4. Mechanisms of heat exchange: biophysics and physiology A . P H A R O G A G G E Human Heat Balance Equation Independent Variables Affecting the Thermal Environment Ambient temper...

Chapter · October 2019

Some of the authors of this publication are also working on these related projects:

On Old Olympus Towering Top View project

4. Mechanisms of heat exchange: biophysics and physiology

A . P H A R O G A G G E | John B. Pierce Foundation Laboratory, Yale University, New Haven, Connecticut

R I C H A R D R . G O N Z A L E Z | U.S. Army Research Institute, Environmental Medicine, Biophysics and Biomedical Modeling Division, Natick, Massachusetts

INITIAL EXPOSURE TO THE THERMAL ENVIRONMENT in all homeotherms starts at the skin surface. This thermal environment extends outward to the surrounding area with varied air temperature, humidity, thermal radiation, various barometric pressures caused by high and low altitudes, and other factors produced by the weather, as well as the integument and clothing. In general, some aspect of temperature characterizes all these factors; additionally, the ambient environment plays a crucial role in modifying heat transfer from the skin surface by four principal avenues of heat loss: radiation, convection, conduction, and evaporation.

This chapter is divided into two sections: the first considers the biophysics of human heat exchange and the second deals with the physiological responses of heat exchange common to all homeotherms. In general, a homeotherm's response to the thermal environment may be divided into three zones: (1) a zone of body cooling in which the regulation of body temperature is controllable by vasoconstriction followed by shivering until regulation becomes impossible and survival is

questionable, (2) a zone of evaporative regulation in which body temperature is regulated by vasodilation and sweating or some respiratory activity, and (3) a zone of body heating in which thermal regulation is impossible. Exposure time is a critical factor in the zones of body cooling and heating.

In humans, environmental temperatures that describe the transitional regions between the zone of body cooling and evaporative regulation govern responses associated with physiological thermal neutrality, neutral sensation, and the sense of thermal comfort. In the extreme zones of body cooling and body heating, the main concern is how tolerance times may be extended in such environments (91). Transitional temperatures at the upper and lower limits of the zone of evaporative regulation vary with the type of clothing worn, metabolic activity, and the type of environment. Humans adapt to their environment not only by structural and physiological responses but by behavioral temperature regulation through changes in clothing and activity and alteration of the thermal environment.

Metabolic heat that reaches the skin surface must leave by radiation, convection, conduction through clothing, and evaporation. Exchange by these processes is governed by the physical characteristics of the environment, such as ambient dry-bulb temperature and humidity, air movement, clothing, and barometric pressure, and by the physical properties of the skin surface: its temperature and wettedness (w) due to sweating.

HUMAN HEAT BALANCE EQUATION

The heat balance equation describing the thermal exchange between an individual's body and its environment (Fig. 4.1) is described in its classic form as:

$$S = M - (\pm Wk) \pm (R + C) - E$$
$$[W \cdot m^{-2}] \quad (Eq. 4.1)$$

S = rate of storage of body heat (+ for net gain)
M = rate of metabolic energy production (always +)
E = rate of evaporative heat transfer (− for net loss)
Wk = rate of work (+ for work against external forces, − for eccentric or negative work)
R = rate of radiant heat exchange (+ for a gain)
C = rate of convective heat transfer (+ for gain).

The outer skin surface is formally considered the boundary separating the homeotherm body core and the thermal environment. All terms in Eq. 4.1 are expressed in watts per square meter of outer skin area (W · m⁻²). In humans, the surface area is classically evaluated by the Dubois surface area (A_D) formula (49):

$$A_D = 0.202 \ (m)^{0.425}(H)^{0.725} \quad [m^2] \quad (Eq. 4.2)$$

where the total skin surface area of the human (A_D) is in square meters, body weight (m) is in kilograms, and height (H) is in meters.

Heat exchange at the skin surface takes the simplified format of Eq. 4.1 as:

$$S = M_{sk} \pm DRY - E_{sk} \quad [W \cdot m^{-2}] \quad (Eq. 4.3)$$

where M_{sk} is the net rate of metabolic heat to the skin surface (+ for net gain), E_{sk} is the net rate of evaporative heat transfer at the skin surface (− for net loss), and DRY signifies the rate of sensible heat flow from the skin surface incorporated by R and C.

In applications using Eq. 4.3 discussed in this chapter, the skin evaporative heat exchange (E_{sk}) always occurs on the skin surface, and the sensible heat exchange ($R + C$) from the human clothed body surface is always

$$S = M - (\pm W) - (R \pm C \pm K) - E$$
$$\dot{Q}_{sk} = (R \pm C \pm K) \pm E = h'(T_{sk} - T_0) + wh'_e (P_{s,sk} - P_a)$$
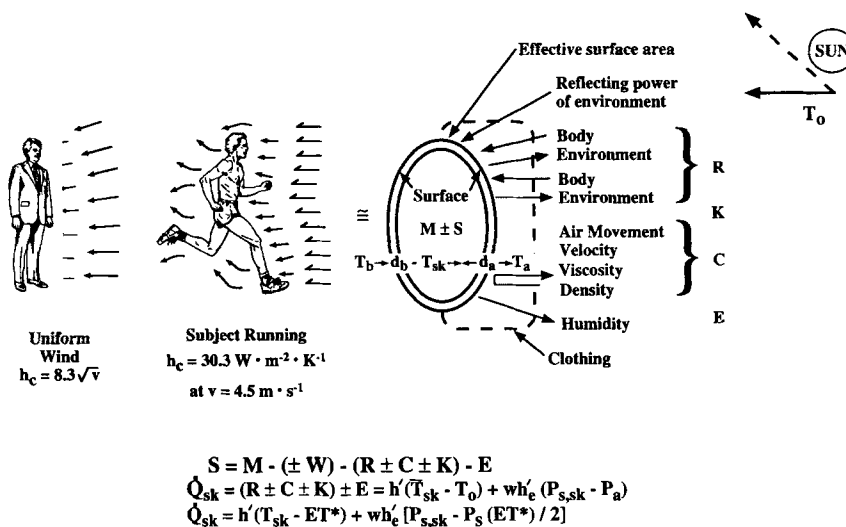$$\dot{Q}_{sk} = h'(T_{sk} - ET^*) + wh'_e [P_{s,sk} - P_s (ET^*)/2]$$

FIG. 4.1. Effects of the complex thermal environment on a clothed standing human and an exercising human. Heat balance is described in conventional and enthalpic formats.

represented as the dry heat flow from the skin surface to the clothing surface. As in Eq. 4.1, all terms in Eq. 4.3 are expressed in values of energy exchange per unit area of body surface, shown in Eq. 4.2.

INDEPENDENT VARIABLES AFFECTING THE THERMAL ENVIRONMENT

Six variables along with exposure time must be accounted for adequately in the description of any thermal environment (41, 49, 50, 61–63, 97, 98).

Ambient Temperature

The ambient temperature (T_a) in °C of a gaseous or liquid environment surrounding the body is measured at a point outside the thermal boundary layers over its surface. The ambient media are generally mixtures of air and water vapor.

Dew Point Temperature and Ambient Vapor Pressure

Dew point temperature (T_{dp}) is a fundamental measure of absolute humidity of air and is defined as the temperature at which condensation first occurs when an air–water vapor mixture is cooled at constant pressure. Ambient vapor pressure (P_a), when measured outside the body boundary layers, is an alternate measure of humidity, equivalent to dew point. Water vapor is always associated with saturation vapor pressure at some temperature (T, °C). Therefore, P_a is also the saturation pressure at dew point temperature and equals P_{dp}^*. The symbol P^* indicates saturation pressure and the subscript describes the given temperature in °C. Several meteorological tables and psychrometric charts are available which show the relationship of saturated water vapor pressure to temperature [units are in kilopascals, kPa, or Torr; 1 atmosphere (ATA) = 101,325 Pascals, Pa, $\approx$ 760 Torr]. The amount of water vapor present in air is characteristically presented in the above pressure units, albeit for calculations of flux transfer, units of concentration (g · m^{-3}) are often favored (97, 98).

The Antoine equation is a useful relationship for calculation of saturated vapor pressure for temperatures ranging from 0° to 60°C, accurate within ±0.02 Torr (47, 49):

$$P_{dp}^* = \text{antilog}_{10}[8.10765 - 1,750.286/(T_{dp} + 235)]$$
$$[\text{Torr}] \quad \text{(Eq. 4.4)}$$

or

$$P_{dp}^* = \exp[18.6686 - 4,030.183/(T_{dp} + 235)]$$
$$[\text{Torr}] \quad \text{(Eq. 4.4')}$$

Other measures of humidity that depend on ambient air (T_a) of dry-bulb (T_{db}) temperature are relative humidity (Φ) and wet-bulb temperature (T_{wb}, aspirated $\geq$ 3 m · s^{-1}). If any two of the five variables (T_a, P_a, T_{wb}, Φ, and T_{dp}) are known, the other three may be evaluated using psychrometric charts (47, 49, 98) or the following formats:

$$\Phi = P_a/P_a^*$$
$$= P_{dp}^*/P_a^* \quad \text{a nondimensional}$$
$$\text{(ND) fraction} \quad \text{(Eq. 4.4'')}$$

$$P_{dp}^* = [P_{wet}^* - 0.00066 \cdot P_B \cdot$$
$$(T_a - T_{wb}) (1 + 0.00115T_{wb})] \quad \text{(Eq. 4.4''')}$$
$$[\text{Torr}]$$

where P_B in Eq. 4.4''' is the barometric pressure in Torr. At sea level where P_B = 760 Torr:

$$P_{dp}^* \approx P_{wet}^* - 0.5 \cdot (T_a - T_{wb})$$

Air and Fluid Movement

The movement (V) of ambient air or other fluids in meters per second (m · s^{-1}) results from (1) the free buoyant motion caused by a warm body in cool air medium, (2) forced ventilation of the environment, and (3) bodily motion caused by activity of the object itself. Air movement is a difficult factor to measure consistently, but some degree of accuracy is possible using anemometers for the evaluation of forced ventilation. Air or fluid motion over a heated object is a fundamental property required for the complete analysis of both convective and evaporative heat exchange.

Mean Radiant Temperature and Effective Radiant Field

The essential environmental variables that govern heat exchange by thermal radiation include (1) in the temperature mode, for humans, *mean radiant temperature* ($\overline{T}_r$ or *MRT*), which can be defined as the temperature of an imaginary isothermal "black" enclosure in which humans would exchange the same amount of heat by radiation, as in the actual nonuniform environment; and (2) in an energy flux mode, *effective radiant field* (*ERF*), a heat transfer term (in W · m^{-2}) that relates *MRT* or the surrounding surface temperatures of an enclosure to air temperature. *ERF* is positive when *MRT* is greater than T_a and negative when *MRT* is less than T_a. In the actual "sensed" environment, the only factors that contribute significantly to *ERF* are surrounding bodies, intense directional radiant sources, and cold sinks, or other bodies at a temperature totally different from the ambient air temperature. Other interrelationships

between the above factors will be discussed in later sections for other homeotherms (3, 112).

Clothing Insulation

The classic unit of clothing insulation (I_{clo}) is the clo, a practical unit which represents the effective insulation provided by a normal business suit when worn by a resting person in a comfortable indoor environment (50). The value of one clo is equivalent to a thermal resistance of $0.1547 \ m^2 \cdot °C \cdot W^{-1}$; its conductance value is equivalent to $6.46 \ W \cdot m^{-2} \cdot °C^{-1}$. Alternatively, in resistance units 1 clo is equivalent to about $2 \ s \cdot cm^{-1}$ (31, 98).

Barometric Pressure

Barometric pressure (P_B) in Torr or kPa is a fundamental variable that affects heat transfer in air–water vapor media both by convection and by evaporation. At sea level P_B is 760 Torr (equivalent to 101.33 kPa, 1,013.33 millibar, or 1 ATA).

PERIPHERAL FACTORS TO HEAT EXCHANGE

Mean Skin Temperature

Skin temperature ($\overline{T}_{sk}$) is optimally measured using a radiometer, other infrared thermographic methods (35, 92), or, less accurately (particularly in cold ambients), various thermistors or thermocouples attached to a skin site (30, 92). In humans, mean $\overline{T}_{sk}$ is generally evaluated over the body as the average of at least eight local measurements of $\overline{T}_{sk}$ over the body surface, each site weighted by a fraction of the total Dubois surface area. One useful weighting scheme (49, 62, 110) partitions a percentage of the skin surface regions into head (7%), chest (17.5%), back (17.5%), upper arms (7%), forearms (7%), hands (5%), thighs (19%), and legs (20%).

In the zone of evaporative regulation, skin surface temperature is fairly uniform, and therefore, weighting factors are less significant in determining $\overline{T}_{sk}$. In cold zones, wide deviations in blood flow over the body surface can be expected, which hinder proper evaluation of $\overline{T}_{sk}$ (92).

Accurate measurements of skin temperature allow evaluations of temperature and skin vapor gradient that affect both the sensible (by R + C) and insensible (by E_{sk}) exchange from the body surface. Skin temperature serves as an important index in modulating thermal responsiveness important in temperature regulation. Skin temperature also serves as an index in sensory judg-

TABLE 4.1. *Relationships Between Skin Temperature ($\overline{T}_{sk}$) and Sensory Responses*

$\overline{T}_{sk}$ (°C)	State
45	Rapid tissue damage
43–41	Threshold of burning pain
41–39	Threshold of transient pain
39–35	Sense of "hot"
37–35	Initial sense of "warm"
34–33	Neutral temperature sense at rest, "comfortable"
33–32	Increasing cold sensation
32–30	
31–29	
25	
20	
15	Intolerably cold
5	
30–36°C range where $\overline{T}_{sk}$ is $\propto T_o$	Independent of heat production

Redrawn from Gagge and Nishi (49).

ments of heat, cold, pain, and hedonic assessment (56) of the thermal environment. Table 4.1 gives some general relationships between sensory state and skin temperature value.

Skin Wettedness

Skin wettedness (w) is defined as the ratio of an equivalent skin surface area (A_w), which, if completely covered with thermoregulatory sweat, would produce the observed skin evaporative heat loss (E_{sk}), to the total skin surface area (A_D) (45).

Body Heat Storage and Rate of Change of Mean Body Temperature

The rate of storage of body heat (S) is directly associated with the rate of change in integrated (that is, by both peripheral and central thermoreception) mean body temperature ($\Delta\overline{T}_b/\Delta t$)

$$S = (0.965 \cdot m/A_D) \cdot \Delta\overline{T}_b/\Delta t \quad [W \cdot m^{-2}] \quad (Eq. \ 4.5)$$

where 0.965 is the specific heat of the body in $W \cdot h \cdot °C^{-1} \cdot kg^{-1}$ (or $3.49 \ kJ \cdot kg^{-1}$, where 1 J/s = watt) and m is the body weight in kg; Δt is time in hours (36, 47, 49). If an initial mean body temperature $\overline{T}'_b$, can be determined accurately prior to an experimental exposure by a weighted average of $\overline{T}_{sk}$ and some measure of internal body temperature, such as the tympanic (T_{ty}), rectal (T_{re}), or esophageal (T_{es}) temperature, then:

$$\overline{T_b} = \overline{T_b'} + \sum_0^t (\Delta \overline{T_b}/\Delta t)\ dt \qquad [°C] \qquad \text{(Eq. 4.6)}$$

or

$$= \overline{T_b'} + [(S \cdot A_D)/(0.965 \cdot m)/t \qquad \text{(Eq. 4.6')}$$

where t is the time of exposure in hours.

In the cold, a probable steady-state weighting ratio for mean skin temperature to rectal temperature ($\overline{T_{sk}}$:T_{re}) is generally recognized as 1:2 (27, 49). In the heat, the probable ratio varies from 1:4 (27, 28) to 1:9 (59, 109) when esophageal or tympanic temperatures are used as measures of core temperature. In the zone of physiological thermal neutrality, which occurs during resting activity and when there is no regulation of body temperature by sweating or by vascular changes, mean skin temperature range is about 33.7° to 34°C, rectal temperature varies from 36.9° to 37.1°C, and esophageal temperature is about 36.6° to 36.8°C. The corresponding range of mean body temperature would be about 36.3°–36.5°C.

