEVEMENT OF THERMAL BALANCE AND ITS MAINTENANCE DURING ENVIRONMENTAL STRESS

I. The Achievement of Thermal Balance.

The discussion that follows cannot aspire to be a complete analysis of the environmental or physiological factors involved in temperature regulation; it attempts only to be an introduction in this field and to offer a critical appraisal of selected interests. If it can afford the beginning student an understanding and appreciation of the general area, and give the advance student and professional reader an outline of current concepts, it will have fulfilled its responsibilities. More detailed information and extended bibliographies are available in several reviews.^{3, 4, 20, 30, 35, 40, 69, 74}

A. *The Homeotherm and His Environment.* In defending its acquired thermal balance, the homeotherm is presented with a dual thermostatic responsibility. Whereas it must appropriately adjust its own physiology to meet environmental thermal demands (during heat and cold exposures), it must also be prepared to rebalance biothermal heat-exchange mechanisms to dissipate metabolic heat loads during exercise, regardless of the environmental thermal imposition. Both internal and external thermal threats generally marshal the same physiological defenses against hyperthermia, as discussed below.

Metabolic processes, like the rates of most chemical reactions, are catalyzed by heat. Metabolic independence from the influence of environmental temperature, within the limits of successfully achieved homeostasis, gives the warm-blooded animal a considerable advantage over the poikilotherm. Thermally affected metabolic processes depend upon the regulated, relatively constant internal temperature for the homeotherm, but for the cold-blooded animal upon the more capricious and extreme environmental thermal changes. This freedom from other than extreme environmental thermal exposure has been identified as having had evolutionary importance.^{15, 20} The observation that all homeotherms thermoregulate within a relatively nar-

row temperature range (36°C for the elephant to 41°C for the birds) supports the suggestion that at least one other homeothermic advantage is internal thermal stability at a metabolically efficient level; ca. 37°C has been suggested to be an optimum temperature for many metabolic reactions.²⁰

Although a reasonably sharp distinction can be made between homeotherms and poikilotherms on the basis of body-temperature stability through physiological, reflexly mediated action, it has been demonstrated that poikilotherms can limit internal-temperature fluctuations through behavioral adjustments. It is the degree of precision and the type of body temperature control that separates the so-called cold- and warm-blooded animals. Whereas most homeotherms can control mean body temperature within a fraction of a degree by reflex adjustments of metabolism, blood flow, etc., poikilotherms are restricted to providing a less discrete body temperature set only by behavior and selection within the environment.

The homeotherm's ability to isolate metabolic processes from the influence of the environment does not come without cost. Total energy dissipation is usually greater than for the poikilotherm even at moderate exposure temperatures due to the steeper gradient between the homeotherm's skin-surface temperature and environmental temperature. In winter, the warm-blooded animal is faced not only with a greater heat loss due to lower ambient and environmental temperatures but also with reduced metabolic energy sources (food supplies). Besides improving, extending, and supplementing its thermal protection by additional fur growth and cold acclimatization processes, the warm-blooded animal so stressed may augment these protections by physically removing itself from the stressing environment (burrowing, migrating, etc.), or surrender its homeothermic status and retreat into a temporary state of modified poikilothermy

(hibernation or pseudo-hibernation). In either event, the obligated high-energy flux is avoided. Humans adapt further by using the "psychosociological"²⁰ tools of shelter, clothing, heating and air-conditioning systems, etc., and thereby establish for themselves greater thermal freedom than any other warm-blooded animal.

B. The Physical Basis for Homeostasis. A constant temperature for any object is achieved (equation 1, Figure 1) when sources of heat gain are equally matched by avenues of heat loss, with such a balance represented by an unchanging heat content ($\Delta H=0$). For animals (equation 2), the thermal steady state is reached when metabolism (always a source of heat production) is coupled with, or balanced against, effective avenues of heat exchange, and no net heat gain or loss takes place. A constant internal temperature is the direct result of the algebraic sum of these influences, and nothing else. Such a thermal situation exists for the homeotherm after enough heat has been stored to elevate its temperature above that of the environment and within the thermal-control zone (37° to 39° C for most mammals).

EQUATION

- 1 $\Delta H = (\text{HEAT GAIN}) - (\text{HEAT LOSS})$
- 2 $\Delta H = \text{METABOLISM} \pm \text{HEAT EXCHANGE}$
- 3 $\Delta H = \text{METABOLISM} \pm C_d \pm C_v \pm R_d - E_v$

WHERE:

ΔH = NET HEAT FLUX; IF EQUAL TO ZERO,
A THERMAL STEADY STATE IS ACHIEVED.

C_d = CONDUCTION

C_v = CONVECTION

R_d = RADIATION

E_v = EVAPORATION

FIGURE 1. Basic heat-exchange equations.

Specifically, the avenues of heat exchange are (equation 3, Figure 1) conduction (C_d), convection (C_v), radiation (R_d), and evaporation (E_v), each of which (except evaporation) can be a source of heat gain or loss, depending on environmental and physiological circumstances. Heat flow along any of these routes requires an energy potential gradient. For conduction, convection, and radiation, a thermal gradient is required; heat loss by evaporation requires a difference in water vapor pressures. Detailed examination of the physical variables associated with each of

these energy exchange avenues is available elsewhere^{4, 16, 20, 64, 74}; the present discussion will be limited to evaluating each of these forces as they supply the physical framework for homeostasis.

Heat transfer by conduction is defined as thermal energy exchange through a medium, or between objects in physical contact, by the transfer of kinetic intramolecular energy, without involving the physical transfer of material. With an existing temperature difference, heat can be exchanged (either gained or lost) between the bottoms of the feet and the floor, between the skin and contact clothing, between and through adjacent body tissues, etc. Since homeotherms normally have only a small body area in contact with other objects in the environment (except for clothing), conduction is usually not a major avenue of environmental heat exchange for the warm-blooded animal.

Convection, a special case of conduction, is most often considered as a separate avenue of heat exchange. It is distinguished as a route of thermal energy flow depending upon the movement of a fluid over a surface that is at a different temperature. Two types of convection are usually identified. In natural (or passive) convection, the fluid (liquid or gas) flow is a function of differences in density within the fluid produced by differences in temperature (for example, warm air rising from a flame or over a warm surface). Forced convection, which increases the rate of heat exchange, requires an additional energy source external to the heat-exchanging system itself to move the fluid (for example, an electric fan or a stirring motor). In addition to the physical considerations for conduction, heat exchange by convection also varies with the fluid characteristics (viscosity, density, etc.), and the surface features of the object that affect surrounding fluid movement (surface texture, irregularities, shape, etc.). Most important and effective convective heat exchange within the body occurs by tissue blood flow. Since the movement of blood through the circulatory system depends ultimately on the expenditure of myocardial metabolic energy, tissue perfusion is an example of forced convection. The free movement of blood through the vasodilated body serves not only to minimize intracorporeal temperature differences, but also is an important mechanism for exchange of body heat with the environment.