Metabolic Energy

The metabolic energy term, M, in the heat balance equation (Eq. 4.1) represents the free energy produced by the transformation of chemical energy during aerobic and anaerobic metabolic activities within an organism (29, 33, 137, 157). Except during transient bursts of exercise

and short periods following such exercise, the anaerobic component may be ignored and M may be measured by the rate of oxygen uptake, as given by the following:

$$M = (0.23[R] + 0.77) \cdot (5.873)(\dot{V}_{O_2}) \cdot$$
$$(60/A_D) \quad (W \cdot m^{-2}) \qquad \text{(Eq. 4.7)}$$

in which [R] is the respiratory exchange ratio that often varies from a value of 0.83 during rest to 1.0 during moderately heavy exercise. In Eq. 4.7, $\dot{V}_{O_2}$ is the rate of oxygen uptake in liters/min at standard temperature and pressure, dry (STPD), (0°C, 760 Torr, dry), and the constant (5.873) is the calorific equivalent of oxygen in watts · hour per liter or 21.14 kJ per liter (49, 103).

Metabolic energy, M, may be released (see Fig. 4.2) in four ways: (1) as net metabolic heat (M_{sk} or $\dot{Q}_{sk}$), which passes through the skin surface; (2) as heat of vaporization of respired water vapor (E_{res}); (3) as heat convected by respiration (C_{res}); and (4) as external work ($+Wk$). The net metabolic heat through the skin surface is:

$$M_{sk} = \dot{Q}_{sk} = M - E_{res} - C_{res} - (\pm Wk) \qquad \text{(Eq.4.8)}$$

in which (41, 150):

$$E_{res} = 0.0023M(44 - P_a) \qquad \text{(Eq. 4.9)}$$

and

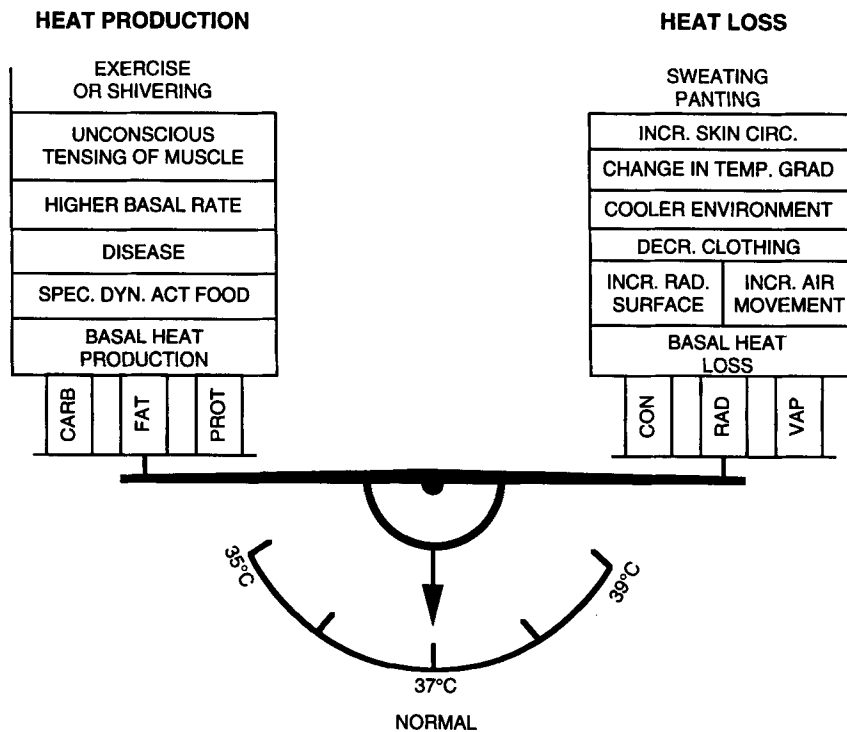$$C_{res} = 0.0014M(34 - T_a)(P_B/760) \qquad \text{(Eq.4.10)}$$

FIG. 4.2. Heat production and heat loss properties and the variables inherent in each.

TABLE 4.2. *Typical Responses to Physical Effort Experienced in an Average-Sized Individual*

Metabolic Effort	M, mets	$\dot{V}_{O_2}$ [$\ell \cdot$ min^{-1} (STPD)]	$\dot{V}_E$ [$\ell \cdot$ min^{-1} (STPD)]	Heart Rate (beats/min)
Basal	0.7–0.8	0.20–0.24	5.5–6.3	50–70
Sedentary	0.9–1.1	0.27–0.33	7.6–8.0	72–82
Very light	1.3–1.7	0.40–0.50	10–13	83–89
Light	2.2–2.8	0.66–0.84	17–23	90–95
Moderate	3.7–4.5	1.1–1.5	32–33	105–115
Heavy	5.2–6.4	1.6–1.9	41–50	125–135
Very heavy	6.7–8.3	2.0–3.4	52–64	145–155
Maximal	10–12	3.5–4.5	85–100	>180

1 met = 58.2 W $\cdot$ m^{-2} [from Gagge and Nishi (49)].

As indicated in the last term in Eq. 4.8, when work is done by the body on an external system, such as in positive work (+Wk), metabolic energy, which is expended as heat, must be reduced by the net value of (+Wk), as in a cycle ergometer or inclined treadmill. When work is done on the body by an external system, such as in eccentric or negative work (−Wk), mechanical heat is subsequently added to M. Net heat flux across the skin boundary layer is given by the rate of heat storage, S, in Eqs. 4.1 and 4.3. When S is positive, heat flows from the environment to the body (as a net gain) and mean body temperature rises.

In a typical environment (that is, T$_a$ of 25°C and 50% rh), the evaporative heat loss by the respiratory tract (E$_{res}$) accounts for about 7% of M and is often ignored for air at sea level. Work (Wk) can be measured accurately on a cycle ergometer or treadmill. The ratio Wk/M represents the mechanical efficiency (η) of the body doing work. In humans, maximum mechanical efficiency occurs while pedaling on a cycle ergometer and ranges about 18%–22% for an average person. During treadmill exercise, η is about 8%–10%. For level walking and during most stationary activities, (Wk) is ignored. Negative mechanical efficiency as occurring during −Wk, can amount to as high as 100% (49, 52).

Table 4.2 shows examples of typical physical effort associated with the expenditure of various levels of metabolic energy during steady-state (49, 50). Metabolic energy is expressed in *met* units (50), defined as 58.2 W $\cdot$ m^{-2} for a person of standard size (1.8 m^2 surface area; 70 kg body weight) in a resting, sitting position.

SENSIBLE HEAT EXCHANGE BY RADIATION AND CONVECTION

The exchange of sensible heat from the skin surface at average temperature ($\overline{T}_{sk}$) is usually accompanied ini-

tially by conduction (K) through clothing, followed by radiation and convection from the outer clothing or skin surface to the surrounding medium. A general biophysical view of sensible heat exchange begins with the formulation of *operative* temperature, addressed here for humans and later applied to other homeotherms.

Operative Temperature

The *operative temperature* (T$_o$) involved in human thermal environments is defined as the temperature of a uniform isothermal "black" enclosure in which a person would exchange the same heat by radiation and convection from the body surface as would be possible in the actual nonuniform environment (46, 154, 155). By definition, all the dry (sensible) heat exchange (DRY) from the body surface, whether clothed or not, at temperature $\overline{T}_{surf}$ is given by:

$$DRY = h(T_o - \overline{T}_{surf}) \qquad [\text{W} \cdot \text{m}^{-2}] \qquad (\text{Eq. 4.11})$$

where h is the combined coefficient for heat transfer by radiation (h$_r$) and convection (h$_c$) in W $\cdot$ m^{-2} $\cdot$ °C^{-1}. In terms of the definition for ambient air temperature (T$_a$) and mean radiant temperature $\overline{T}_r$ (or *MRT*):

$$DRY = h_c(T_a - \overline{T}_{surf}) + h_r(\overline{T}_r - \overline{T}_{surf}) \qquad (\text{Eq. 4.12})$$

in which h_c = convective heat transfer coefficient in W $\cdot$ m^{-2} $\cdot$ °C^{-1} and h_r = a linear radiation exchange coefficient.

By comparing Eqs. 4.11 and 4.12, the operative temperature becomes

$$T_o = (h_r\overline{T}_r + h_cT_a)/(h_r + h_c) \qquad [°C] \qquad (\text{Eq. 4.13})$$

and

$$h = h_r + h_c \qquad [\text{W} \cdot \text{m}^{-2} \cdot °\text{C}^{-1}] \qquad (\text{Eq. 4.14})$$

Equation 4.13 defines operative temperature as the average of T$_a$ and $\overline{T}_r$, weighted by each respective governing heat transfer coefficient.

Clothing Properties Effective in Sensible Heat Exchange

The role of clothing in attenuating heat transfer between the environment and specific skin segments of the human body varies greatly from person to person; it also depends on various metabolic activities and on type of clothing ensemble and fabric worn (22, 23, 64). The effective conductance of a set of clothing may be defined as the ratio of the dry heat exchange from the skin surface during thermal equilibrium to the average temperature gradient from the skin surface at $\overline{T}_{sk}$ to the outside clothing surface at the temperature $\overline{T}_{cl}$. If measured accurately (infrared radiometer), $\overline{T}_{cl}$ should theoretically have the same weighting as found in the individual

surface sites used in $\overline{T}_{sk}$ and also include temperatures of exposed skin areas. By such definition:

$$DRY = h_{cl}(\overline{T}_{cl} - \overline{T}_{sk}) \qquad [W \cdot m^{-2}] \qquad (Eq.\ 4.15)$$

where h_{cl} is the effective clothing conductance in $W \cdot °C^{-1}$ per square meter of skin surface rather than the clothing surface. The reciprocal of h_{cl}, or I_{cl}, is defined as the effective insulation of the clothing.

The dry heat exchange (DRY) from the clothing surface at temperature $\overline{T}_{cl}$ is given by:

$$DRY = h(T_o - \overline{T}_{cl}) \qquad [W \cdot m^{-2}] \qquad (Eq.\ 4.16)$$

In terms of $\overline{T}_{sk}$:

$$DRY = hF_{cl}(T_o - \overline{T}_{sk}) \qquad (Eq.\ 4.17)$$

where the factor

$$F_{cl} = h_{cl}/(h_{cl} + h) \qquad [ND] \qquad (Eq.\ 4.18)$$

or

$$F_{cl} = I_o/(I_o + I_{cl}) \qquad [ND] \qquad (Eq.\ 4.18')$$

In Eq. 4.18', I_o incorporates the thermal resistance of the *operative* environment as the reciprocal of h (the combined heat transfer coefficient), for example, $I_o = 1/(h_r + h_c)$.

The factor F_{cl}, first coined by Burton and Edholm (28) as a "thermal efficiency factor," can be used for quantifying sensible heat transfer from the skin surface to the ambient in terms of operative temperature. Using this factor, the effective combined heat transfer coefficient from the skin surface includes the product, $h \cdot F_{cl}$, which equals the sum of $(I_o + I_{cl})$ or the total clothing insulation I_T.

A uniformly heated, human-sized static or articulated copper manikin is generally used to evaluate intrinsic insulation, I_{cl}, for wide assortments of clothing ensembles because of the precise and controllable heat flux produced from such instruments (24). Observations of the $\overline{T}_{sk}$ and sensible heat exchange (power input to the total manikin surface area) are made during steady-state in a uniform environment. The product hF_{cl} or $(I_T)^{-1}$ can be calculated easily. From the measurements, I_{cl} may be found and the insulation in clo units (I_{clo}) is derived by $I_{cl}/0.1545$.

The Burton thermal efficiency factor, F_{cl}, can be redefined as:

$$F_{cl} = 1/(1 + 0.1545 \cdot I_{clo} \cdot h) \qquad (ND) \qquad (Eq.\ 4.19)$$

in which the insulation in terms of clo unit (I_{clo}) is the effective insulation of the clothing. By eliminating h in Eqs. 4.16 and 4.17 defining DRY:

$$F_{cl} = (T_o - \overline{T}_{cl})/(T_o - \overline{T}_{sk}) \qquad [ND] \qquad (Eq.\ 4.20)$$

and

$$\overline{T}_{cl} = T_o + F_{cl}(\overline{T}_{sk} - T_o) \qquad [°C] \qquad (Eq.\ 4.21)$$

If any three variables $\overline{T}_{sk}$, I_{clo}, and h are observed or calculated, it is possible using Eq. 4.21 to estimate the clothing temperature. Alternatively, the Burton clothing efficiency factor, F_{cl}, may also be calculated by direct measurements of T_o, $\overline{T}_{cl}$, and $\overline{T}_{sk}$. Other factors relating latent heat transfer, enthalpic gradients, and permeation through clothing will be considered in the section on Enthalpy and Rational Indices of the Human Environment.

RADIATION EXCHANGE

Mean Radiant Temperature and Effective Radiant Field

Mean radiant temperature ($\overline{T}_r$, °C) can be incorporated into the definition of an effective radiant field (ERF) using the following format (46, 48, 49):

$$ERF = h_r(\overline{T}_r - T_a) \qquad [W \cdot m^{-2}] \qquad (Eq.\ 4.22)$$

or

$$\overline{T}_r = T_a + ERF/h_r \qquad [°C] \qquad (Eq.\ 4.23)$$

Similarly, by substituting Eq. 4.23 into the original definition for *operative* temperature of Eq. 4.13:

$$T_o = T_a + ERF/h \qquad [°C] \qquad (Eq.\ 4.24)$$

or

$$ERF = h(T_o - T_a) \qquad [W \cdot m^{-2}] \qquad (Eq.\ 4.25)$$

Using these formats, the sensible heat exchange (R + C) in the heat balance equation may be rewritten separately for each respective transfer coefficient as:

$$R = h_r(T_{cl} - \overline{T}_r) \qquad (Eq.\ 4.26)$$

$$R = h_r(T_{cl} - T_a) - ERF \qquad (Eq.\ 4.26')$$

$$R = ERF \cdot F_{cl} + h_rF_{cl}(T_a - \overline{T}_{sk}) \qquad (Eq.\ 4.26'')$$

and

$$C = h_cF_{cl}(T_a - \overline{T}_{sk}) \qquad (Eq.\ 4.27)$$

$$C = h_c(T_{cl} - T_a) \qquad (4.27')$$

Dry heat exchange (R + C) for humans is therefore expressed as the sum of two modes: one directly associated with the effective radiant field, $ERF \cdot F_{cl}$, and the other in terms of heat flux from the skin surface to a uniformly heated ambient environment, $hF_{cl}(T_a - \overline{T}_{sk})$.

The Effective Radiant Field term in Eq. 4.26 incorporates the equivalent radiant heat absorbed and exchanged at the outer surface of the body, if such a surface were theoretically at ambient temperature. The extent of ERF is affected by the absorptance (α) of either the clothing (and its emissivity, ε) or the exposed skin

surfaces to the wavelength (λ) corresponding to the temperature of the radiating source. The radiant field absorbed is altered by the thermal efficiency factor (F_{cl}) of the clothing system.

In describing the sensible heat loss in terms of Eq. 4.26, the radiative heat transfer coefficient (h_r) is treated as a linear constant over a wide range of ambient temperatures. Its exact quantification may be derived by (46–48):

$$R = \varepsilon \cdot \sigma(A_r/A_D) \cdot f_{acl} \cdot [(\overline{T}_r + 273.15)^4 - (\overline{T}_{cl} + 273.15)^4] \quad [W \cdot m^{-2}] \quad (Eq.\ 4.26')$$

or

$$h_r(\overline{T}_r - \overline{T}_{cl})$$

where

$$h_r = \varepsilon \cdot 4\sigma \cdot (A_r/A_D) \cdot f_{acl} \cdot [(\overline{T}_{cl} + \overline{T}_r)/2 + 273.15]^3$$
$$(Eq.\ 4.26'')$$

The constant (σ) in Eqs. 4.26' and 4.26'' is the Stefan-Boltzmann constant and equals $5.67 \times 10^{-8}\ W \cdot m^{-2} \cdot °K^{-4}$. The Stefan-Boltzmann constant includes the emissivities of human skin ($\varepsilon = 0.98$) and clothing ($\varepsilon = 0.95$) (49, 61, 97).

The term (A_r/A_D) is the ratio of a 4π radiating area of the body surface "open" to thermal radiation over its Dubois total surface area ($A_D \cdot m^2$). A_r is always less than A_D because extremities or other surfaces radiate to each other, rather than to exterior surfaces. Other variables in the assumption of A_r depend on posture (sitting, standing, or crouching) and clothing folds (41). Several researchers have provided useful methods to establish A_r in respect to A_D (24, 25, 41), with variations occurring from $0.65 \cdot A_D$ for sitting to $0.85 \cdot A_D$ for a "spread eagle" position. A value of $0.72 \cdot A_D$ is a usual coefficient for a standing, clothed person. The factor f_{acl} expresses the increase in A_D due to clothing and can range from 1.0 for 0 clo to as high as 1.5 for 3 clo (22, 24).

ERF describes the net radiant exchange with all complex sources that radiate at temperatures different from the isotropic ambient (T_a). $(ERF)_n$ is a function of the radiating temperature of the source n in absolute temperature, the absorptance α of the exposed body or clothing surface for the emission of σ °K^4 by the source, and an appropriate view factor (F_{h-n}) that describes the fraction of a person's total body surface irradiated by the source (48, 49).

Any radiant source n contributing to total ERF is (48, 49):

$$(ERF)_n = f_{acl}(A_r/A_D)F_{h-n}\alpha_n [(T_n + 273)^4 - T_a + 273)^4] \quad [W \cdot m^{-2}] \quad (Eq.\ 4.28)$$

The view factor F_{h-n} refers to the body area A_D itself, rather than to the radiating area of the body surface (A_r

$\cdot f_{acl}$) corrected for clothing; the factor includes the fraction of heat flux leaving site h that reaches site n. Only for the simplest geometrical arrangements can values of F_{h-n} be easily evaluated (41).

In each condition during body thermal equilibrium, $(ERF)_n$ will include the energy absorbed by the body system via the outer surface of the body and is solely a function of T_r and T_a and is not a function of the temperature of the asymmetric source.

The ERF allows the facile assessment of the "radiation-needed" zone when outdoor air temperature drops below 20°C in the clothed condition. Radiation is based on a direct, easily quantified concept taking into account the possible solar radiation load necessary to maintain an optimum comfort zone. It offers a unique concept to clothing interaction with the environment in humans and, by specific modification of constants, can be applied to other animals as well (3, 98).

CONVECTIVE HEAT EXCHANGE

Heat Transfer Theory

Heat exchange occurring by convection between a heated source and the surrounding fluid has been quantified extensively by a series of fundamental nondimensional numbers (33, 34, 75, 90, 100, 102, 107, 114, 137). For various mammals, and humans in particular, extensive research has characterized the convective heat transfer coefficient (h_c, $W \cdot m^{-2} \cdot °C^{-1}$) applied in the heat balance equation as the ratio of the convective heat loss to the thermal gradient (ΔT) between the body surface and the ambient air, presumably at a lower body surface temperature. The convective heat transfer coefficient is formulated classically using the Nusselt number (Nu) as the product ($h_c \cdot d/k$), in which d is the diameter of the heated object (meters) and k refers to the thermal conductivity constant ($W \cdot m^{-1} \cdot °C^{-1}$) of the surrounding medium to which the object can effectively transfer heat.

Convection is a more complex process than conduction because it entails the mass movement of the medium which is transferring thermal energy from one site to another (90, 114). A simple illustration may be shown by connecting a source and sink of thermal energy with a tube of liquid or gas. At the warm end, the fluid rises to give place to a cooler fluid. At the other end the warmed fluid flows along the top from the warm to the cold end, where it deposits the thermal energy to the sink. Energy is conveyed from source to sink continuously unless blocked by a resistor. At the source or sink of thermal energy, air flow is low enough to be streamlined. Outside this film, where speeds are higher, the fluid becomes turbulent. In humans, the skin–air boundary layer flow is described as laminar or turbulent

according to the ratio of buoyancy forces to viscous forces in the layer [Grashof number, cf. ref (114)]. Buoyancy forces dominate if the Grashof number is greater than 10^{10} and the layer becomes turbulent. For example, a naked standing person at T_a of 15°C will have laminar flow up to the plane of center of the abdomen, a transitional region of 50–60 cm, and turbulent flow about the upper torso and head (35). The conventional non-dimensional numbers (114) are applicable in describing convective exchange primarily for uncovered or semicovered cylinders. Rationally derived equations extended for animal and human shapes (32, 100, 102) have been formulated, but there is little information on clothed humans or furred animals other than by direct or partitional calorimetry (36, 56, 97).

For forced convection the Nusselt number is a function of the Reynolds (Re) number and the Prandtl (Pr) number and is described by the following relation:

$$(Nu) \sim (Re)^{0.55} \cdot (Pr)^{0.33} \qquad (ND) \qquad (Eq.\ 4.29)$$

where

$$Re = \rho \cdot V \cdot \chi/\mu \text{ or } V \cdot d/\upsilon$$

in which ρ is the density (kg/m³), V the velocity in m/h, μ the viscosity in kg/(h · m), χ either the diameter (d) or distance (ℓ), and υ the kinematic viscosity in m²/h.

It is useful to point out (49) that: (1) for free convection, h_c varies with temperature gradient ΔT, the gravitational constant G (in the Grashof number) and density; (2) for forced convection, h_c varies with density and air motion and is independent of ΔT; (3) for both free and forced convection, h_c varies inversely with some function of χ included as a factor of the diameter (d) or length (ℓ) of the heated object; (4) in a space shuttle, however, free convection does not exist, as G = 0, and all convection becomes forced. Barometric pressure (P_B) affects density and thus h_c. Finally, the h_c directly proportional to V for a large object (mammal) would be lower than that for a small object in any given environment (97).

In the transitional region between free and forced convection, there is no simple relationship as described above. For the resting human body with a probable ΔT of 5°C from surface to ambient air, the transition occurs at 0.2–0.3 m · s⁻¹ (40–60 fpm) ambient air movement. Below this level h_c may be considered a constant (35, 114, 120, 121).

The convective heat transfer coefficient h_c, defined as the ratio of the total heat exchange by convection from body surface to the temperature gradient from the body surface to the ambient air temperature, is difficult to evaluate accurately in humans. Optimum calorimetric measurements with the heat balance equation (36) have been worked out and are, to an extent, accurate enough if the total dry heat exchange (R + C) is accurately mea-

sured from the observed M_{sk} and E_{sk}. This assumes conditions of thermal equilibrium when S is no greater than ±10% of M. Calorimetric measurements of the dry (R + C) heat exchange under these conditions result always in an accurate value only for the combined heat transfer coefficient (h = h_c + h_r). The value of h_c is now found by subtracting from h a calculated value for h_r (36, 47, 56).

Measurement of the Convective Heat Transfer Coefficient

The physical factors associated with h_c are not easily measurable. Conditions of free and forced convection may exist simultaneously; convection is also affected by the motion of the ambient air and by activity level. The ambient air movement (V), caused by either turbulent or laminar flow, may also affect h_c nonuniformly (75, 98, 101).

Nishi and Gagge (110) initially applied the sublimation properties of naphthalene to time average wind speeds associated with convective heat transfer in humans at rest and during exercise. This was done by attaching naphthalene spheres 2.4 cm from the skin boundary layer surface on various body segments. The regional h_c at each of the body segments as well as the mean weighted, whole-body h_c were derived from theoretical extrapolation between the Nusselt and Sherwood numbers applied to human shapes.

Typical formulae for measuring h_c in the indoor environment are given in Table 4.3 (49).

Effect of Altitude (Barometric Pressure) on Convective Heat Loss

The values of h_c shown in Table 4.3 can be utilized accurately in the evaluation of convective heat exchange only for sea-level environments. The effect of lower barometric pressures, such as those found in high terrestrial zones, alters h_c by a factor ($P_B/760$)exp, where

TABLE 4.3. *Equations for Calculating the Convective Heat Transfer Coefficient, h_c, (W · m⁻² · °C⁻¹)*

Activity Level	Equations for h_c
Sitting rest, stationary arm work	$h_c = 11.6(V_{air})^{0.5}$
Stationary treadmill walking	$h_c = 6.5(V_{move})^{0.39} + 1.96(V_{air})^{0.86}$
Free walking	$h_c = 8.6(V_{move})^{0.53} + 1.96(V_{air})^{0.86}$
Stationary cycle-ergometer at 50 rev · min⁻¹	$h_c = 5.5 + 1.96(V_{air})^{0.86}$

Adapted from Gagge and Nishi (49). V_{air} and V_{move} refer to air velocity due to a person's movement on a treadmill or a cycle ergometer or free walking.

the exponential factor (exp) has been shown to vary anywhere from 0.45 to 1.0 (49). A higher exponential factor will lower the effective convective heat exchange (C) in the skin heat balance equation by:

$$C = h_c F_{cl}(\overline{T}_{sk} - T_a)(P_B/760)\exp \qquad [\text{W} \cdot \text{m}^{-2}]$$
(Eq. 4.27'')

EVAPORATIVE HEAT EXCHANGE

Heat loss that occurs by evaporation of secreted sweat or liquid vapor from the skin surface in humans and animals provides for an effective biophysical mechanism opposing hyperthermia. In humans, evaporative heat loss (E) is a practical physiological strain indicator of increases in environmental heat stress (7, 8, 14, 15). Since the first application of direct calorimetry (12, 27, 28, 104, 154, 155), observed evaporative weight loss less the metabolic heat produced $(M - E)$, compared to skin to ambient temperature differences $(\overline{T}_{sk} - T_a)$, continues as the classical quantitative means by which a combined heat transfer coefficient is obtained (56).

Direct Measurement of Evaporative Heat Loss

Evaporative heat loss determined by direct calorimetry is generally obtained from alterations in the humidity ratio ($\text{kg}_{H_2O}/\text{kg}_{dry\ air}$) at the outlet of the calorimeter (29, 56, 62). During partitional calorimetry, total evaporative weight loss (E) is first found by determining the rate of change by body mass, $\dot{m}$ ($\delta m_b/\delta t$), measured by a sensitive balance or force transducer (49). E may be calculated by:

$$E = \dot{m} \cdot \lambda/A_D \quad [\text{W} \cdot \text{m}^{-2}] \quad (\text{Eq. 4.30})$$

where $\dot{m}$ is obtained by continuous measurement of rate of change of body weight (g/min) and λ is the latent heat of sweat evaporation (40.8 W $\cdot$ h $\cdot$ g^{-1}; 2.45 J $\cdot$ g^{-1}). Application of the technique in humans during heavy exercise, requires that $\dot{m}$ be corrected for the rate of change of CO_2 loss over O_2 gain by $\dot{m}_M = [(44(R) - 32)/22.4] \cdot \dot{V}_{O2}$, in g/min, where the respiratory exchange ratio R is unity. To determine E_{sk} (W $\cdot$ m^{-2}), evaporative heat loss must also be corrected for C_{res} and E_{res}.

Typically, in humans without active thermoregulatory sweating, weight loss due to respiratory and skin diffusion amounts to about 0.5 g $\cdot$ min^{-1}. During exercise of about 50% $\dot{V}_{O2max}$, in a moderately warm environment ($T_a = 30°C$; 40%–50% rh), a person's sweating rate can be about 10–12 g $\cdot$ min^{-1}. The steady-state sweating rate used in the Belding-Hatch heat strain index (7) estimates values of about 18–20 g $\cdot$ min^{-1}, though the maximal whole-body sweating observed in

extreme heat for a euhydrated person is some 25–30 g $\cdot$ min^{-1} for a short burst of activity (49, 75).

Maximum Evaporative Heat Loss from the Human Skin Surface

The extent to which rate of body weight loss is an accepted indicator of evaporative heat exchange, due to constant thermoregulatory sweating and its evaporation on the skin, is governed by the evaporative heat transfer coefficient and humidity (8, 14). The coefficient E_{sk} should always be lower than the maximum evaporative heat loss (E_{max}) possible for a total wet skin surface. Excessive sweating above E_{max} results in dripping and offers no evaporative cooling to the body, limiting tolerance to the environment (8, 55, 57).

This E_{max} from the totally wet skin surface is directly proportional to the concentration gradient ($\rho_{sk} - \rho_a$) from the exchange of water vapor on the skin surface to that in the ambient air and is described by the relation:

$$E_{max} = \lambda h_D(\rho_{sk} - \rho_a) \qquad (\text{W} \cdot \text{m}^{-2}) \qquad (\text{Eq. 4.31})$$

PSYCHROMETRICS OF THE HUMAN HEAT BALANCE EQUATION

In the previous two sections, sensible and insensible heat transfer were treated as analogous but distinct phenomena. The coefficient originally deriving from the Lewis relation (88) was extracted to link the coefficients of sensible and latent heat transfer and the law of conservation of energy, and the coefficient is used to determine the amount of evaporative heat transfer. The transfer of sensible heat is imposed by the environmental conditions; whatever remaining amount of heat transfer is necessary to satisfy the human energy balance occurs via evaporation of sweat (75, 97, 98).

The above approach is justified when emphasis is placed on attaining optimum thermal balance in the comfort zone, that is, when regulation of body temperature by evaporation (requirements for thermoregulatory sweating) remains a minor factor. The same approach holds true for cold environmental exposure. In warm conditions, however, regulation of body temperature by sweating becomes a major factor; in fact, sweating may become the only significant means of dissipating the metabolic energy when skin temperature equals ambient temperature (14, 15). Gonzalez et al. (55) showed that for exercising persons in hot environments, dew point temperature > 15°C plays a role as important as that of air temperature.

An integrated analysis of heat and mass transfer for all environments provides the only rational definition

for effective temperature (ET*), for humid operative temperature (T_{oh}) (49), and for adiabatic equivalent temperature (97, 98). Indeed, these three indices are directly related to enthalpy of the human environment. Using a Mollier-type psychrometric chart, Fobelets and Gagge (44) demonstrated that these three indices and an operating point, representing the actual exposure environment for a human, can be located on a straight line. This isothermal line depicts, graphically, the human body heat balance equation, for example, serving as the locus of environmental combinations of operative temperature, T_o, and water vapor partial pressure, P_a, which result in the same total amount of energy being exchanged between the skin and the ambient surroundings.

The Lewis relation ensues from the similitude between heat and mass transfer and relates the appropriate transfer coefficients in terms of a unity ratio of the combined heat transfer coefficient ($h = h_r + h_c$) to the product of the mass transfer coefficient and specific heat (for example, $h/h_m \cdot k \approx 1.0$). The formal explanation was initially characterized by Chaddock (personal communication, 1990) in clear distinction from the Lewis number, a dimensionless ratio of the thermal to mass diffusivity. Ascribing a value of unity for the ratio of $h/(h_m \cdot k)$ permits quantification of the sensible and latent heat flux from a completely wet surface in terms of the enthalpy difference as the driving force for the heat transfer from one surface to another. Application of Lewis's original theory to humid sea-level environments allows for the derivation of a coefficient that links sensible and latent heat transfer that becomes fully applicable to the human heat balance equation. It is assumed that latent heat transfer results from the evaporation of water and is directly related to the water vapor transfer. The Lewis relation coefficient evaluated at conventional sea-level environmental conditions for human heat transfer is 16.5 K/kPa (or 2.2°C/Torr).

Application of the theory also permits use of the interpretation of wet-bulb temperature and enthalpy loci on the Mollier chart in terms of combined heat and mass transfer. Chaddock's modification of Lewis's theory can be generazlized to quantify the transfer of heat from human skin by including the mass transfer properties of clothing, radiative heat transfer, and provision for incompletely wet skin areas (49, 62, 75).

Lewis Relation: Interpretation of Wet-Bulb Temperature and Enthalpy

Use of the Lewis relation for examining insensible heat transfer in the human body heat balance equation began initially in the late 1960s (120, 139). The coefficient was originally defined as the ratio of the heat to mass transfer coefficients:

$$\frac{h_c}{h_m} = \rho c_p \cdot P_{am} \cdot \left[\frac{\alpha}{D}\right]^{2/3} = 0.92 \cdot \rho \cdot c_p \quad \text{(Eq. 4.32)}$$

where,

h_c = convective heat transfer coefficient ($\text{W/m}^2 \cdot \text{K}$)

h_m = convective mass transfer coefficient (m/s or $\text{m}^3/\text{m}^2 \cdot \text{s}$)

ρ = specific mass of humid air (1.16 kg/m^3 at 25°C, 50% relative humidity)

c_p = specific heat of humid air (1.013 kJ/kg $\cdot$ K)

α/D = Lewis number, ratio of thermal to mass diffusivities

P_{am} = 1.04 correction factor, resulting from the fact that water vapor diffusion must be compensated by a counterflow of air to satisfy mass balance

The Lewis relation coefficient (LR) may be derived from the above equation by writing the equation for the latent heat exchange that corresponds to the previously mentioned mass transfer:

$$LR = \frac{H_{fg}}{\rho c_p 0.92} \cdot \frac{M_w}{R_u \cdot T} \quad \text{(Eq. 4.33)}$$

where

H_{fg} = latent heat of vaporization of water (2,450 kJ/kg)

M_w = molecular weight of water (18.015 kg/kgmole)

R_u = universal gas constant (8,314 J/kgmole $\cdot$ °K)

T = absolute temperature, °K

From Eqs. 4.32 and 4.33, at 25°C and 50% relative humidity:

$$LR = 16.5 \text{ K/kPa} \quad \text{(Eq. 4.33')}$$

The evaporative (latent) heat loss ($\dot{Q}_{m,cv}$) by convection per square meter of totally wet surface is given in terms of the convective heat transfer coefficient as:

$$\dot{Q}_{m,cv} = LR \cdot h_c(P_{s,sk} - P_a) \quad \text{(Eq. 4.34)}$$

$$\dot{Q}_{h,cv} = h_c(\overline{T}_{sk} - T_a) \quad \text{(4.34')}$$

Combining Eqs. 4.34 and 4.34':

$$\dot{Q}_{cv} = h_c[(\overline{T}_{sk} + LR \cdot P_{s,sk}) - (T_a + LR \cdot P_a)] \quad \text{(Eq. 4.34'')}$$

where $T_a + LR \cdot P_a = H/c_p$ is the enthalpy of air divided by its specific heat ($c_p = 1.01$ kJ/kg $\cdot$ K). By definition, when using a Mollier-type psychrometric chart, loci of constant wet-bulb temperature represent the combination of air temperature and humidity which will result in the same total heat transfer with the wet surface. The slope of these lines is $-LR$, analogous to the $-\gamma$ of Monteith (97, 98). Lines of constant wet-bulb temper-

ature and of constant enthalpy nearly coincide. As a result, enthalpy is a measure of the energy transfer potential of the environments, and the total energy transfer can be expressed in terms of this enthalpy potential (Eq. 4.34″). The reciprocal of LR (equivalent to the slope of wet-bulb loci on a Carrier-type psychrometric chart) is also referred to as the psychrometer ratio (97).