Heat exchange by convection between the body surface and the surrounding air (or water) can be reduced by the interposal of any material that would inhibit free fluid flow. Trapping air within or beneath clothing or within fur provides a still fluid layer in contact with the skin, which reduces heat exchange by convection and establishes an important protective microenvironment immediately surrounding the animal. Any factor that damages the integrity of this still fluid layer facilitates increased convective heat exchange. For example, exercise in the cold air can increase heat loss markedly from the skin surface due to limb and body movements.^{24, 43} Less heat would be lost were the same amount of exercise performed isometrically.

Heat transfer by the exchange of electromagnetic energies between facing surfaces is called radiation. This heat-exchange route is unique in that it does not depend upon contact between heat-exchanging masses, but can take place through low-density environments (for example, as solar radiation does through "space"). All objects above a temperature of absolute zero (-273°C) exchange radiative energy with every other facing object (or with its own facing surfaces), with the net gain or loss of heat depending upon differences in temperature and other surface characteristics (e.g., emissivity: whether an object is a good "reflector" or "emitter" of radiant energy).^{16, 44}

Heat loss by evaporation depends upon thermal transfer in the conversion of a material from a liquid to a gas phase. Evaporation is always a heat-dissipating phenomenon; the energy stored by gas molecules can be released during the process of condensation. The dissipation of heat by evaporation can occur from any wetted body surface exposed to a less-than-saturated gaseous environment (e.g., from the respiratory-tract surfaces, the skin with or without sweat-gland activity, or from the wetted fur). Homeotherms exposed to environmental temperatures higher than skin temperature depend solely upon heat loss by evaporation to retain a thermal steady state (equation 3). If exposed under such a condition in air that is saturated with water vapor (i.e., 100% relative humidity; no vapor-pressure gradient can be established), progressive hyperthermia develops at the accumulated rates of metabolic heat production and heat gains from the environment through conduction, convection,

and radiation. Even though sweat-gland activity (or panting) were maximal under these conditions, no effective cooling could occur since evaporative processes would be precluded.

An analogy expressing the interaction of these factors is presented in Figure 2.

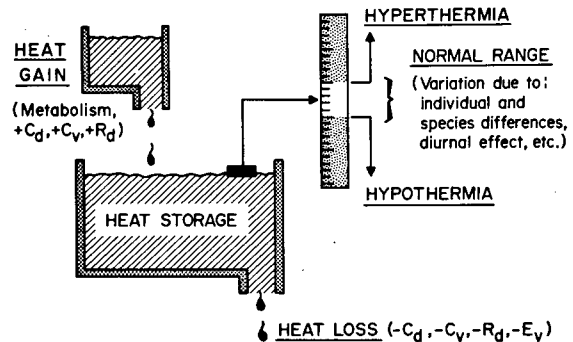


FIGURE 2. Body temperature is maintained within a normal range as the amount of stored heat is balanced by rates of net heat gain and loss.

In conceptualizing a specific thermal exposure, it is important to recognize that seldom do any of the four avenues of heat exchange operate alone in effective biothermal responses; the thermal threat to the homeotherm and the counteracting physiological defense is effected rather in terms of the algebraic sum of these interacting forces.

C. The Physiological Basis for Biothermal Control. Figure 3 shows how the avenues of heat exchange operate together to transfer thermal energy (during an air exposure) from the clothed or bare skin surface. This body cross-section shows the three major temperatures to be considered, air (T_a), skin (T_s), and "core" (T_r) or deep body temperature, usually evaluated as rectal temperature. Modifying heat-exchange rates along this axis are the summing insulations of the outer body area ("shell") [I_t], the clothing [I_{cl}], and the air [I_a]. Tissue insulation [I_t] can vary due to rates of local blood flow [C_{ve}] (i.e., internal body convection) to make the "shell" (that portion of the body mass in which there are temperature gradients and that will change its temperature being thermal stress) occupy approximately one-half of the total body mass (during massive peripheral vasoconstriction and extended cold exposure), or bring the "core" (that portion of

the body that is at a near constant temperature)* close to the skin surface (during cutaneous vasodilation and heat exposure). Heat exchange (double-headed arrows) can occur by conduction (C_d) both within the body between adjacent tissues at different temperatures or to objects touching the skin surface (including clothing). Heat loss (single-headed arrows) by evaporation can occur directly from deep body masses (within the "core") by evaporation from nasopharyngeal, buccal, lingual, palatine, and respiratory tract surfaces (E_{va}) or from the skin and clothing surfaces (E_{vb}). Heat exchange by radiation (R_d) and convection (C_v) to the surrounding environment can take place from any exposed surface. The terms "core" and "shell" are, of course, functionally defined in terms of the heat content and thermal gradients within different body masses; they do not have fixed anatomical counterparts.

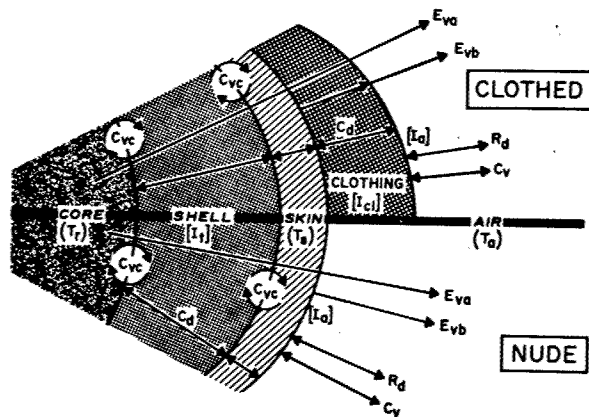


FIGURE 3. Internal and environmental physical heat-exchange routes are indicated for both clothed (upper) and nude (lower-air exposures).

Partitional calorimetric testing has shown that during a whole-body exposure (human) with air and wall temperatures at 24°C ., with low relative humidity and little forced-air movement, evaporation accounts for approximately 21%, radiation 37%, and conduction and convection together 42% of the total dissipated heat.⁷⁴ These proportions can be drastically different for ex-

* Small temperature differences exist between organs and structures deep in the body and even in the ventricular cavities during different phases of the cardiac cycle (due to heat released during myocardial contraction). Although these differences in temperature should be considered in evaluating the rationale for selecting a single temperature measurement to reflect accurate internal mean temperature, it should not be confusing in the general but conceptually valuable identification of the "core."

posures in environments with other characteristics.

A situation analogous to that presented in Figure 3 can be envisioned for a water rather than an air exposure. Additional appreciation of the physical factors involved in the avenues of heat exchange may also be gained by speculating how the equations in Figure 1 would have to be re-balanced, or the avenues of heat exchange shown in Figure 3 reevaluated for a water exposure or during exposure to environments of low density ("space").