Generalization to Energy Transfer between Humans and the Environment

The transfer of energy (sensible or latent) through various nodes—from the human skin, through the air layer trapped underneath clothing, through clothing itself, and finally, to the ambient environment—can be associated with the theoretical formulation above. Energy exchange at the skin surface is:

$$\dot{Q}_{sk} = Dry + Latent\ heat \qquad \text{(Eq. 4.35)}$$

$$\dot{Q}_{sk} = M - Wk - (E_{res} + C_{res}) - (\pm S)$$
$$\text{(per Eq. 4.8)} \qquad \text{(Eq. 4.35′)}$$

The rate of heat storage term $(\pm S)$ is positive for body heating and negative for cooling. Equation 4.35′ of the original heat balance (Eq. 4.8) can be rewritten in the *enthalpic* format as:

$$\dot{Q}_{sk} = h' \cdot (t_{sk} - t_o) + w \cdot h_e' \cdot (p_{s,sk} - p_a)$$
$$(W \cdot m^{-2}) \qquad \text{(Eq. 4.36)}$$

where h_e' is effective insensible (latent) heat transfer coefficient and w skin wettedness. Solving Eq. 4.36 for w gives the following analytic definition of w:

$$w = \frac{\dot{Q}_{sk} - h' \cdot [\overline{T}_{sk} - T_o]}{h_e' \cdot [p_{s,sk} - p_a]}$$
$$= \frac{E_{req}}{E_{max}} \qquad \text{(ND)} \qquad \text{(Eq. 4.36′)}$$

The numerator of Eq. 4.36′ has been evaluated experimentally from observed rate of weight loss (55) and is equal to E_{req}, the evaporative heat loss required for thermal equilibrium; the denominator is the maximum possible evaporative cooling (E_{max}) and is a function of skin temperature, $\overline{T}_{sk}$, and ambient water vapor partial pressure, P_a.

Equation 4.36′ is linearly associated to T_o and P_a, the independent variables on the psychrometric chart. As a result, the original Eq. 4.36 (describing human thermal balance in the enthalpic format) is represented by a straight line on the psychrometric chart. Interpretation of that line is analogous to that done previously for a constant wet-bulb line (44, 97, 98); that is, it is the locus

of the combination of T_o and P_a that will result in the same energy transfer from the total skin surface to the environment. The slope of that line is given by $-w \cdot h_e'/h'$.

In a previous section, it was shown that:

$$h' = \left(\frac{1}{f_{cl} \cdot [h_c + h_r]} + \frac{1}{h_{cl}}\right)^{-1}$$
$$[W \cdot m^{-2} \cdot K^{-1}] \qquad \text{(Eq. 4.37)}$$

Rewriting the latent heat transfer coefficient in a consistent form:

$$h_e' = \left(\frac{1}{LR \cdot f_{cl} \cdot h_c} + \frac{1}{LR \cdot h_c \cdot i_{cl}}\right)^{-1}$$
$$[W \cdot m^{-2} \cdot kPa^{-1}] \qquad \text{(Eq. 4.38)}$$

where LR is the Lewis relation coefficient defined above. $LR \cdot h_c$ and $LR \cdot h_{cl} \cdot i_{cl}$ are, respectively, the evaporative heat transfer coefficients for the air boundary layer and for the clothing layer. The clothing constant (i_{cl}) is the vapor permeation efficiency factor for the clothing layer (94, 113). This factor has been shown to be an intrinsic property of general porous fabrics used in the clothing layer. It is nondimensional and measures the mass (water vapor) to heat transfer property of clothing relative to that of air.

Oohori et al. (113) originally described both h_{cl} and i_{cl} as i_L [coined after Lotens and van de Linde (94)] They introduced the factor i_a, signified as the permeation efficiency of the air layer itself. As such, $i_a = h_c/(h_c + h_r)$. Thus Eq. 4.38′ becomes:

$$h_e' = \left(\frac{1}{LR \cdot f_{cl} \cdot (h_c + h_r) \cdot i_a} + \frac{1}{LR \cdot h_{cl} \cdot i_{cl}}\right)^{-1}$$
$$[W \cdot m^{-2} \cdot kPa^{-1}] \qquad \text{(Eq. 4.38′)}$$

The two factors i_a (for radiation and convection through the air layer) and i_{cl} (for clothing) are interpreted as follows: (1) they correspond to the ratio of the actual mass transfer coefficient to the hypothetical mass transfer coefficients of a purely convective boundary layer having the same sensible heat transfer characteristic; and (2) they are limited to values <1.0 (49, 113). Typically, values of i_{cl} range from 0.35 to 0.45 for porous clothing fabrics. It has been shown that the ratio:

$$h_e'/h' = i_m \cdot LR \qquad [K/kPa] \qquad \text{(Eq. 4.39)}$$

where i_m is known in the literature as the Woodcock water vapor permeation constant (22, 159), which is related to i_a and i_{cl} by application of the relation (113):

$$\frac{1}{i_m} = \frac{F_{cl}}{i_a} + \frac{1 - F_{cl}}{i_{cl}} \qquad \text{(ND)} \qquad \text{(Eq. 4.40)}$$

where F_{cl} is Burton's clothing efficiency factor $h_{cl}/(h_{cl} + f_{cl} \cdot h)$, discussed above under Clothing Properties Effec-

tive in Sensible Heat Exchange. The same relation can be expressed in terms of a factor, F_{pcl}, originally described by Nishi and Gagge (111):

$$i_m = F_{pcl} \cdot i_a + (1 - F_{pcl}) \cdot i_{cl} \qquad [ND] \quad (4.40')$$

Enthalpy of the Human Environment

By incorporating the expression for h'_e, Eq. 4.36 of the original heat balance can be written in a form analogous to Eq. 4.40':

$$\dot{Q}_{sk} = h' \cdot [(\overline{T}_{sk} + w \cdot i_m \cdot LR \cdot P_{s,sk}) - (T_o + w \cdot i_m \cdot LR \cdot P_a)] \quad (\text{Eq. 4.41})$$

where $(T_o + w \cdot i_m \cdot LR \cdot P_a)$ performs the role of enthalpy divided by c_p, the specific heat of humid air $(c_p = 1.01$ kJ/kg · K). In Eq. 4.41, the enthalpy gradient is measured between the skin surface and the environment. Equation 4.41 can substitute as a useful enthalpic format for the human heat balance equation. The heat balance equation, during steady-state, can be expressed as a straight line on the psychrometric chart with slope $-(w \cdot i_m \cdot LR)$ or $-(w \cdot h'_e/h')$. The equation describes uniform loci of combinations of observed T_o and P_a that will result in the same energy transfer from the skin to the thermal environment (97, 98).

Enthalpy and the Rational Indices of the Human Environment

It is clear from their definitions that effective temperature (ET*), humid operative temperature (T_{oh}), and Monteith's adiabatic equivalent temperature (49, 97, 98) are easily characterized by discrete loci depicting the heat balance equation on a psychrometric chart in graphic format. Indeed, all three indices are defined as the temperature of hypothetical environments at 50%, 100%, and 0% relative humidity, respectively, in which a person would experience the same energy transfer as in the real ("sensed") environment. Consequently, there is a close relationship between these three indices and enthalpy as defined by Eq. 4.41. As an example, the definition of ET* (51, 55) is:

$$\dot{Q}_{sk} = h' \cdot (\overline{T}_{sk} - ET^*) + w \cdot h'_e \cdot$$
$$(P_{s,sk} - 0.5 \cdot P_{s,ET^*}) \qquad [W \cdot m^{-2}] \quad (\text{Eq. 4.42})$$

which can be rewritten in terms of the enthalpy potential:

$$\dot{Q}_{sk} = h' \cdot [(\overline{T}_{sk} + w \cdot i_m \cdot LR \cdot P_{s,sk}) - (ET^* + w \cdot i_m \cdot LR \cdot p_{s'}(ET^*)/2)] \quad (\text{Eq. 4.42'})$$

Defining the effective enthalpy of the human environment (tH_a):

$$(tH_a) = ET^* + w \cdot i_m \cdot LR \cdot p_s(ET^*)/2 \quad [\text{for } ET^*] \quad (\text{Eq. 4.43'})$$
$$= T_{oh} + w \cdot i_m \cdot LR \cdot p_s(T_{oh}) \quad [\text{for } T_{oh}] \quad (\text{Eq. 4.43''})$$
$$= T_o + w \cdot i_m \cdot LR \cdot p_{s(dp)}$$
$$[\text{for actual } T_o, P_a, \text{ or } P_{s(dp)}] \quad (\text{Eq. 4.43'''})$$

Enthalpy is expressed here in temperature units (instead of specific energy units) (Fig. 4.3), taking advantage of the fact that the conversion factor of c_p has a value very close to 1 kJ/(kg · K).

Similarly, at the total skin surface site, enthalpy may be defined as:

$$(tH_{sk} = [\overline{T}_{sk} + (w \cdot i_m \cdot LR) \cdot P_{s,sk}] \quad [\text{kJ/kg}] \quad (\text{Eq. 4.44})$$

Although there are many other ways of expressing the energy transfer from the skin surface (75, 97), the enthalpic form expressed here and earlier (44) has the advantage of integrating heat and mass transfer from a wet surface, for which there is a considerable amount of data (49), and also allows specific application to human heat balance.

Enthalpy temperature rationalized for the human skin surface is a useful index applied to both sensible and insensible heat transfer. It allows for a refined generalization of the fundamental heat and mass transfer principles (relation between total energy exchange and enthalpy gradient) to the human thermal environment.

PIERCE TWO-NODE MODEL OF THERMOREGULATION

As we can see, determination of the rational environmental indices can be introduced into a simple model that integrates the environment and predicts responses over a wide range of thermal challenges. In general, computer simulations of thermoregulatory responses have the advantage that they incorporate rational sets of equations for physically passive and physically active modes describing heat exchange (81, 137, 145, 157). Both passive and active systems interact in a closed-loop fashion (59, 145). The passive system defines the geometry of the body container as one or more segments, divides each of the segments into one or more tissue compartments, and calculates temperature distributions and rates of heat transfer within compartments and segments and between the container and the environment. The active system defines the response of physiological control mechanisms that attempt to alter rates of heat transfer in the passive system in response to deviations in compartment temperatures from certain threshold levels (59). The most rigorous physiological control system quantified to date is that of the thermoregulatory
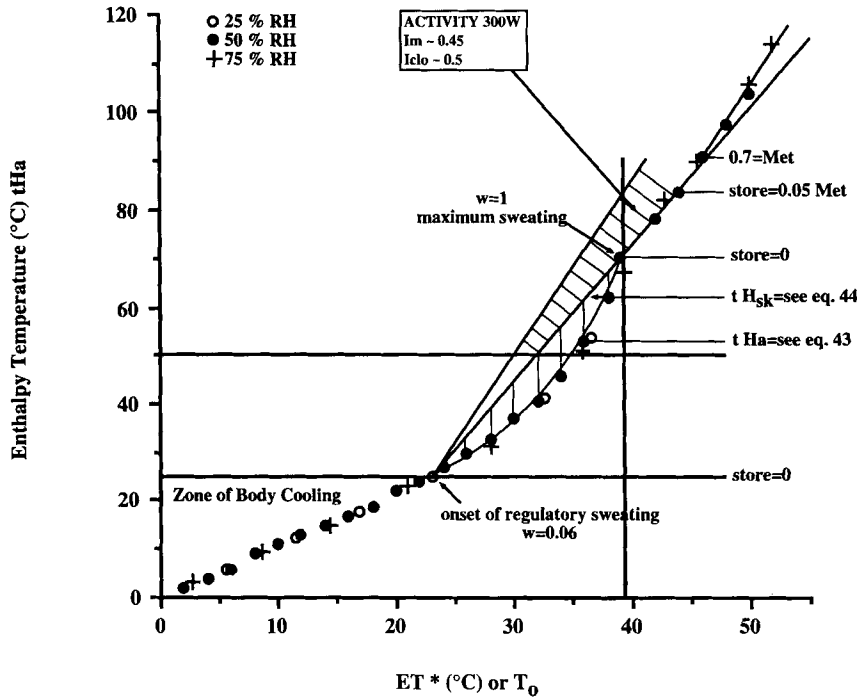
FIG. 4.3. Enthalpy temperature (°C) as a function of effective temperature (ET*). Adapted from Fobelets and Gagge (44).

sweating mechanism (59, 67, 104); the least-well-modeled are those physiological systems with multiple interactions, such as the peripheral and central cardiovascular and endocrine systems (157).

The original Pierce two-node model (51) was set up along parallel concepts developed to follow existing models of the core:shell separation concept formulated by Aschoff and Wever (2) and the basic equations developed by Stolwijk and Hardy (145).

The Passive State

For a standard-sized individual, weight ($\dot{m}_b$) of 70 kg and 1.8 square meter DuBois surface area, the following variables were derived:

Skin mass $= \alpha(\dot{m}_b)$, kg
Core mass $= (1 - \alpha) \cdot (\dot{m}_{b,o})$, kg
Latent heat of sweat, λ $= 0.68 \ W \cdot h \cdot g^{-1}$
Minimal skin conductance $= 5.28 \ W \cdot m^{-2} \cdot °C^{-1}$
Neutral skin blood flow [$\dot{V}_{skbf,o}$] $= 6.3\ell \cdot m^{-2} \cdot h^{-1}$
Thermal capacity of the body $= 0.965(W \cdot h) \cdot kg^{-1} \cdot °C^{-1}$
Thermal capacity of the blood $= 1.163(W \cdot h) \cdot \ell^{-1} \cdot °C^{-1}$

At any given time, t (hour) heat balance for the skin and core nodes (W · m^{-2}) is:

$$H_{sk} = (5.28 + 1.163\dot{V}_{skbf}) \cdot (T_{cr} - \overline{T}_{sk}) - E_{sk} - hF_{cl}(\overline{T}_{sk} - T_o) \quad \text{(Eq. 4.45)}$$

$$H_{cor} = M_{net} - (5.28 + 1.163\dot{V}_{skbf}) \cdot (T_{cr} - \overline{T}_{sk}) \quad \text{(Eq. 4.46)}$$

In the above equations, rate of heat storage $S = H_{sk} + H_{cor}$ and T_{cr} refers to core temperature (blood) best described by esophageal temperature.

The thermal capacity of skin shell and core nodes (W · h · °C) may be expressed by:

$$J_{sk} = \alpha(0.965) \cdot (\dot{m}_b) \quad \text{(Eq. 4.47)}$$

and

$$J_{cr} = (1 - \alpha)(0.965) \cdot (\dot{m}_b) \quad \text{(Eq. 4.48)}$$

The two-node model predicts changes in mean skin temperature ($\Delta\overline{T}_{cr}$) per unit time ($\Delta t = \frac{1}{60}$ h or 1 min) for a given body surface area (A_D) as:

$$\Delta\overline{T}_{sk}/\Delta t = (A_D \cdot H_{sk}/J_{sk}) \quad \text{(Eq. 4.49)}$$
$$\Delta\overline{T}_{cr}/\Delta t = (A_D \cdot H_{cor}/J_{cr}) \quad \text{(Eq. 4.50)}$$

Iterations may be formulated until the end of each succeeding minute of exposure at a given environment set by $t = t + \Delta t$, $\overline{T}_{sk} = \overline{T}_{sk} + \Delta\overline{T}_{sk}$, and $\overline{T}_{cr} = \overline{T}_{cr} + \Delta\overline{T}_{cr}$. When rapid changes in $\Delta\overline{T}_{sk} > 0.1$, the increment in time (Δt) can be shortened to $\frac{1}{600}$ for optimum integration.