At least three separate thermal functions can be attributed to that portion of the body near the skin surface, the "shell," which is not (as defined) at deep body temperature. In addition to serving as a site for convective heat exchange by perfusing blood, decreased heat loss during cold exposure results as the uniformly warm body mass (the "core") withdraws from the skin surface to deeper body areas, interposing a vasoconstricted tissue mass between itself and the environment.⁷⁵ This "shell," so established, offers some degree of thermal insulation ($[I_i]$, Figure 3) that can be calculated.^{6, 7, 20} These circulatory adjustments can bring about a change in tissue insulation from approximately 25 to 10 kcal/hr \cdot m² \cdot $^{\circ}\text{C}$ with a change in ambient temperature from 35 to 10°C .³⁰ Deposition of subcutaneous fat layers can increase tissue insulation over that achieved by vasoconstriction alone in the human,^{22, 25} as it can in arctic animals.⁵¹ Since the "shell" can transiently change its heat content, it can serve an additional role of thermal capacitance that acts as a buffer to internal-temperature changes by means of its ability to affect either positive or negative heat storage. This effect has been shown to be of functional importance at least to desert mammals.⁷¹

Besides the physiological adjustment of the avenues of heat exchange ("physical temperature regulation"), most homeotherms can provide large amounts of thermal energy during an acute cold exposure by involuntary muscular activity, shivering⁴⁹ ("chemical temperature regulation"), or by voluntary activity (Table 1). For the human, both metabolic heat production and skin-surface evaporation of secreted sweat are critically important in extending the environmental thermal limits provided only by circulatory adjustments in which a thermal steady state can

be enjoyed, as shown in Figure 4. Although the ambient temperature at which "chemical temperature regulation" begins (so-called "critical temperature")⁷³ is only a degree or so below normal room temperature for the human, some furred animals are able to maintain a constant internal temperature without augmenting physical defenses by shivering in air temperatures as low as -40°C .⁷³ Increases in heat production by shivering are effective in maintaining thermal balance only in acute cold stress; protection during chronic cold exposure must rest upon mechanisms for retaining rather than generating heat.⁵¹

TABLE 1. Different levels of physical activity with corresponding value of heat production³

Activity	Metabolic rate*	"Met"***
Sleeping	36	0.7
Basal	40	0.8
Sitting (resting)	50	1.0
Standing	60	1.2
Walking (level)		
1.5 mph	90	1.8
3.0 mph	130	2.6
4.0 mph	180	3.6
Running (level)		
10 mph	500	10.0
Heavy work	860	17.2

* kcal/hr · m².

** 1 "met" 50 kcal/hr · m².

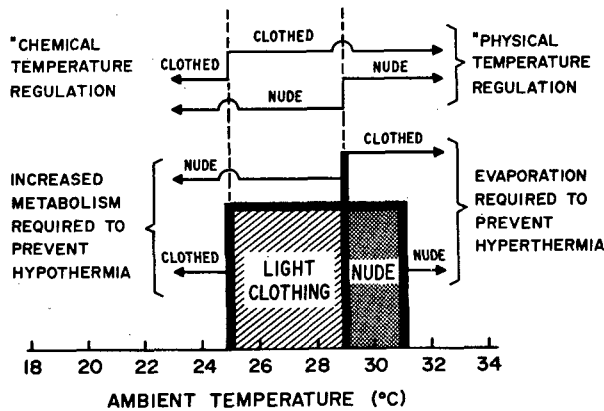
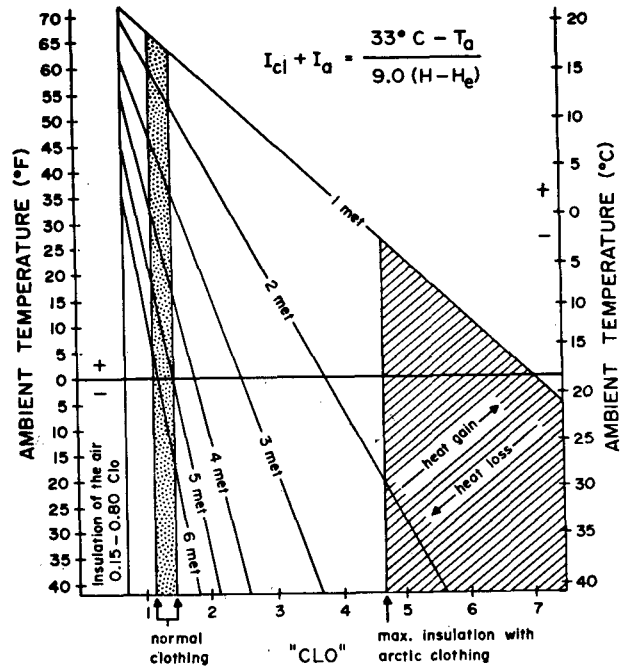


FIGURE 4. The temperature ranges of effective vaso-motor regulation are shown for the human nude and with light clothing. Exposure to temperatures above each upper limit requires sweat-gland activity to maintain normothermia; normal temperature stability during an exposure to temperatures below each lower limit requires metabolic heat production.

A very general estimate of the flexibility of the interacting influences of clothing and physical activity during exposure to cold is shown in Figure 5. More accurate relations for "comfort"

and behavioral integrity are available elsewhere.^{20, 53, 74} Predictions of homeothermal status are being made available for extreme exposures through the use of computer analogues of operating thermoregulatory systems^{18, 28} as well as through human testing.



1 met = 50 kcal/hr · m²

1 met = 0.18 °C / kcal · m² · hr

the amount of insulative clothing required to maintain a resting-sitting man with a metab. of 1 met indefinitely comfortable at an envir. temp. of 21°C (70°F)

Reference: Adams, T. Environmental Factors Influencing Thermal Exchange. Tech. Rept. No. 59-22, Ladd AFB, Alaska, 1960.

FIGURE 5. This chart shows the approximate relationship between ambient temperature and the units of insulation (expressed as "clo") required to maintain thermal comfort. It will in addition indicate the varying degrees of heat loss (or gain) and levels of thermal equilibrium under varying degrees of heat production and exercise. No estimates can be made with this diagram to include the effects of wind velocity greater than 20 ft/min.

Example:

72° F. with normal clothing at rest (1 met) is thermally equal to 54° F. with 3 clo, is equal to 41° F. with 4 clo, is equal to 27° F. with 5 clo, etc.

Example:

0° F. with 7.2 clo at rest is equal to 3.7 clo at 0° F. at 2 met, is equal to 2.5 clo at 0° F. at 3 met, etc.

Example:

-40°F . with 2.5 clo at 4 met is an isothermal condition equal to 57° F. with normal clothing at 2 met, is equal to 54° F. with 3 clo at rest, etc.

(Continued on page 6)

(Continued from page 5)

Example:

The heat loss (degree of thermal stress) at rest with normal clothing at 40° F. is equal to the thermal stress encountered at 45° F. with no clothing at rest, is equal at 20° F. with 3 clo at rest, etc.