The Control System

Definitions of the thermoreceptor information coming from the deep core or skin surfaces, affecting appropriate threshold and slope, can be found from appropriate data in the literature for various acclimation states, fitness, age, gender, etc. (55, 79, 106, 116, 124, 145, 146).

Initial Warm and Cold Signals

From the skin:	From the core:	Reference
$(Signal)_{sk}$	$(Signal)_{cor}$	
$= (T_{sk} - 34)$	$= (T_{cr} - 36.8)$	(104)
Warm signal		
$= (Signal)_{sk+}$	$+ (Signal)_{cor+}$	(145)
Cold signal		
$= (Signal)_{sk-}$	$+ (Signal)_{cor-}$	(145)

Control of Skin Blood Flow

The rate of thermoregulatory skin blood flow ($\dot{V}_{skbf}$) is primarily patterned as a linear summative function of hypothalamic temperature (for example, blood directly bathing the preoptic area neurons) and mean skin temperature but is also adjusted by local skin temperature ($\overline{T}_{sk}$), posture, and exercise intensity and is affected transiently by the beginning and end periods of exercise (71, 81, 82, 125). The proportional control coefficients (59) initially used in the original two-node model for ascertaining control of $\dot{V}_{skbf}$ were based primarily on rational equations developed by Stolwijk and Hardy (145).

Definition of skin blood flow (in liters $\cdot$ m^{-2} $\cdot$ h^{-1}):

$(Stric)$ $= *0.5(Signal)_{sk-}$ "Vasoconstriction"
$(Dilat)$ $= *150(Signal)_{cor+}$ "Vasodilation"

$\dot{V}_{skbf} = [*6.3 + (Dilat)]/[1 + (Strict)]$

(Eq. 4.60)

The coefficients with asterisks were initially determined from studies based on rectal temperature as the index of core temperatures (51, 145). As better data appear in the literature describing various thermal responses, the model input format is flexible enough so that the default physiological control coefficients may be easily altered. An estimate of the default proportional control coefficient for maximal vasodilation (150 ℓ $\cdot$ m^{-2} $\cdot$ h^{-1}), for example, corresponds roughly to 4.5–5 liters/min for trained individuals assuming complete thermal exchange or conductance of the whole skin surface area (A_D) of from 1.8 to 2.0 m^2 [81]. Other empirical predictive coefficients have been formulated (81) based on measurements of skin blood flow by venous occlusion plethysmography (VOP) during upright exercise coupled with mild heat stress. Some of these coefficients

also incorporate effects of activity level inferred from many other studies (103, 106, 125).

Kraning (81) developed a paradigm, with skin blood flow appearing as a linear approximation of central blood temperature (T_{bl}) (for example, blood bathing the central preoptic neurons) estimated by esophageal temperature (T_{es}). In recent studies (71, 123) both $\overline{T}_{sk}$ and exercise were shown to effectively modulate the relationship of the skin blood flow:T_{es} intercept on the T_{bl} axis. Increasing $\overline{T}_{sk}$ reduces the value of T_{bl} necessary to produce a given value of skin blood flow, while exercise augments the temperature displacement drive of T_{bl} required to produce a given level of skin blood flow.

New research shows that exercise creates graded reductions in maximal skin blood flow (evaluated by VOP) from 7.0 liters $\cdot$ min^{-1} at rest to 5.0 liters $\cdot$ min^{-1} at a $\dot{V}_{O_2}$ of 2.0 liters $\cdot$ min^{-1} (71). A recent report sets minimum skin blood flow (+ nutritive blood flow) during intense vasoconstriction at about 0.330 liters $\cdot$ min^{-1} (81). The assumption is that all inactive activity is in the upright seated position and that all exercise exertion is in the upright standing position.

Control of the Whole-Body Sweating Drive

Whole-body sweating drive, $\dot{M}_{sw}$, in g $\cdot$ m^{-2} $\cdot$ h^{-1}, as used in the Pierce two-node model, incorporates the summed effects of an integrated mean body temperature ($\overline{T}_b$) from a threshold ($\overline{T}_{bo'}$) discussed previously (Eqs. 4.6 and 4.6') in which ($\overline{T}_{bo'}$) is derived via partitional calorimetry using initial weighting factors for skin temperature thresholds (33.5°C) and core temperature thresholds (37°C) in which:

$$\overline{T}_{bo'} = \alpha \ (33.5) + (1 - \alpha)(37.0) \quad \text{(Eq. 4.70)}$$

and

$$\overline{T}_b = \alpha \ \overline{T}_{sk} + (1 - \alpha)\overline{T}_{cr} \quad \text{(Eq. 4.70')}$$

Thus $\dot{M}_{sw} = A'[\overline{T}_b - \overline{T}_{bo'}]$, where A' comprises the whole-body sweating proportional control coefficient (g $\cdot$ m^{-2} $\cdot$ h^{-1} $\cdot$ °C^{-1}) (59) that may be adjusted in the program ranging from initial values of 186–195 g $\cdot$ m^{-2} $\cdot$ h^{-1} $\cdot$ °C^{-1} for untrained persons to as high as 250–290 g $\cdot$ m^{-2} $\cdot$ h^{-1} $\cdot$ °C^{-1} for elite, acclimated athletes (Gonzalez and Gagge, unpublished observations). When $\overline{T}_b <$ $\overline{T}_{bo'}$ the predicted drive for thermoregulatory sweating is zero and the default evaporative heat loss (E_{sk}) assumes the value for skin diffusion only in which E_{sk} $= 0.06 \cdot E_{max}$. When the skin surface is fully wet by thermoregulatory sweat secretion described by $w_{rsw} = 1$, skin wettedness is also constrained to 100% since the excess (E_{drip}) is of no benefit for evaporative cooling. The E_{sk} is based on $\dot{M}_{sw}$ and there is no skin diffusion.

FIG. 4.4. The two node (core and shell) model of thermoregulation. Adapted from Gagge et al. (51).

Control of the Skin Shell Properties

Vasoconstrictor activity increases the thickness of the skin shell node that is composed of skin, fat, and, to a degree, underlying muscle layers (particularly during cold acclimation). When skin blood flow ($\dot{V}_{skbf,o}$) is less than a minimal value (6.3 $\ell \cdot m^{-2} \cdot h^{-1}$), α is increased by the function:

$$\alpha = 0.1 + {}^{*}0.25(\dot{V}_{skbf,o} - \dot{V}_{skbf})/\dot{V}_{skbf,o} \qquad (\text{Eq. } 4.71)$$

The controller coefficient ($^{*}0.25$) may also be arbitrarily set (49).

Control of Shivering

The two-node model also incorporates a multiplicative function describing increases in metabolic heat production (W $\cdot$ m^{-2}) due to shivering (in sea-level ambients) as the product of cold signals from the core and the shell in which:

$$M = 58.2 + {}^{*}\beta[(\text{Signal})_{sk-}] \cdot [(Signal)_{cor-}] \qquad (\text{Eq. } 4.72)$$

The default controller coefficient $^{*}\beta$ (initially set at 19.4 W $\cdot$ m^{-2} $\cdot$ $^{\circ}$C^{-1}) is from Stolwijk and Hardy (145). Addi-

tional control coefficients and algorithms have been modified for cold water immersion by others (146). Shivering thermogenesis has been quantified for a wide range of air environments as well (147, 157). Addition of percent body fat into the control equations has contributed to the overall predictive capabilities for immersin in cold water (147). Figure 4.4 gives a schematic of the model.

HEAT LOSS FACTORS IN WARM AND COLD ENVIRONMENTS: PHYSIOLOGY

The previous section established that responses effective in heat exchange involve multiple processes and interactions of an organism's thermoregulatory system with its surroundings. These incorporate adaptations of the passive system as well as active controllers of the total system. Heat exchange is also facilitated by discrete modifications in the structural properties of all organisms. Attention was directed to thermal exchange in clothed persons. This section considers those heat loss mechanisms in homeotherms (principally in humans but also in other animals) that are activated in response to changes in internal and peripheral body temperatures as

a consequence of endothermic thermal load or as a result of exposure to various environments. We emphasized earlier that the skin is the final barrier influencing the physical processes of energy exchange between the body and the ambient. The skin is the primary interfacial node (82) for heat exchange by radiation and convection, conduction, and evaporation, or as Porter and Gates (117) aptly stated, the skin is "a transducer of the environment." Human skin (and the pelt in other animals) is also the integral node which is adapted exclusively for such immediate interaction; clothing, alternatively, is a passive means in the regulation of energy processes between the body and the environment. However, the skin does not function equivalently throughout a variety of species (97, 99, 134).

The processes considered in response to a warm or cold sea-level environment include alterations in the internal body and surface temperature; heat flow and distribution, which affects the boundary layer of the surface; and the thermal resistance of the skin surface, which acts in series with each axial and radial layer (62, 63, 114, 159).

Each organism is potentially in a physical or functional state that incorporates passive and active heat exchange. This exchange includes responses that are in quasi-steady-state (that is, nonvarying with time) or which vary from instant to instant (transient) as a response to a forcing (driving) factor (123). It is apparent also that the classical definition of homeostasis should be modified, as proposed by Yates (160):

> We should define a homeostatic performance in terms of two aspects of time-series data on a physiological state variable. The first aspect is the epoch (period) over which a mean state stability can be detected. The second aspect concerns the magnitude and structure of the variance around the mean state.

Further, with regard to the stability observed in a general physiological response, homeostatic stability is considered as "clearly a dynamic and not a static stability. It is also clearly not a linear stability: mammals are not at equilibrium rest points, nor are they on trajectories toward such rest points" (160).

It is apparent that the consolidation of multiple physiological variables, thermal receptors, and a mixture of redundant feedback information (19, 132, 140, 141) is necessary for a complete quantitative description of a homeothermic response (67, 112).

Heat and Mass Transfer from the Body to the Environment

Organisms continuously lose water by diffusion through the skin (E_{dif}) into the air (21, 47, 54, 67, 97, 134). In mass diffusion in a binary mixture, such as air-

water, the temperature gradient does not produce excessive thermal diffusive flow. Characteristic first principle analogs are used to model biophysical and physiological properties (90, 97, 114).

Mass flux, characterized in a three-dimensional plane, is a vector which incorporates three components; *momentum flux* acts on a shape or element of a fluid (such as viscous forces) in multiple components, characterized by discrete tensor properties. In general, a given flux (the time rate of flow per unit area) of either momentum, energy, or mass is directly associated with, and opposes, the direction of a gradient (rate of change per unit length) of its potential force (velocity, temperature, or concentration, respectively). Typically,

$$Flux = -(Specific\ coefficient) \cdot (Gradient)$$
(Eq. 4.73)

Previously, Fourier's law of heat conduction was expressed by heat flow in one direction (that is, the y direction):

$$q_y = -k \cdot \delta T/\delta y \qquad \text{(Eq. 4.74)}$$

where q_y is heat flux in the y plane equivalent to Q/A_s, the rate of heat flow per overall unit area A_s, the distinction being that the surface area is considered so large that a minimal temperature gradient, if any, occurs in the x or z planes. $\delta T/\delta y$ is the temperature gradient of the driving potential energy flow (that is, from a higher locus to a lower one) in the y direction. The negative sign is a general format signifying that heat flow is in the direction of decreasing temperature.

For most physiological systems, heat flow occurs in all planes of a volume so that a partial derivative of thermal gradient must be considered as:

$$q_y = -k \cdot \partial T/\partial y \text{ as well as } q_x =$$
$$-k \cdot \partial T/\partial x \text{ and } q_z = -k \cdot \partial T/\partial z$$

where k, the thermal conductivity coefficient, may or may not be equivalent in each plane and temperature is expressed as the temperature flowing along a specific plane.

Net flux is a vectorial heat flux expressed generally in most biological systems as:

$$q = k \nabla \cdot T \qquad \text{(Eq. 4.75)}$$

where $\nabla \cdot T$ is the net temperature gradient. Mass transport by diffusion is the common mode most analogous to heat conduction and momentum transfer (98, 99).

Convective mass transfer, in which a coupling diffusion to fluid motion is brought about by external forces, is a common phenomenon in physiological systems. Simple examples include active transport and facilitated transport. Facilitated transport can occur, for example, when a potentially noxious chemical enters the skin,

whereby diffusion is aided by a carrier (134). Active transport occurs when transport is counter to the concentration gradient, and the energy required for this is provided by specific biochemical reactions (29, 33, 157).

For a great number of situations, diffusion is a passive property appearing over wide types and sizes of molecular processes and diffusing media. Diffusion can result from a change in the concentration and pressure gradient and from thermal and electrochemical effects. Ordinary diffusion is generally considered analogous to energy and momentum transport. In this form, the concentration gradient has similar effects to that of heat conduction, in which a scaler component of ρ_x replaces T, where ρ_x is the mass concentration of a diffusing component, i. The analogous counterpart to energy flux governing the gradient and diffusion flux is shown in Fick's first law of diffusion:

$$j_{i,y} = -D_{i,o}\,\delta\rho_i/\delta y \qquad \text{(Eq. 4.76)}$$

where $j_{i,y}$ is the mass flux of species i in a y direction and $D_{i,o}$ is the diffusivity constant of the mixture of species i,o.

The format of the equation is equivalent for molar, mass fluxes, and diffusional concentrations. As in the equation for energy transport, if ρ_i changes in the x and z planes simultaneously, Fick's law shows that for all directions y, x, and z

$$j_{i,y} = -D_{i,o}\,\partial\rho_i/\partial y \qquad \text{(Eq. 4.76')}$$
$$j_{i,x} = -D_{i,o}\,\partial\rho_i/\partial x \qquad \text{(Eq. 4.76'')}$$
$$j_{i,z} = -D_{i,o}\,\partial\rho_i/\partial z \qquad \text{(Eq. 4.76''')}$$

As in the energy flux equation, all diffusion fluxes may be considered as net vector in a mass flux equation:

$$J_{i,o} = -D_{i,o}\,\nabla\rho_i \qquad \text{(Eq. 4.77)}$$

where $\nabla\rho_i$ is the net mass concentration gradient.

$D_{i,o}$ is independent of $\nabla\rho_i$, but it varies as a function of temperature and barometric pressure. The product of $J_{i,o}$ and the latent heat of vaporization (670 W · h · kg⁻¹) determine the effective flux density of latent heat (J_v) in W · m⁻² (99, 139). An analogous application for sensible heat flux has been derived from J_v that is useful for describing thermal resistance (r_T) through the pelt and fur of animals (31, 32).

Often $\nabla\rho_i$ in Eq. 4.77 is expressed in the equivalent form, C_v, in the ideal gas law (97) as:

$$C_v = [h_D \cdot \lambda_s/(R_w \cdot T)] \cdot \Delta P^* \ (\text{kg} \cdot \text{m}^{-3}) \qquad \text{(Eq. 4.78)}$$

where R_w is the gas constant for water vapor, 3.46 Torr · m³/(kg · °K) and $T = [(Tsk + Ta)/2 + 273.15]$ in °K. The term ΔP^* expresses the partial pressure gradient in the gaseous surface system from a high concentration ($P_{s,sk}$) to a lower vapor pressure (P_a), as discussed in earlier sections on Evaporative Heat Exchange. The quantity $[h_D \cdot \lambda_s/(R_w \cdot T)]$ is the effective evaporative heat transfer coefficient (h_e, in W · m⁻² · Torr⁻¹).

As pointed out before, heat and water vapor conservation equations (that is, continuity) are uniquely tied together (94, 97, 99, 114, 137). However, for practical purposes, the boundary condition for highest water vapor concentration is conveniently assumed as that saturated vapor existing at the skin surface ($P_{s,sk}$). Also, the specific heat of air is a function of the vapor concentration, which is itself affected by the skin–air temperature (159).

Rationally derived heat and mass transfer equations extended for human and animal shapes (29, 61, 100, 102, 107, 121) have been formulated. For many animal surfaces, h_c derived from various shape factors related to air movement cannot be wholly quantified (100, 102), but several equations have been rationally derived. Mitchell's equation (102) appears most accurate when used in small animals and when the animal's shape is considered as a sphere of equivalent mass:

$$h_c = 6.77V^{0.6} \cdot d^{-0.4} \qquad [\text{W} \cdot \text{m}^{-2} \cdot \text{K}^{-1}]$$
$$d = (\text{volume})^{1/3}$$
$$\text{(Eq. 4.79)}$$

where d is the typical dimension of the animal studied (in m) and V is external wind velocity in m · s⁻¹. Equation 4.79 has been shown to predict h_c within 5%–15% of animals tested in the field (112), errors in the h_c becoming more apparent in large animals with longitudinal shape. Currently, there remains sparse information on direct experimental evaluation of convective heat properties in animal species other than that derived from calorimetric experiments or rational equations of heated models. Measurements of free convection are difficult to quantify, particularly in small mammals. This is because, whereas the rate of convective heat transfer varies as $h_c \cdot \delta T$, where $\delta T \approx (T_{sk} - T_a)$, h_c is never wholly independent (and varies as a function) of δT (100).

Body Motion

Body motion alters effective heat exchange by convection both inside and outside the skin surface boundary layer (22, 64, 93, 97). Thus the rates of convective and evaporative heat loss are both augmented. In humans, body motion also affects heat exchange within clothing (22). Belding (5, 6, 9, 10) initially showed that a 50% reduction occurred in the intrinsic clo value of an Arctic ensemble primarily by the "stirring" and exchange of air within and below the clothing by body movement. A "pumping coefficient" owing to body motion appears more prevalent at external wind speeds of ≤2–2.5 m ·

s^{-1} (defined as a critical wind speed zone) but becomes minimal as a prominent exchange factor with wind speeds >2.5 m $\cdot$ s^{-1} (Breckenridge, personal communication). There is a reduction in both the intrinsic and the effective thermal resistances (64) with body motion in the critical wind speed zone.

In cold environments, with wind speeds greater than 3 m $\cdot$ s^{-1}, the effect of wind penetration on a clothing system far outweighs the balance of internal air circulation by body movement alone. Latent heat cannot be dissipated, and in very cold climates, condensation of moisture occurs (22). In humans, heavily clothed in cold environments, the only remedial option for dispelling metabolic heat is by selective openings in the garment (93). In certain Arctic animals (Arctic fox and polar bear) the hairs of the fur are adapted to maximally withstand extremes of cold and wind (67, 98).

Homeothermy. Heat loss from the skin to the environment is often described by Newton's law of cooling (67, 76). The rate of temperature loss based on Newton's law varies in proportion to the temperature difference between the cooling body and the environment (76, 114). Kleiber (76) suggested, however, that if firm definition of a homeotherm's mechanisms were strictly upheld, there should be no cooling, since body temperature remains nearly constant (76) and Fourier's law of heat conduction (Eq. 4.75) would predominate. The association between the two laws is unified adequately by incorporation of the heat capacity of the body [c_b = 0.965 W $\cdot$ h/(K $\cdot$ kg) and mass, m_b (kg)] (76,135).

The prominent feature that differentiates homeotherms from poikilotherms, relative to temperature regulation, is that their core temperature is not altered appreciably and specialized fuses, developed through phylogeny, are available to regulate energy metabolism and to dispel or conserve heat. As long as the body remains competent, the energy expense to maintain internal body temperature in homeotherms is economical and lasts well into senescence. Unfortunately, this independence from the environment also places limits on a homeotherm's general adaptation by requiring a firm feedback control system mediated by specialized regulatory centers to maintain biological stabilization. Part of this is the necessity of a continual food requirement to maintain thermal stability. Prosser (118) pointed out that maintenance of dynamic equilibrium, or steady-state, prescribes the rhythm to homeostasis, which he defined as "ability to survive in a varying environment—the central property of all living organisms." A key point, highlighted by the studies of Adolph (cited in cf. 67) and many others (118, 135), is that homeotherms are differentiated by structure and function from poikilotherms, even to the elemental levels of tissue metabolism and enzymatic processes. One of the most

significant characteristics is the ability to maintain high levels of aerobic power and the consequences of a high $\dot{V}_{O_2max}$ prevalent in homeotherms, as documented by Bennett and Ruben (11) and shown for a variety of vertebrates in Figure 4.5.

According to Bennett and Ruben, the prospects of a high maximal aerobic capacity are not possible in vertebrates with low body temperature and necessitate increases in tissue temperature to optimal levels. Additionally, higher aerobic capacity is associated with the presence of greater concentrations of muscle mitochondria in endotherms (11).

For the most part, the resting internal body temperature of homeotherms falls within a range of temperatures of $\pm 0.5°C$ from a uniform level of $36°-39°C$ primarily because heat exchange and heat conservation mechanisms are facilitated readily (28, 67). Some homeothermic animals, nevertheless, can dwell in the hyperthermic and hypothermic range for extended periods of time (67, 135). Rate of heat storage generally varies within $\pm 10\%$ of M without altering the heat balance of an organism. To an extent, the rise in body temperature occurs by obligatory displacements of average tissue temperature in large body parts, such as legs and arms (28, 29, 97, 135). Nevertheless, accounts (59, 87) show that some individuals (Central Australian aborigines, for example) lower their core temperature and allow their extremities to cool drastically compared to the trunk when encountering cold sleeping conditions.

In many other mammals (camel, Cape hunting dog) elevations of core temperature at the expense of hypohydration by as much as as 1.5°C above normal core temperature are tolerated with little effect. Heat storage is made up soon after a hunt, for example, in the hunting dog, or the water deficit is made up later in the night in the camel (67, 135). Additionally, the hunting dog spares water by panting less than a normal dog (135).

In poikilotherms, body temperature ($T_b \approx$ core and shell) is affected by the thermal environment exclusively and always responds to changes inherent in the integrated ambient ($T_b = T_o$, the operative temperature). The dependent variable is the internal body temperature and the independent variable is the operative temperature; the latter coincides with magnitude of passive heat exchange, M $-$ [R $+$ C], possible by the organism. There is no proportional control system or "gain" per se in the T_b responses when plotted as a function of T_o apparent in the poikilotherm. For example, a random distribution of environmental temperatures depending on different times of the day or other factors (clouds, etc.) can potentially alter the T_b level in a poikilotherm if behavioral regulation were blocked (65). Nevertheless, internal body temperatures (hypothalamic, subcortical, and extrahypothalamic) in homeotherms are kept constant within a finite range by specific regulatory
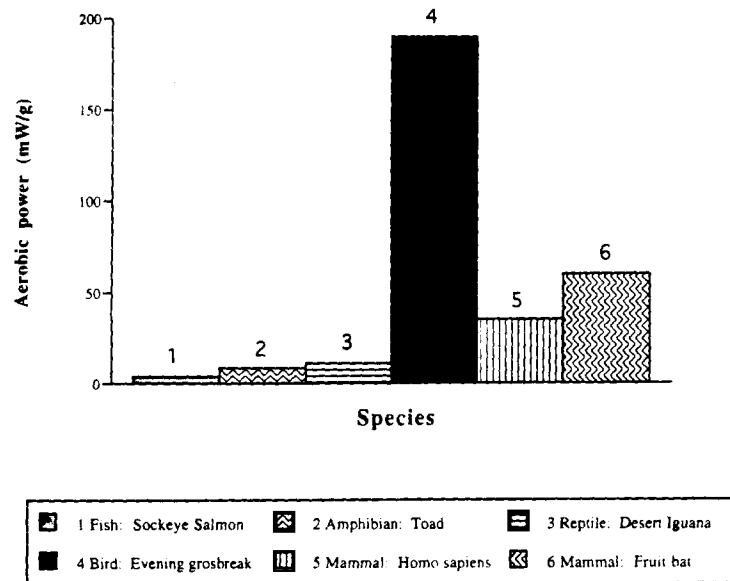
FIG. 4.5. Maximal aerobic power plotted in various vertebrate species of endotherm and poikilotherm. Adapted from Bennett and Ruben (11) and Gonzalez (52). (1) ☐, Fish (sockeye salmon); (2) ▨, amphibian (toad); (3) ▤, reptile (desert iguana); (4) ■, bird (evening grosbeak); (5) ▥, mammal (human); (6) ▧, mammal (fruit bat).

mechanisms augmented as a function of deviation of a reference temperature (59, 67). Temperature sensors feeding information to such central controllers have been considered as analogous to a constant pressure device which monitors a specific water level at the outlet of a tank, consistently changing the level by altering supply and extent of loss of the fluid (97). Some elements of the thermal response activated by changing peripheral and brain temperatures have been shown to occur in various species of poikilotherm (37); however, the key property attributed to homeothermy is direct linkage with the activation of heat loss and heat conservation mechanisms in response to changes in internal core temperature. If a homeotherm maintained its normal heat flux properties at the surface of the skin without active production of heat, other than by normal biochemical processes described by a Q_{10} of 2–3, then the core and skin temperatures would eventually equilibrate with the ambient and $T_b \approx T_o$ and the organism would become effectively poikilothermic. There is evidence that during regional anesthesia (by blockade of nerve fibers that transmit both cold and warm signals to the hypothalamus) such a poikilothermic state exists in patients with concomitant redistribution of heat flow from the core to skin sites causing hypothermia (67, 69).

A general plot of the avenues of heat exchange and metabolic heat production as a function of ambient or operative temperature often serves to define the envi-

ronmental limits of a species (95), as shown in Figure 4.6.

Figure 4.6 depicts schematically a generalized thermoneutral zone, previously described for humans, in which metabolism is at a minimum. In this zone, no excessive physiological effort is necessary by sweating, vasoconstriction, or vasodilation. For other homeotherms, such thermoneutral zones vary from species to
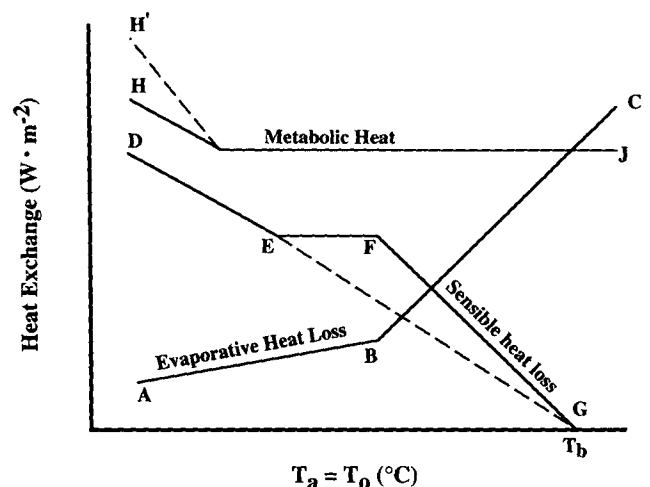

FIG. 4.6. Heat exchange plotted as a function of operative temperature. Adapted from McLean (95).

species in response to changes in peripheral, hypothalamic, and extrahypothalamic temperature inputs and according to morphology and fur thickness (19, 59, 67, 95, 97, 132, 141). Heat exchange mechanisms are also modulated by differences in integrity and function of afferent and efferent information arriving at the central nervous system (CNS) of a given species, and adaptation to environmental stress plays a critical role in any description. As shown in Figure 4.6 (95), applied to unclothed humans, for example, this zone is relatively small; in clothed humans or a wide variety of homeothermic animals, the zone increases by a wider span, which is also governed by size and the insulative value of the clothing or pelt.

Thermal Aspects

Structural Modifications for Optimal Heat Loss. Typically, the larger the homeotherm, the greater is the metabolic energy per surface area and the thickness of insulation (6, 27–29, 63). All homeotherms must balance any heat gain through metabolism by a subsequent heat loss through skin and respiratory passages. Since a greater proportion of heat loss is through skin, the surface area of the skin and the body size relate closely to the dissipation of heat. Two rough axioms have been used to describe the association between body size and morphology with environmental temperature (87). Bergman's rule suggests that when comparing two bodies having similar shape, the larger body will always have a smaller surface area per unit mass. Allen's rule suggests that short extremities augment the ratio of mass to surface area. Thus applying both rules to animals of cold climates, compared to similar species in warmer climates, would show that cold-adapted animals tend to be large and have relatively short limbs or appendages. Both rules have been disputed, however, in regard to human heat loss responses. For example, Eskimos are small and stocky but have short limbs and some natives of East Africa are exceptionally tall and slim and have very large extremities (87).

Radial and axial temperature gradients. The characteristic zone of thermoneutrality varies widely from species to species. In all homeotherms, resting in their characteristic zone of thermoneutrality, the surface temperatures of the body have a lower overall average than the internal temperature in accordance with heat transfer laws (27, 62, 67, 137), as illustrated in Figure 4.7. Additionally, temperatures are lower along the longitudinal axes of the extremities, and this effect results in a definitive radial and axial gradient that dominates heat flux at these sites (27, 157).

Although all tissues of the body metabolize, thereby producing variable heat flow, some sites produce heat at a much higher rate than others. For example, as
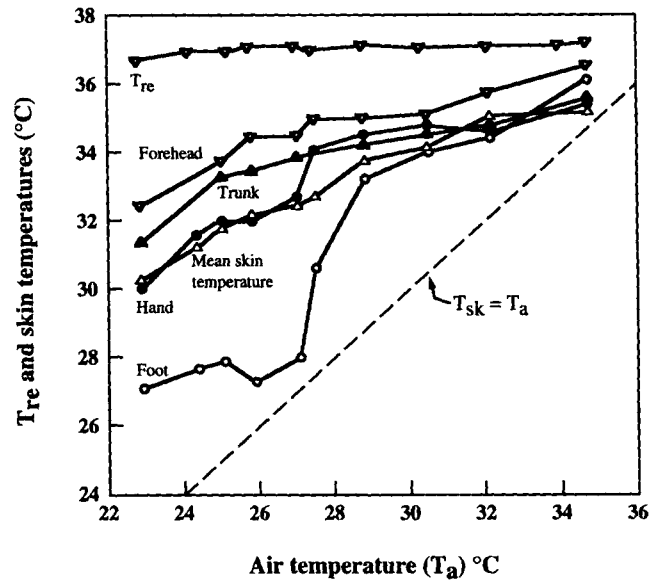

FIG. 4.7. Rectal (T_{re}) and regional skin (T_{sk}) temperatures in humans exposed to various ambient temperatures. Adapted from Hardy and DuBois (62).

shown in Figure 4.8, in many animals, other specialized structures (rabbit ear, bat wing, and dog tongue) are modified exclusively for heat loss above that limited by surface to volume ratio or size (56, 67, 77, 97, 135).

Heat production is a function of the blood flow and oxygen extraction of a tissue and its mass. Areas such as the brain necessitate high-maintenance energy cost and heat production, but mass varies little. Skeletal muscle, however, may increase dramatically to offer a major source of heat. Organs in the thorax (liver and lungs) and other viscera may alter their mass, but they do not offer equivalent heat compared to mass as does skeletal muscle. Generally, increases in heat production are principally considered a function of the intensity and relative increases in muscular activity. Effective heat
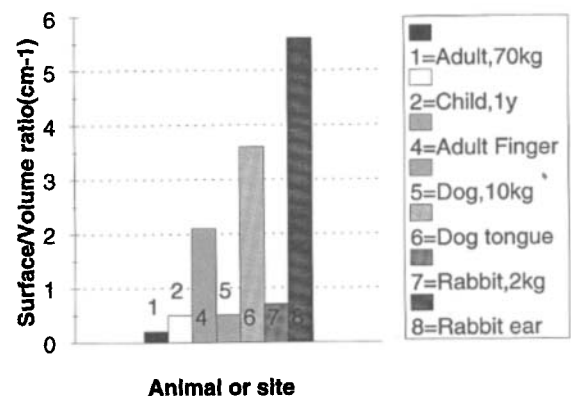

FIG. 4.8. Surface to volume ratios appearing among various animal forms and sites. Adapted from Hensel et al. (67).

flux is also altered by changes in the local heat production of various organs, inconsistency in geometric forms (not all conform to human shape), difference and types of insulation and skin evaporation, and variability in convective heat flow through blood. The generalization applied to modeling has been made specifically for humans by the core and shell concept (2, 29) seen in Figure 4.9.

Aschoff and Wever (2) first described this concept very loosely to illustrate the relative gradient of heat transfer in humans. The core is the section of the body least variable in temperature, comprising the brain, parts of the skeletal muscles, and viscera. The shell is a variable section (including skin, pelt, and, to an extent, fat layers) whose temperature responds tightly with alterations in the ambient temperature. In sea-level environments, during wide excursions of ambient dry-bulb temperature, the shell may comprise as much as 10%–20% of the human body (28, 29, 49), but during cooling the shell may comprise as much as 30%–40% of the total body. The core–shell concept is not wholly applicable for many other homeotherms.