Example:

The thermal stress at -35° F. with 3 clo at 3 met is equal to 46° F. with no clothing at 3 met, is equal to 0° F. with 2.1 clo at 3 met, is equal to 30° F. with normal clothing at 3 met, etc.

Example:

The heat loss at 15° F. with 4.5 clo at 1 met is equal to the heat loss at 10° F. with 2.7 clo at 2 met, is equal to the heat loss at 5° F. with 2 clo at 3 met, etc.

Internal body temperature can be protected during a cold exposure not only by appropriate and effective physiological reflexes that reduce heat loss at the body surface and increase metabolic heat production, but also by critically redistributing stored calories. Analogous to the conservation of body fluids by renal counter-current mechanisms, decreased heat loss has been described to take place in the extremities of many animals (including humans) by a circulatory, counter-current heat-exchange system.⁷² Figure 6 shows how this effect occurs. During a cold exposure, venous blood normally returning through cutaneous channels is shunted by vaso-motor adjustments to veins deeper in the axis of the extremity. Through the anatomical juxtaposition of these arteries and veins (venae comitees) warm arterial blood coursing peripherally can lose heat by conduction (C_d) to the corresponding vein carrying cooler blood centrally. The temperature difference between the warm arterial blood and the cooler venous blood is due, of course, to some heat loss to the environment at the skin surface. The end effect is to establish an additional site for this exchange of heat within the body (by heat transfer to the returning venous blood), which keeps the thermal energy within the body and reduces heat loss to the environment (extremity temperature is reduced since the arterial blood entering the peripheral portions of the limb is cooler). This type of thermal response, in conjunction with skin vasoconstriction reflexes, established a temperature gradient longitudinally as well as transversely in the extremity.¹⁹

Figure 7 illustrates how the alternate superficial venous-return pathways can also have ther-

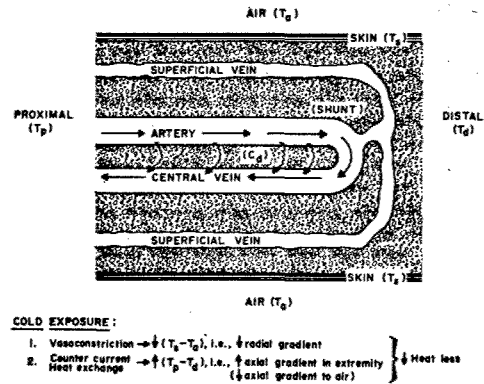


FIGURE 6. Section through an extremity showing the circulatory basis for counter-current heat exchange.

moregulatory importance. During exposure to air warmer than skin temperature, venous blood shunted to the skin surface decreases the gradient along which (and the rate at which) heat is gained from the environment (skin temperature approaches environmental temperature). Similarly, during exercise at ambient temperatures lower than skin temperature, the increased heat loss required to maintain thermal balance in the face of increased metabolic heat production is facilitated by increasing (even more) the skin-ambient temperature gradient.

Both in the case of heat exposure and of exercise at lower air temperatures, required heat loss from the skin surface by evaporation is increased due to the increase in water-vapor pressure accompanying the rise in skin temperature; sweat-gland activity consequently becomes a more

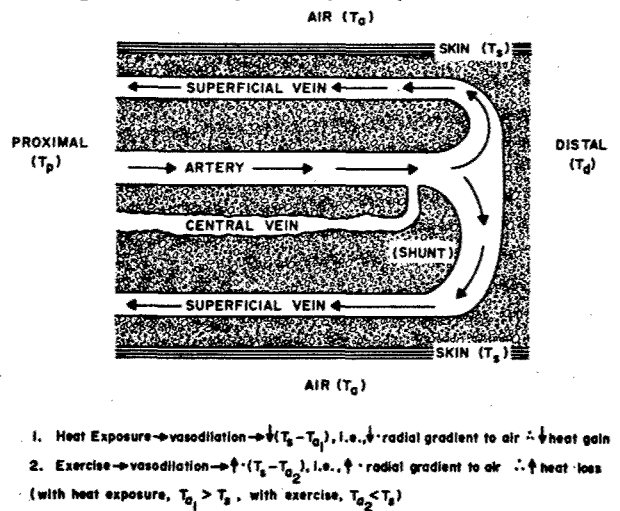


FIGURE 7. Section through an extremity showing the circulatory adjustment for environmental heat exchange at the skin surface.

efficient mechanism for losing heat as long as the environment is not saturated with water vapor.

In addition to satisfying skin metabolic requirements, the cutaneous circulation can vary considerably in performing its role in thermoregulation. Skin blood flow in a nude resting man at 82° F. ambient temperature is estimated to be 400 cc/min.³⁸ This flow can be increased by a factor of 3 with work at 70° F.,¹⁷ or during rest at 100° F; working at 100° F. will raise cutaneous circulation to approximately 9,000 cc/min.¹⁷

Succinctly, an acute human response to cold is characterized by: (1) peripheral cutaneous vasoconstriction (which results in an increase in tissue insulation); expansion of the "shell;" withdrawal of the "core;" (2) increase heat production by shivering; and (3) shunting of venous blood in the extremities to deep vessels for counter-current heat exchange.* An acute human response to heat stress would entail: (1) peripheral cutaneous vasodilation (decrease in peripheral tissue insulation); expansion of the "core;" reduction of the "shell," (2) increased sweat-gland activity, and (3) the superficial return of venous blood.**

In spite of the cursory presentation here, it should be clearly recognized that the physiological responses to heat and cold stress and the consequent readjustment of heat within the body are far from simple and their details are not completely understood. More detailed descriptions of the characteristics of heat, cold, hibernation, etc., are available elsewhere.^{20, 42}

D. *The Neurophysiological Control of Body Temperature.* As might be expected for any unit that remains as stable as homeothermic deep body temperature (although not necessarily total body-heat content due to thermal transients within the "shell") under the buffeting of internal and external thermal loads, the subserving control system is complex. As with any control system, however, it retains the basic components of input (internal and skin temperature) and output (metabolism, shivering, sweating, vasomotor control, etc.) operating through an in-

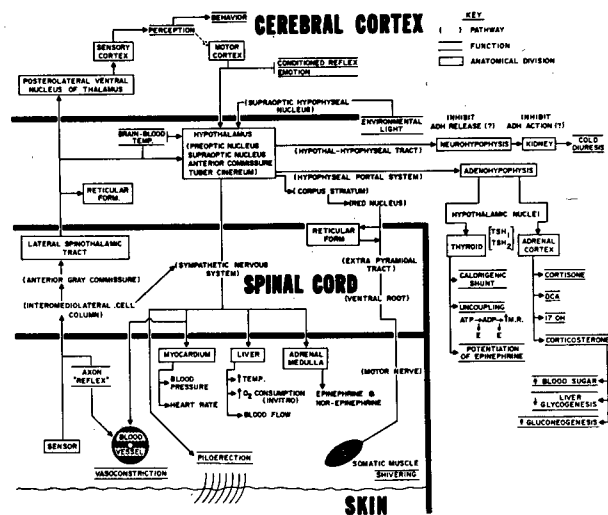


FIGURE 8. A general outline of the reflex pathways involved in homeothermic protections against hypothermia.

tegrational level (hypothalamic central-nervous-system areas). Figure 8 presents an outline of these operational levels for responses to cold.