Extremity protection is a vital determinant in adaptation to cold. During extreme conditions hypothermia can occur, and the body's core temperature often becomes lowered excessively to the point of death. Structural changes, resistance value of the pelt in various animal species, and clothing in humans must prevent excessive heat loss from the body as a whole. One of the typical biophysical explanations credited for the susceptibility of extremities to cold environments is their shape, which resembles slender cylinders that act

like heat transfer fins. The consequence is augmented heat loss to the environment, with the ensuing accelerated drop in peripheral temperature. This physical property of an extremity is not specific to cold exposures. Its effects, however, are much more pronounced under such conditions, mainly due to the increased temperature gradient from the respective surface site to the ambient. In the hot tropic environments in which a large prehistoric dinosaur (Stegosaurus) likely roamed, there is some evidence that such analogous heat transfer plates facilitated enhanced heat loss, but the plates became ineffectual for receiving radiant energy (42).

An additional physical limitation affecting heat transfer relates to the thickness of thermal insulation which may be applied on cylindrical surfaces. Since the surface of a cylinder is curved, each layer of applied insulation would be of a larger diameter and hence of an increased surface area. Thus there is a critical thickness beyond which heat loss to the environment would be increased, rather than decreased. This limitation does not apply to flat surfaces due to the absence of curvature (32, 108, 138, 148).

Heat flux through a cold skin must pass variable insulation barriers that are often higher than the fixed resistance typically seen at full dilation (1–1.5 cm of fatty tissue) (27–29, 67, 97, 108). Variations in cutaneous blood flow distribution thereby allow the skin to alter its resistance at various sites to attenuate thermal gains derived from metabolic heat production as well as from the environment. In the cold, the outer layers of the body offer a fixed resistance through fat, muscle layer, and skin thickness (2, 5, 27, 29), but internal body heat alone is not able to maintain the proper heat balance. Appropriate clothing in humans and pelt and feathers in birds and mammals (3, 32, 97, 151, 152) are added factors that allow adjustments to enhance structural skin resistance. They become vital factors for the prevention of heat loss in the cold, for maintaining heat balance, and for health and survival (53). One mechanism the body uses in circumstances where outside weather conditions are too cold for a shivering mechanism to act adequately (or the fur, feathers, and clothing are insufficient for maintaining heat balance for the total body) is to sequester the warm blood in toward the vital organs and, in effect, limit the body heat flow to such regions as fingers, lower extremities, and ears. Various animal species have adapted, through phylogeny, specialized structural mechanisms for enhancing heat exchange by which arterial blood is precooled and excessive extremity heat loss is prevented. One of these mechanisms is illustrated in Figure 4.10 (67).

Since the pelt in animals and clothing in humans effectively adds another layer to the skin, in warm environments they both generally impose a barrier to heat transfer by all heat transfer avenues: radiation, convec-

37°C
36°C
32°C
28°C
34°C
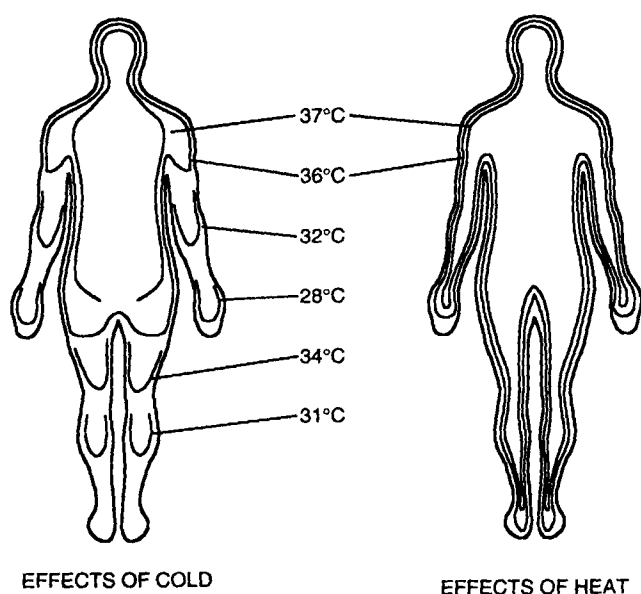31°C

EFFECTS OF COLD   EFFECTS OF HEAT

FIG. 4.9. Core and shell concept of Aschoff and Wever (2). Adapted from Carlson and Hsieh (29).
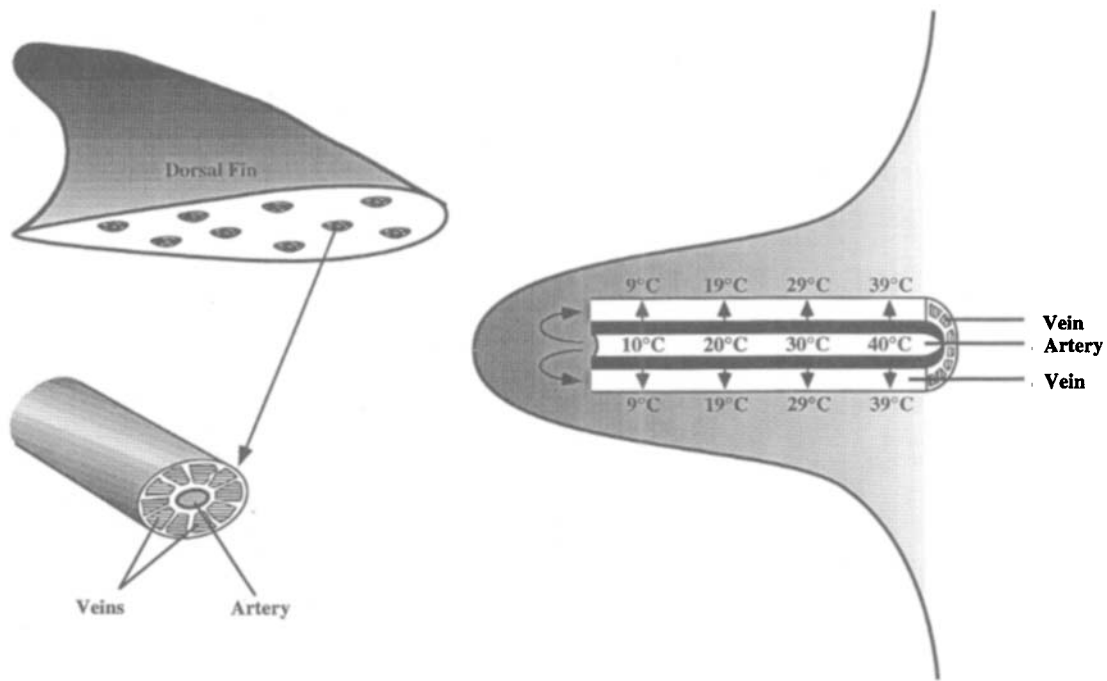
FIG. 4.10. A typical example of a countercurrent heat exchanger mechanism present in the dorsal fin of the porpoise. Redrawn from Hensel et al. (67).

tion and conduction, and evaporation (8, 29, 47, 49). Interference with air motion across the skin decreases transfer by convection and the consequent potential to lose heat by evaporation. The decrease in the boundary layer immediately surrounding the shell becomes an impediment to heat loss when air temperature is lower than skin temperature but often can serve as a protective measure when air temperature is higher than skin temperature (7, 67, 98, 135).

The thermal resistance properties of clothing affect the evaporative potential primarily when the ambient dew point is high (6, 47), particularly during exercise in very humid zones (dew point >15°C) or if the clothing type is especially impermeable.

Characteristics of the Homeostatic Response.

In an interesting review, Henane (66) points out that heat acclimation at most causes a decrease of 1°C in internal body temperature, which represents about a 243 kJ change in heat content for a 70 kg person. Since light exercise produces 7,525 kJ and heavy exercise some 16,800 kJ, the overall percent change in body heat content is some 3% and 1.4%, respectively. Henane cites the results of one study (136) in which, over a 40 h period of 400 W metabolic intensity, 57,600 kJ were produced. Heat acclimation caused a decrease of only 208 kJ in the subjects. Relative efficiency of heat acclimation amounts to 208/57,600, or 0.36%, which appears insignificant as a biological process and would not seem to alter the homeo-

static response enough to defend humans against a harsh environment.

The explanation, of course, is that the thermoregulatory changes due to the 1°C change are enough to permit significant modifications in sweating and circulatory responses which control efficient heat exchange processes and intracellular compensatory mechanisms (not present in the unacclimated person) that allow adequate defense against hyperthermia. For example, Senay et al. (136) showed that following heat acclimation individuals are spared excessive decreases in blood volume during moderate exercise by the fact that they retain protein to expand the initial plasma volume. By having an elevated initial plasma volume, there is adequate cardiac filling pressure, despite an augmented filtration of plasma water from the vascular compartment. Also evident during heat acclimation is augmentation in sweat gland function (84, 131) and nonthermal effects related to water economy (103, 133).

Thermoregulatory Control Relative to Heat Loss Factors

Metabolic Consequences. The conversion of metabolic free energy into mechanical work varies in efficiency with type of muscle and speed of contraction. Efficiency is least at high and low speeds and is variable depending on type of terrain, load, and other factors (115, 157). Typical efficiencies range from 10% to about 20%. Most of the energy liberated is in the form of heat. This

heat is primarily dissipated at the skin surface, though some loss occurs via the lungs (49). The ability to dissipate the heat the body produces is proportional to the surface area (the Dubois surface area in humans) (63).

Central and Peripheral Nervous Systems. In certain species of homeotherm, magnitude of thermosensitivity (neuronal or whole-body response) is predominantly identified using alterations (heating and/or cooling) of hypothalamic and subcortical (spinal cord, abdomen, etc.) areas (19, 59, 70, 77, 141). The extent that an integrated thermoregulatory activity follows a hierarchy of responses has been formulated in many reviews (132, 140). Additionally, for a wide variety of endotherms it has been shown that peripheral thermal sensitivity decreases relative to central thermal sensitivity as body size enlarges (124).

In humans, measurement of the hypothalamic temperature is difficult, but there are abundant studies indicating that if the anterior hypothalamus is not the sole central integrating structure of temperature regulation, it is the main one and other subcortical structures amount to no more than relay stations or redundant integrators (59, 67). As important as the biophysical factors controlling heat exchange in most homeotherms are the physiological mechanisms active during changes in thermal load. Any characterization of heat loss properties should consider both. Important also is the association between changes in internal and peripheral temperatures (as afferent signals to the integrator) and the consequences of the change by appropriate efferent response (sweating rate, skin blood flow, or metabolic heat production, ΔM). One technique used universally to characterize such thermoregulatory responses is the application of estimates of open loop gain (OLG) (59). OLG quantifies the magnitude and degree of output called for by the change in input to a thermal controller. The output response, classified as the dependent variable (that is, sweating rate, panting, skin blood flow, or ΔM), is plotted as a function of the critical independent variable, internal or integrated mean body temperature, instead of time or exogenous load (ambient temperature) or endogenous load (exercise intensity). This approach assumes that the thermoregulatory control system responds exactly in a negative feedback manner, which has been debated by many (140). Nevertheless, for many effector variables operative in humans and other species (18, 19) such a conceptual model has validity in describing the thermoregulatory system.

One early characterization of the overall response of the heat exchange system using the OLG approach was by Benzinger and Kitzinger (12). Their results showed that the principal heat loss mechanisms operating against hyperthermia were strongly associated with deviations in tympanic temperature, alluding to changes

mediated solely by anterior hypothalamic temperature. Since the early 1960s, substantial evidence has verified, primarily in humans, that the stimulus for heat dissipation responses is a function (summative or multiplicative with local skin temperature) of temperature signals from both internal and peripheral thermoreceptors (19, 20, 59, 67, 85). These temperature signals are integrated primarily in the anterior/preoptic hypothalamus. One approach to ascertain the magnitude and degree of contribution of various thermal signals from the skin and internal body temperature (regardless of actual location such as rectal, muscle, esophageal, spinal, etc.) has been to derive an integrated mean body temperature (52).

Thermoregulatory Changes. In humans, since only 20% of the energy is utilized as work, 80% of the energy must be dissipated as heat. This internal thermal load, occurring as a result of physical exertion, therefore constitutes an important physiological challenge to the thermoregulatory system. Rates of heat production (by measuring $\dot{V}_{O2}$) can go as high as 20 times the basal level (1,100–1,200 W·m^{-2}). For continuous exercise, internal body temperature rises by about 0.14°C for each multiple above the resting state. To maintain thermal equilibrium, autonomic responses activating heat production and heat loss are called forth when the internal and skin temperatures are altered from one steady-state level to another. In the transient phase of exercise, the net metabolic heat flux must be dissipated to the environment or heat storage ensues. Rate of heat storage most often occurs when either the thermoregulatory response mechanisms are inefficient, as in untrained or unacclimated individuals, or thermal conduction through clothing materials is impeded (83). For an average person, some 280 to 335 kJ of total heat storage (equivalent to a 1°–1.4°C rise in core temperature) can be typically tolerated prior to collapse (49). During the first 20 min, rate of metabolism often far exceeds the heat dissipation required to maintain homeostasis. Heat balance is re-established mainly by matching heat loss by evaporation ($E + [E_{res} + C_{res}]$) with the endogenous and environmental heat load.

In humans, a number of thermoregulatory responses occur in parallel during exercise. During relative thermal equilibrium, extensive research shows that a person's steady-state core temperature is independent of air temperature (Nielsen effect) ≤35°C at low dew point temperature. The internal core temperature characterically reaches a new elevated level according to the intensity of exercise (% $\dot{V}_{O2max}$, relative aerobic power) or absolute quantity of energy metabolism ($M - Wk$). However, during exercise at ambient temperatures below 30°C, mean skin temperature is primarily a function of T_a and is, to a large extent, independent of the

level of work load (52, 103). Generally, only when the combined metabolic and external heat load exceeds the body's ability to dissipate heat by evaporation, caused either by an insufficiency of thermoregulatory sweating or a decrease in evaporative capacity of the environment (E_{max}), do skin and internal body temperatures rise appreciably.

Also evident from multiple independent studies (52) is that during steady-state levels of work intensity (% $\dot{V}_{O2\ max}$), skin evaporation generally increases with ambient temperature and mean skin temperature. For a given constant environment, thermoregulatory sweating (E_{sk}) is closely associated with absolute heat production and integrated mean body temperature (49). Additionally, skin evaporative heat loss is very closely associated to skin thermal conductance (K_{sk}). The value of E_{sk} increases almost linearly with K_{sk} from a threshold value ($K_{sk} = 19$ W $\cdot$ m^{-2} $\cdot$ °C^{-1}) up to about 300 W $\cdot$ m^{-2} $\cdot$ °C^{-1}.

Two passive and active regulatory processes contribute to effective K_{sk}: convective heat transfer by blood circulation and heat transfer by conduction through the core to tissues to the outer layers of the skin. Skin thermal conductance is therefore the sum total of the effect on the net metabolic heat dissipated at the skin (H_{sk}) divided by the difference between the core and skin temperatures ($T_c - T_{sk}$), that is, $K_{sk} \approx H_{sk}/(T_c - T_{sk})$. To an extent, heat flow transducers have been used to quantify net heat loss from peripheral areas when properly calibrated (39, 52). This index is limited as a measurement of effective skin blood flow, particularly when the core to skin temperature gradient is small but the net heat flux (H_{sk}) is high, as in exercise in hot, humid situations (6, 75). For this reason, various other techniques of measuring skin blood flow (for example, venous occlusion plethysmography, VOP, Laser Doppler, and water-perfused heat-flow capsules) (71, 78–80, 125) have been used to establish the relationships of thermal drive (composed of skin and internal body temperatures) and effective heat loss produced by skin blood flow (105). It has been possible to confirm that thermoregulatory control of both skin blood flow (arm VOP) and thermoregulatory sweating are undoubtedly linked to the same efferent drive and to each other.