The general response pattern for cold-elicited reflexes shown in Figure 8 is that temperature receptors within the skin⁴⁶ provide information (via the lateral spinothalamic tract) afferently into neurointegrational levels within the diencephalon (hypothalamus), which reflectly relays appropriate effector responses through the sympathetic and somatic motor pathways and into metabolic channels. A similar, grossly oversimplified circuit can be described for defenses against hyperthermia.

Just before the turn of this century, it became apparent that internal body temperature, in addition to the more obvious influence of local skin temperature, was influential in triggering thermal responses.^{66, 68} It became clear later that the neurons and synaptic activity in regions of the anterior hypothalamus were especially susceptible to local temperature effects,^{44, 60} and that the temperature of this area serves functionally as an additional thermal input into the biothermal control system. Since there are no anatomically identifiable temperature receptors in the anterior hypothalamus, as there are in the skin, it should be emphasized that the effect of temperature within the central nervous system is a direct, thermal modification of activity rather than one in which information (in the form of action potentials) is fed into a central

* In furred animals, this series of responses would be supplemented by piloerection, which would trap air near the skin and reduce the rate of skin-surface convection heat loss.

** In furred animals, in the absence of effective sweat-gland activity, increased evaporative cooling would occur through panting, and in extreme body heating, by copious salivation and licking of the fur.

integrational area; the terms "thermal sensibility" (to denote the action of temperature directly on central nervous system cells) and "thermal sensitivity" (to identify peripheral (skin) temperature receptor function) seem appropriate.⁶

Local anterior hypothalamic heating and cooling in unanesthetized animals^{6, 33, 36} have emphasized the earlier suggestion that local central temperature as well as skin temperature must be considered as functional temperature-regulation control-system inputs. Local diencephalic heating produces many of the characteristic heat responses as does (to a more limited extent) local hypothalamic cooling initiate predictable cold defenses. Questions related to the preferential influence of either of these factors in cold and heat exposure and acclimatization largely remain open. The identification of the neural characteristics of anterior hypothalamic neurons, in contrast to others within the central nervous system, is not complete.⁴⁰ The basic features of control-system regulation that are most likely involved in body-temperature control have been presented.⁴²

It should not be forgotten that thermal-control effectors serve other functions for the organism and may operate effectively outside of hypothesized biothermal control. For example, it has been shown recently that sweat-gland activity is triggered almost immediately upon initiation of voluntary activity,^{13, 61} before there are any measurable changes in skin or central temperature; the total response, however, can (under some circumstances) be greatly influenced by skin temperature.^{13, 14} Although it can be argued teleologically that this response is involved in the preparatory phases of thermal control and is effected in anticipation of the increased heat production associated with exercise, details of its action are missing. The control of sweating during exercise is discussed in greater detail in the following section.

Sweating, panting, shivering, peripheral vasomotor responses, and other thermoregulatory effector actions serve the same biothermal ends, to offset net heat fluxes impinging through environmental and exercise induced heat stress and to retain a constant internal temperature. Matching of appropriate responses to the thermal situation is the role of central neural reflexes subserving temperature regulation. Research leading to the

detailed elucidation of these controls has only begun.

II. Thermal Balance During Exercise and Environmental Stress.

When man exercises, he must dissipate the heat produced by the muscles or increase body temperature. If heat generated during exercise can be lost to the environment, a new thermal steady state can be achieved; voluntary activity is then limited by factors other than elevated body temperature. If humidity is high, or if clothing inhibits heat loss by radiation, convection, conduction, and the evaporation of sweat, body temperature increases as a function of the exercise rate with voluntary activity terminating as hyperthermia ensues.

A. *Exercise in Heat and Humidity.* Semi-nude man resting at 82° F. (a "neutral" temperature) produces heat at the rate of 80 kcal/hr. Approximately 20 kcal/hr are dissipated as water evaporates from respiratory and skin surfaces (without sweating, so-called "insensible water loss"); 60 kcal/hr are lost by radiation, convection, and conduction. During heavy exercise, heat production may rise as high as 960 kcal/hr (12 times resting). The major portion of this heat is brought to the body surface by increased skin blood flow (400 cc/min at rest compared to 1,200 cc/min during exercise)¹⁷; skin-surface heat loss is further increased by the evaporation of sweat. Since each cubic centimeter of water evaporated requires 0.58 kcal, a sweating rate of 3,000 cc/hr can dissipate heat at a rate of 1,740 kcal/hr.

In addition to attempting heat dissipation during exercise, an advantage may be taken of the thermal capacitance of the "shell" with some heat being stored (increasing body temperature; see page 00). This effect is valuable in heavy exercise for short periods (e.g., track events of a mile or less). No satisfactory explanation is available to indicate why body heat storage is tolerated better during exercise than by resting man during heat exposure.

The neurophysiological control system through which heat is dissipated by increased skin-surface evaporative water loss is particularly important in extending the upper limit of "physical temperature regulation" (see Figure 4) during whole-body heat exposure, and especially during prolonged exercise. There appears to be a ther-

moregulatory response to exercise in which internal body temperature is controlled at a new, higher level. The elevation over resting internal temperatures depends upon the rate of work⁶⁵ and is generally uninfluenced by ambient temperature (within the limits of biothermal ability). Further, during different steady state workloads at a uniform ambient temperature, sweat rates and internal temperatures are linearly related and skin temperature remains constant. Conversely, when the same level of work is performed at different ambient temperatures, internal body temperature remains constant and sweat rates rise as a linear function of average skin temperature.⁶⁵ These data provide a valuable extension of the relationship between sweat rate and internal temperature reported earlier.¹⁴

Resting man (non-heat-acclimatized) exposed to a hot environment (as high as 120° F.) can maintain a normal body temperature by increasing cardiac output and skin blood flow and evaporating sweat. Superimposition of requirements to dissipate metabolic heat during exercise increases circulatory strain (the requirement to provide blood flow to the exercising muscles and to the skin for heat dissipation), leading to increased internal-body and skin-surface temperatures, nausea, dizziness, and syncope.⁷⁰ Heat production approximately 8 times resting at 100°F increases skin blood flow about 4 times over resting levels at the same temperature.¹⁷ Heat dissipation at ambient temperatures greater than skin temperature depends solely on the evaporation of secreted sweat. The less clothing that may form a vapor barrier and the drier the surrounding air, the greater ease with which exercise can be performed in the heat. Human heat acclimatization involves changes in blood volume and peripheral circulation, which reduces cardiovascular strain during exercise in the heat and improves sweating efficiency.⁵⁷

The comfort and ease of physiological adjustment to a heat stress, either at rest or during exercise, is a combined function of ambient temperature and humidity.^{37, 50} Those geographical areas that typically have high ambient temperatures in the summer (100°F or more), usually have low relative humidity. This allows for the rapid and complete evaporation of sweat on the skin and clothing surfaces and a relatively effortless thermal adjustment. Those areas, however, with high water-vapor pressures (although

lower ambient temperatures) have a much less tolerable summer climate. Since the ready evaporation of sweat is not possible with high relative humidity, exercise thermal tolerances are thereby limited.⁷⁶ The same, or greater, amount of water and salt may be lost in secreted sweat in the wet-hot climate, in contrast to the dry-hot environment; the thermal balance question is more related to its evaporation than simply its production: Table 2 illustrates how sweat secretion increases when humidity is increased. Resting man at 110°F ambient temperature more than triples sweat secretion when relative humidity increases from 30% to 84%. Working man at the same dry-bulb temperature almost doubles sweat secretion when relative humidity increases from "low" (about 30%) to 57%. The excellent chapter by Ladell⁵⁷ should be consulted for a comprehensive treatment of the physiological effects of humid heat in man.

B. Water and Work. Sweating removes from the body both water and salts. The loss of water leads to dehydration and, eventually to decreased performance. As blood volume decreases with the progressive loss of body water, metabolic as well as heat-exchanging circulatory functions become impaired. Further dehydration (3.0% of body weight) reduces sweat-gland activity.⁶⁷ Sweat-gland function during exercise in either hot or cold environments brings about the same general effects. After exercise, rehydration by voluntary water intake does not replace the total amount of water lost until several days^{8, 47}; the physiological mechanism for this latency is not clear.

Inattention to body-hydration levels during competitive sports can result directly in reduced performance or, more seriously, set the stage for heat exhaustion. A decrease in body water by 5% reduces maximum oxygen consumption (the highest rate at which aerobic work can be performed)²¹ and performance (the skill required for a particular task).⁸ Whereas particular attention should be paid to drinking adequate amounts of water during and after exercise,⁵⁶ the requirement to replace lost Na, K, and Cl can be normally met during meals.^{34, 52} Since both water and inorganic salt are lost in the sweat, excessive water intake alone can result in below normal levels of tonicity and consequent discomfort or heat cramps or both. The periodic replacement of body water and salts during work

TABLE 2. Sweat rates for two levels of activity at two dry-bulb temperatures and at various humidities.

Rest (80 kcal/hr)	Ref.	Dry bulb °F	Wet bulb °F	Relative humidity %	Sweat rate l/hr
	37	80	66	47	0.05
	37	110	82	30	0.42
	37	110	95	57	0.84
	37	110	105	84	1.38
Work (350 kcal/hr)					
	8	80	—	low	0.45
	8	110	—	low	1.05
	50	110	90	46	1.60
	50	110	95	57	1.90

in hot environments, or at any time when sweating is maintained, is essential to insure performance standards and general well being.⁵² Figure 9 illustrates the importance of water replacement when men work in a hot environment. Rectal temperature of the "no-water" group exceeds 102°F after 4 hours of work, while rectal temperature of the "water ad lib" group remains below 102°F for more than 5 hours.

EFFECT OF WATER CONSUMPTION ON MARCHING IN THE HEAT
TEMPERATURE 100° RELATIVE HUMIDITY 35 - 45 %

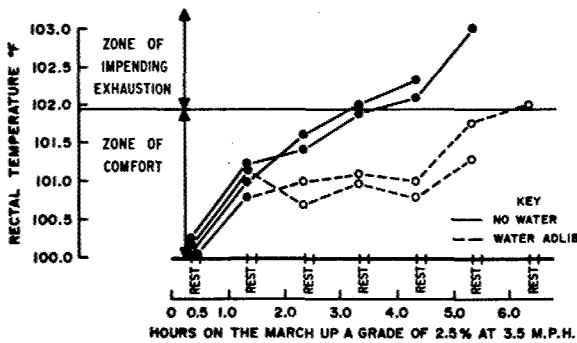


FIGURE 9. The effect of water intake on rectal temperature during work in the heat.⁵²

C. *Thermal Acclimatization.* Chronic exposure to hot or cold environments results in a progressive change in the way an animal defends internal thermal stability. These adjustments have been characterized³⁵ as:

1. *Acclimation:* systemic or cellular changes in an individual on a daily basis in direct response to an identified stress,
2. *Acclimatization:* long-term responses or adjustments of an individual to an identified stress,
3. *Adaptation:* phylogenetic adjustment or compensation to any environmental condition.

Physiological heat acclimatization processes for humans are well defined,^{10, 11} although for cold acclimatization they remain somewhat equivocal. Heat-acclimatized individuals demonstrate an increased ability to work in the heat and respond to an acute heat stress with a more rapidly elicited sudomotor reflex and the production of a more dilute sweat. Although the total potentials for sweat secretion is enhanced, less sweat is produced for mildly stressing heat exposures (at rest) compared to matching responses of non-heat-acclimatized humans. Heat acclimatization has also been reported to be accompanied by a decrease in "cardiovascular stress"¹¹ during acute heat exposures and an increase in total blood volume. This latter claim may have to be identified by the measurement method and the experimental condition.^{12, 69} The most striking component of human heat acclimatization is the increased ability and ease with which the superimposed stresses of exercise and acute heat exposure are met during the first 5 to 7 days of chronic heat exposure. It is thought that acclimatization to heat is not complete unless work is performed in the heat; exposure to heat alone confers but little acclimatization. Recent work, however, indicates that acclimatization may be accomplished by controlled hyperthermia alone.³²

This evidence may have practical importance in competitive events involving teams with different thermal histories. Men normally exercising in warm or hot environments may well be expected to perform better in competition at high ambient temperatures than others usually exercising at comfortable or cool temperatures. Individuals from climates cooler than that in which exercise is to be performed may improve competitive potential by thermal "preconditioning".

Moderate exercise (about 350 kcal/hr) for 2 to 3 hours daily at an ambient temperature a few degrees warmer than that anticipated for an impending event, 10 to 14 days before competition, could conceivably extend thermal-exercise limits, improve performance, and reduce physiological strain.

The experimental identification of human, whole-body cold acclimatization has stirred a controversy for many years, which at present is far from resolved. Although regional skin cold acclimatization for the human is well documented,^{5, 31, 59, 62, 63} the precise character of whole-body changes with chronic cold stress remains elusive. Experimental programs reporting physiological changes in "indoor" men who (for the purposes of the experiment) live in a cold environment must face the question of possible artifacts introduced by physical fitness changes.^{2, 45, 55} These programs must also identify how thermal stress occurs when, in many studies, the subjects are provided with thermally insulating clothing; it would seem that exposure to the microclimate established by such clothing would preclude the required experimental condition of cold exposure (except perhaps for the hands, feet, and face). Attempts to demonstrate cold acclimatization in human populations indigenous to arctic or subarctic regions leaves unresolved problems of (at least) racial, ethnic, and dietary involvements.¹ Although many reports have been published in this area, equivocation on the basis of subject selection, use, training and maintenance, the nature of the chronic and acute cold exposures used in the tests, adequacy, appropriateness and precision of employed physiological tests, and, of course, individual data interpretation in view of these variables, serve to cloud the issue.

Nonetheless, some experiments in which subjects have been chronically exposed to cold (unprotected by microenvironments generated by thermally insulating clothing) strongly suggest that increased heat production without shivering (so called "nonshivering thermogenesis") can be developed in humans^{26, 29} as it can in other species.^{27, 39} Although many more

data must be available before these questions are answered, the general pattern of human cold acclimatization is hinted to be related to increased heat production through shivering⁴⁸ and endocrine or intracellular adjustments, a greater heat flux with increased "shell" and decreased "core" partitioning of the main body mass (i.e., increased tissue insulation),²³ but reduced peripheral vasoconstriction,⁷⁵ leading to warmer hands and feet. Although obligating a source of increased heat loss, the warmer extremities may play the protective role of reducing peripheral cold injury and maintaining manual dexterity. There is little evidence to support the suggestion that a reduced thermoregulatory "set-point" accompanies these changes.⁵⁸ Parallel tests of heat acclimatization and exercise, some effort has been directed toward defining in man cold acclimatization in terms of metabolic responses to exercise in the cold.⁹

In spite of difficulty in identifying the whole-body cold acclimatization indexes, local cold acclimatization for the human has been well documented both by "field" and laboratory testing.^{5, 31, 62, 63} Humans who chronically expose their extremities to cold air or water show an alteration in local circulation during acute cold stress (extremity immersion in well-stirred ice water) that can be interpreted as a protective adjustment. Their extremities do not cool as deeply and remain generally warmer than those of persons not having experienced long-term local cold exposure. Increased protection against local cold injury and maintained manual dexterity may well be achieved prior to *de facto* cold stress by repeated, local, cold exposures.

The physiology of exercise cannot be separated from the homeothermic demands of the environment. Performance capability can be easily sacrificed by a lack of understanding or respect for thermoregulatory requirements. The cost is, of course, the termination of voluntary exercise by hyperthermia before cardiovascular, muscular, and pulmonary limits are met. The recognition of only a few thermoregulatory concepts can pay a multifold return in improved individual and group performances.

REFERENCES

1. ADAMS T. and COVINO, B. G.: *Jour. Appl. Physiol.* 12: 9, 1958.
2. ADAMS, T., and HEBERLING, E. J.: *Jour. Appl. Physiol.* 13: 226, 1958.
3. ADAMS, T.: The Control of Body Temperature. TR 59-21, AAC, AAL, Alaska, 1960.
4. ADAMS, T.: Environmental Factors Influencing Thermal Exchange. TR 59-22, AAC, AAL, Alaska, 1960.
5. ADAM, T., and SMITH, R. E.: *Jour. Appl. Physiol.* 17: 312, 1962.
6. ADAMS, T.: *Jour. Appl. Physiol.* 18: 772, 1963.
7. ADAMS, T.: *Jour. Appl. Physiol.* 18: 778, 1963.
8. ADOLPH, E. F., and COWORKERS: *Physiology of Man in the Desert.* Interscience Publishers, Inc., New York, 1947.
9. ANDERSON, K. L., STROMMIE, S., and ELSNER, R. W.: Metabolic and Thermal Responses to Muscular Exercise in the Cold. TD Rpt. No. AAC-TDR-61-52, Fort Wainwright, Alaska, 1952.
10. BASS, D. E., KLEEMAN, C. R., QUINN, M., HENSCHEL, A., HEGNAUER, A. H.: *Medicine.* 34: 323, 1955.
11. BASS, D. E.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 28. Reinhold Publ. Corp., New York, 1963.
12. BAZETT, H. C., SUNDERMAN, F. W., DOUPE, J. and SCOTT, J. C.: *Am. J. Physiol.* 129: 69, 1940.
13. BEAUMONT, W. VAN, and BULLARD, R. W.: *Science.* 141: 643, 1963.
14. BENZINGER, T. H., KITZINGER, C., and PRATT, A.W.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 56. Reinhold Publ. Corp., New York, 1963.
15. BOGERT, C. M.: *Scientific American.* 200:105, 1959.
16. BRENGELMANN, G., and BROWN, A. C.: In *Physiology and Biophysics.* T. C. Ruch and H. D. Patton, eds., Chap. 54. W. B. Saunders Co., Phil, 1965.
17. BROUHA, W. L., and RADFORD, E. P., Jr.: *Science and Medicine of Exercise and Sports,* W. R. Johnson, ed., Chap. 10. Harper and Brothers Publishers, New York, 1960.
18. BROWN, A. C.: Analogy Computer Simulation of Temperature Regulation in Man. TD Rpt. No. AMRL-TDR-63-116. Wright-Patterson AFB, Ohio, 1963.
19. BRUCK, K. and HENSEL, H.: *Pflug. Arch. ges physiol.* 257: 70, 1953.
20. BURTON, A. C., and EDHOLM, O. G.: *Man in Cold Environment.* Edward Arnold Ltd., Lond., 1955.
21. BUSKIRK, E. R., IAMPIETRO, P. F., and BASS, D.E.: *Jour. Appl. Physiol.* 12: 189, 1958.
22. BUSKIRK, E. R., THOMPSON, R. H., and WHEDON, G. D.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 37. Reinhold Publ. Corp., New York, 1963.
23. CARLSON, L. D., YOUNG, A. C., BURNS, H.L., and QUINTON, W. F.: Acclimatization to Cold Environment. USAF Tech. Rept. No. 6247, 1951.
24. CARLSON, L. D., PEARL, D., and SCHEYER, W.: *Am. J. Physiol.* 179:625, 1954.
25. CARLSON, L. D., HSIEH, A. C. L., FULLERTON, F., and ELSNER, R. W.: *J. Aviat. Med.* 29:145, 1958.
26. CARLSON, L. D., and THURSH, H. L.: Human Acclimatization to Cold. Contract No. AF41 (657)-212, 1959.
27. COTTLE, W. H., and CARLSON, L. D.: *Proc. Soc. Exper. Biol. Med.* 92: 845, 1956.
28. CROSBIE, R. J., HARDY, J. D. and FESSENDEN, E.: Temperature, Its Measurement and Control in Science and Industry, Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 55. Reinhold Publ. Corp., New York, 1963.
29. DAVIS, T. R. A.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 38. Reinhold Publ. Corp., New York, 1963.
30. DAY, R.: The Effect of Cold on Man. Josiah Macy, Jr. Foundation Review Series, 1(2)L1-64, 1943.
31. ELSNER, R. W., NELMS, J. D., and IRVING, L.: *Jour. Appl. Physiol.* 15: 662, 1960.
32. FOX, R. H.: The Physiology of Human Survival, O. G. Edholm and A. L. Bacharach, eds., Chap. 3. Academic Press, New York, 1965.
33. FREEMAN, W. J., and DAVIS, D. D.: *Am. J. Physiol.* 197: 145, 1959.
34. FREGLY, M. J., and IAMPIETRO, P. F.: *Metabolism* 7: 624, 1958.
35. FRY, F. E. J.: *Ann. Rev. Physiol.* 20: 207, 1958.
36. FUSCO, M.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 51. Reinhold Publ. Corp., New York, 1963.
37. GOLDMAN, R. F., GREEN, E. B., and IAMPIETRO, P. F.: *Jour. Appl. Physiol.* 20: 271, 1965.
38. GREENFIELD, A. D. M.: Circulation, W. F. Hamilton and P. Dow, eds., Chap. 39. American Physiological Society, Washington, D.C., 1963.
39. HANNON, J. P.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 41. Reinhold Publ. Corp., New York, 1963.
40. HARDY, J. D.: The Physiology of Temperature Regulation Rept. No. 22, NADC-MA-6015. U. S. Naval Air Development Center, Johnsville, Pa., 1960.

41. HARDY, J. D., and HAMMEL, H. T.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 54. Reinhold Publ. Corp., New York, 1963.
42. HARDY, J. D.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed. Reinhold Publ. Corp., New York, 1963.
43. HART, J. S.: *Canad. J. Zool.* 30: 90, 1952.
44. HASAMA, B.: *Arch. f. exper. Path. u. Pharmak.* 153: 257, 1930.
45. HEBERLING, E. W., and ADAMS, T., *Jour. App. Physiol.* 16:226, 1961.
46. HENSEL, H.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 19. Reinhold Publ. Corp., New York, 1963.
47. IAMPIETRO, P. F., VAUGHAN, J. A., MACLEOD, A. R., WELCH, B. E., MARCINEK, J. G., MANN, J. B., GROTHEER, M. P., and FRIEDEMANN, T. E.: Caloric Intake and Energy Expenditure of Eleven Men in a Desert Environment. Tech. Rept. EP-40, QM, R & D Center, U. S. Army, Natick, Mass., 1956.
48. IAMPIETRO, P. F., BASS, D. E., and BUSKIRK, E. R.: *Metabolism.* 7: 149, 1958.
49. IAMPIETRO, P. F., VAUGHAN, J. A., GOLDMAN, R. F., KREIDER, M. B., MASUCCI, F., and BASS, D. E.: *Jour. Appl. Physiol.* 15: 632, 1960.
50. IAMPIETRO, P. F., and GOLDMAN, R. F.: *Jour. Appl. Physiol.* 20: 73, 1965.
51. IRVING, L.: *Scientific American.* 214: 94, 1966.
52. JOHNSON, R. E., BELDING, H. S., CONSOLAZIO, F. C., and PITTS, G. C.: The requirements of water and of sodium chloride for the best performance of men working in hot climates. Harvard Fatigue Lab. Rept. No. 13, 1942.
53. KAUFMAN, W. C.: *Aerospace Med.* 34:889, 1963.
54. KAUFMAN, W. C., and PITTMAN, J. C.: *Aerospace Med.* 35: 1167, 1964.
55. KEATINGE, W. R.: *J. Physiol.* 157: 209, 1961.
56. LADELL, W. S. S.: *J. Physiol.* (London), 12: 11, 1955.
57. LADELL, W. S. S.: Adaptation to the Environment, D. B. DILL, E. F. ADOLPH, and C. G. WILBER, eds. American Physiological Society, Washington, D.C., 1964.
58. LEBLANC, J.: *Jour. Appl. Physiol.* 9: 395, 1956.
59. LEBLANC, J., HILDES, J. A., and HEROUX, O.: *Jour. Appl. Physiol.* 15: 1031, 1960.
60. MAGOUN, H. W., HARRISON, F., BROBECK, J. R., and RANSON, S. W.: *J. Neurophysiol.* 1: 101, 1938.
61. MEYER, F. R., ROBINSON, S., NEWTON, J. L., TS'AO, C. H., and HOLGERSEN, L. O.: *Physiologist.* 5: 182, 1962.
62. MILLER, L. K., and IRVING, L.: *Jour. Appl. Physiol.* 17: 449, 1962.
63. NELMS, J. D., and SOPER, D. J. G.: *Jour. Appl. Physiol.* 17: 444, 1962.
64. NEWBURGH, L. H.: Physiology of Heat Regulation and the Science of Clothing, L. H. Newburgh, ed. W. B. Saunders Co., Phil., 1949.
65. NIELSEN, B. and NIELSEN, M.: *Acta Physiol. Scand.* 64:314, 1965.
66. OTT, I.: *J. Nerv. Ment. Dis.* 14: 152, 1887.
67. PEARCY, M., ROBINSON S., MILLER, D. I., THOMAS, J. T., and DEBROTA, J.: *Jour. Appl. Physiol.* 8; 621, 1956.
68. RICHET, C.: *Pflueger Arch ges physiol.* 37: 624, 1885.
69. ROBINSON, S.: *Ann. Rev. Physiol.* 14: 73, 1952.
70. ROBINSON, S.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 27. Reinhold Publ. Corp., New York, 1963.
71. SCHMIDT-NIELSEN, K.: *Scientific American.* 201: 140, 1959.
72. SCHMIDT-NIELSEN, K.: Temperature, Its Measurement and Control in Science and Industry. Vol. 3, Part 3, Biology and Medicine, J. D. Hardy, ed., Chap. 14. Reinhold Publ. Corp., New York, 1963.
73. SCHOLANDER, P. F., HOCK, R., and WALTERS, V.: *Biol. Bull.* 99: 237, 1950.
74. WINSLOW, C. E. A., and HERRINGTON, L. P.: Temperature and Human Life. Princeton Univ. Press, Princeton, N. J., 1949.
75. WOOD, J. E., BASS, D. E., and IAMPIETRO, P. F.: *Jour. Appl. Physiol.* 12: 357, 1958.
76. WYNDHAM, C. H., STRYDOM, H. B., MORRISON, J. F., WILLIAMS, C. G., BREDELL, G. A. G., MARITZ, J. S., and MUNRO, A.: *Jour. Appl. Physiol.* 20: 37, 1965.