Kraning and Gonzalez (83) studied skin heat exchange properties during continuous and intermittent exercise at $T_a = 30$°C. Two separate scenarios were examined, one in which heat stress could be adequately determined by heat exchange mechanisms active in the heat balance equation *(compensable)* allowing steady-state and one in which steady-state was impossible *(uncompensable)*. Intermittent exercise involved repeated 10 min periods consisting of 4 min of moderate walking exercise (800 W), followed by 2 min episodes of heavy jogging (978 W), and a seated resting period (134 W). During the continuous mode of exercise, heat

production was picked according to a time-weighted average of the intermittent exercise (408 W) Figure 4.11 illustrates the time course of the core and skin differences ($T_{re} - \bar{T}_{sk}$) during the intermittent work experiments. Peculiar oscillations, attributable to changes in K_{sk}, representing heat flow from the underlying skin vasculature are readily apparent. During either compensable or uncompensable heat stress, it was observed that the core to skin temperature gradient was always higher during the jogging phase and lowest during the recovery phase. For the most part, the core–skin gradient observed during compensable heat stress was almost twice that of the uncompensable one, within the prediction limits of the skin blood flow requirements based on partitional calorimetry. Interestingly, during compensable heat stress the amplitude of the core to skin gradient enlarged with time in the walking and jogging phases (presumably this would allow reduced cutaneous blood requirements as the steady-state becomes achieved). However, little growth in the core to skin gradient is apparent during the uncompensable heat stress. It is hypothesized that growth of the core to skin gradient apparent during compensable heat stress fosters a physiological mechanism allowing the body to counteract the effects of transient vasoconstriction, which is not totally possible during severe heat stress.

Vasomotor control. The skin of the human body is supplied with a rich network of blood vessels and also responds to adrenergic drives (71, 128) as well as to local thermal changes. Because arterioles are richly innervated by the sympathetic nervous system, the cutaneous circulation is controlled principally by reflex activity. There are two distinct structural regions characterized according to control: acral (or apical) and nonacral sites (71, 128). *Acral* sites include skin regions with high surface area compared to volume ratios, such as tips of extremities, nose, ears, and lips. Along with an abundance of arterioles, significant numbers of arteriovenous anastomoses (AVAs) abound in acral sites (128, 149). Characteristic *nonacral* sites, such as the torso, forearms, upper arms, and legs, are distinguished morphologically from acral sites by a lack of AVAs.

AVAs provide convenient shunts between the arterioles and venules, which detour blood flow from exchange vessels and thereby provide oxygenated blood to specific regions. The AVAs also allow dumping of heat as the flow passes through the cutaneous venous system (90, 135).

Steady-state blood flow (Bf) in acral sites may approach very high values (71, 128). In the human hand, Bf can approach as much as 60–70 ml $\cdot$ min^{-1} $\cdot$ 100 cc^{-1}. Since the total hand is composed of about 50% skin surface, for example, with full opening of the hand AVAs there will be some 120 ml $\cdot$ min^{-1} $\cdot$ 100 cc^{-1} diverted directly to the region as skin blood flow. Con-

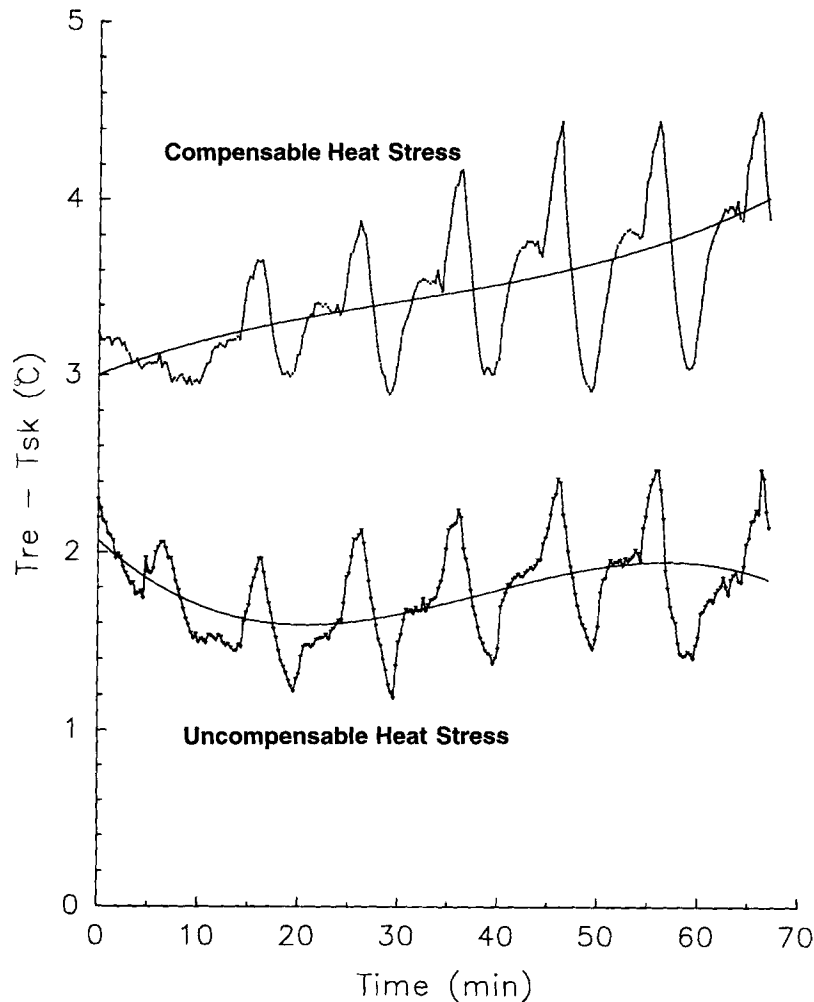FIG. 4.11. Time response (15 s intervals) of the gradient between $\overline{T}_{re}$ and $\overline{T}_{sk}$ occurring during intermittent work with compensable and uncompensable heat stress.

trol of skin blood flow, and therefore heat flow, in the acral sites of humans is thought to be primarily due to adrenergic vasoconstrictor nervous activity (71, 128), mediated by both α_1- and α_2-adrenergic receptors. The extent of β-adrenergic receptor activity is limited. Consequently, augmentation of hand blood flow, as a homeostatic response to increase heat loss during hyperthermia, occurs principally by the release of vasoconstrictor activity. Local stimulation by warm receptors affects skin blood flow as well, augmenting the full skin blood flow response. In so-called neutral ambient temperatures (Fig. 4.6) (for example, where $T_{sk} = T_{sk,o}$ in which $T_{sk,o}$ is a reference threshold temperature where no vasodilation occurs), elevations in skin blood flow in a given acral site generally occur by reflex withdrawal of sympathetic adrenergic drives.

With continual cold stress, blood flow to the skin is greatly reduced, but transient heat flow to extremities is often augmented in a few individuals and animal species

by cold-induced vasodilation (CIVD) (28, 29, 74, 149), which is modified to a great extent by body heat content. Keatinge (74), however, challenged the physiological significance of CIVD in preventing frostbite for humans, suggesting that the variability in onset time and the fact that it is not a continuous phenomenon precludes its homeostatic practicality. The main effect of CIVD, according to Keatinge, is a detrimental one affecting heat loss in acral regions. For example, Roddie (128) cites studies showing that exposure of the hand to ice water induced heat loss, which resulted in a drop of esophageal temperature by some 4°C/h. Further evidence for the detrimental role of CIVD, rather than improvement of homeostasis, is seen in the responses to cooling in isolated vessels of various homeotherms (73). Results indicate that those cutaneous blood vessels habitually exposed to cold respond to constrictor drugs at lower threshold temperatures than in similar unacclimated vessels (73). Other studies, based on analytical

theory applied to human extremities, indicate that thermal insulation has the greatest impact on conservation of heat, and, at most, the countercurrent efficiency amounts to no more than 15% (119).

Flavahan's model of action represents the mechanism of CIVD (43). Although the dispersion of α_1- and α_2-adrenoceptors and changes provoked by cooling are similar throughout human vascular tissues, α_2-adrenoceptors apparently have the most prominent responsivity in cutaneous blood vessels. Flavahan shows that preferential blockade of the α_2-adrenoceptors inhibits the vasoconstriction normally occurring by local cooling. Consequently, this causes an inability of norepinephrine released at specific sites to induce contraction of smooth muscle at cold temperatures. Persons with Raynaud's phenomenon exhibit a complication in which spasmodic digital vasoconstriction develops due to a change in the balance of the α-adrenoceptors, favoring an increase in the α_2-adrenoceptors (43).

Sensible heat loss is regulated by the mechanisms which improve the temperature gradient between core and skin to ambient. This can be by an increase in skin surface temperature to a level higher than the ambient, thereby enhancing heat exchange. Reflex mechanisms in various animals allow facile control of heat loss in specialized structures (67, 77, 141). Not as well characterized are the mechanisms associated with release of adrenergic vasoconstriction and neurogenic cutaneous vasodilation in humans and other animals during exercise in hot environments. During passive (nonexercising) heating of a cool human subject, Roddie (128) showed an initial inhibition in the vasoconstrictor tone followed by active vasodilation when core temperature reached a threshold. The initial phase of the biphasic rise in forearm skin blood flow, resulting from the release of adrenergic vasoconstrictor tone, was not responsive to arterial atropine injection. However, the second phase was blocked by local atropine injection and, to date, has been classically associated with active cholinergic vasodilation. Various vasodilatory substances have been suggested as mediators active in vasodilation (128). More recently, Kolka and Stephenson (78) showed in unacclimated men, exercising at 55% of their $\dot{V}_{O2peak}$ that systemic atropine resulted in a higher slope in the forearm blood flow to esophageal temperature response (at a constant skin temperature of 34°C). This is shown in Figure 4.12 from that study. They hypothesized that active cutaneous vasodilation (albeit accompanied with the anticholinergic activity of atropine on the eccrine sweat gland) could be mediated by the release and activity of one substance, vasoactive intestinal polypeptide (VIP), which is present in neurons supplying the sweat gland. Although plasma samples were not analyzed for VIP in their study, another study (26) did show that VIP is significantly elevated postex-
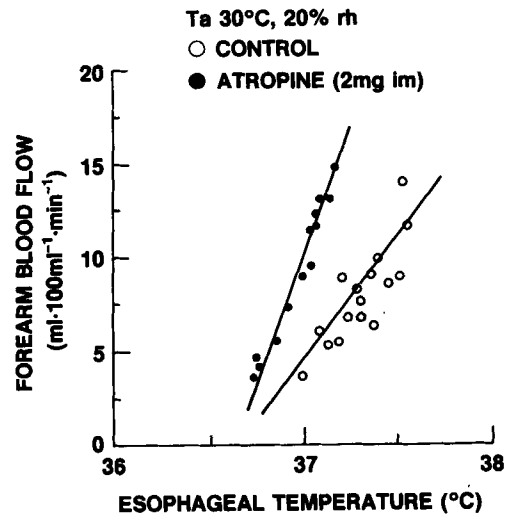

FIG. 4.12. Forearm blood flow plotted as a function of esophageal temperature during exercise before and after atropine injection. From Kolka and Stephenson (78).

ercise (60% $\dot{V}_{O2max}$) and a significant relationship between whole-body sweating rate ($r = 0.73$) and skin blood flow ($r = 0.46$) to ΔVIP levels was demonstrated.

A clear example of generalized cutaneous vasodilatory action, without sweat gland inhibition, also occurs during the ingestion of niacin, as seen in Figure 4.13. Ingestion of niacin (300 mg, or 5 mg/kg body weight) induced a provocative increase in forearm blood flow concomitant with the increases in sensible heat flux evident in Figure 4.13 by a rapid rise of mean skin temperature. The effects of the tremendous heat loss reduced core temperature by some 0.55°C within 53 min after ingestion of the niacin. However, coupled with the cutaneous heat loss, increased forearm blood flow responses, and core temperature lowering there was a large decrease in mean arterial pressure, which precludes general use of the vitamin for potential pretreatment medication for hyperthermia in many individuals.

Sudomotor control. In a variety of homeotherms, water loss from the skin surface is augmented by the secretion of sweat from specialized glands. Factors which control sweating responses in many species have been documented in many reviews (17, 40, 68, 126, 127). Robertshaw (126, 127) has surveyed the neural and endocrine effects on sweating in a variety of species. It is clear that there exists a great species diversity in the function and control of sweating. In a variety of homeotherms water loss from the skin surface is augmented by the secretion of sweat from specialized glands. Bligh and Allen (17) reviewed many of the morphological and functional distinctions in two types of gland that they introduced as epitrichial and atrichial. *Atrichial* glands (in which the duct opens onto skin surfaces independent
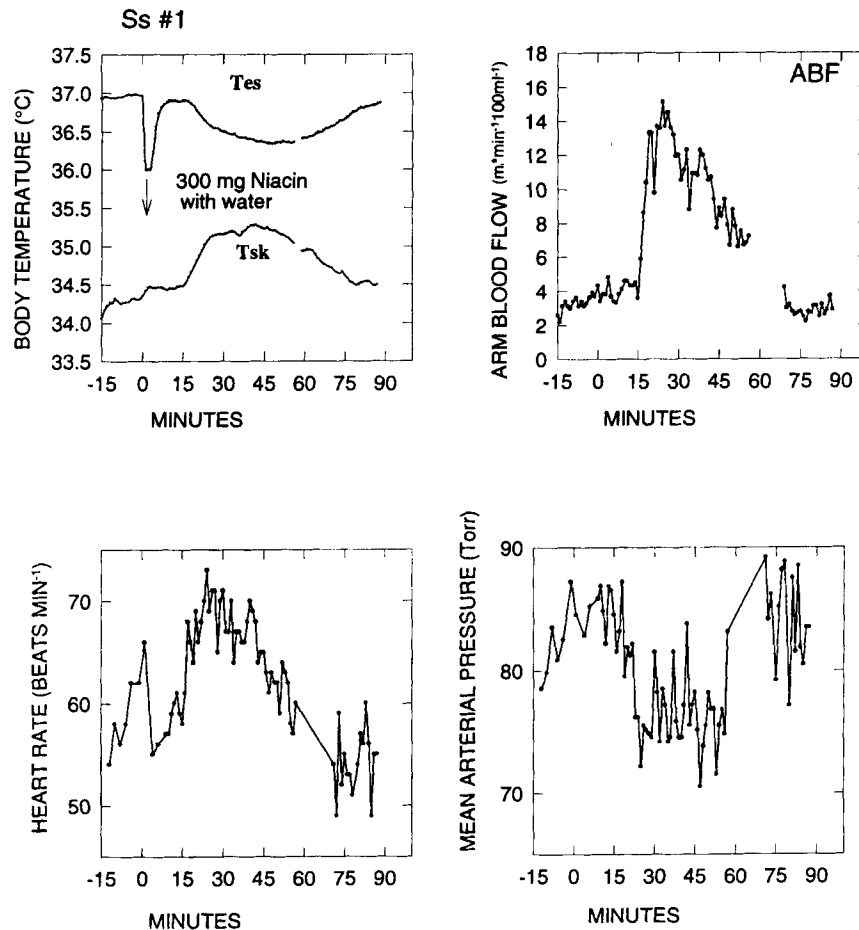
FIG. 4.13. Physiological responses in a subject following ingestion of niacin. From Stephenson and Kolka (144).

of hair follicle units) are innervated largely by cholinergic nerves and are conventionally referred to as eccrine. *Epitrichial* sweat glands are referred to as apocrine and for the most part are innervated by adrenergic nerves.

Characteristic of both humans and subhuman primates is that sweating activity responds proportionally to $\delta T_{cor}/\delta t$ and peripheral temperatures above a certain threshold temperature. The pattern of eccrine sweat gland secretion is cyclical rather than continuous, and in humans, sweating is synchronous to a large extent over the entire skin surface. This characteristic sweating response suggests that regulation is via efferent activity directly stemming from CNS controller activity. Robertshaw (126) considers that the fact that there is a relatively fast hydrolysis of acetylcholine by acetylcholinesterase allows for a "delicately controlled system" in the eccrine sweat gland. Numerous studies (19, 59) have alluded to the direct link between anterior/preoptic hypothalamic heating and sweating responses. However, verification that sweating rate is stimulated by direct hypothalamic warming in the rhesus monkey at

different mean skin temperature levels was unequivocally confirmed by Elizondo and Johnson (40). In humans there are substantial studies showing an enhancement of the sweating response with physical training and/or acclimation by a certain work intensity and period of time in the heat (106, 115, 125). For example, Nadel et al. (105) showed that exercise training increased the slope of the response of local skin sweating rate to the esophageal temperature, in comparison to the untrained state, presumably enhancing the gain of sweat gland response to integrated CNS efferent drive. During heat acclimation, however, their subjects decreased the threshold esophageal temperature for the initiation of sweating, and the slope in sweating rate:T_{es} curve remained unchanged compared to the unacclimated state when subjects were challenged to a similar exercise intensity ($50\%-60\% \dot{V}_{O2max}$). This response was also observed in another independent study in which heat acclimation was produced by daily exercise ($25\%-27\% \dot{V}_{O2max}$) but coupled with humidity transients elevated to an extreme level to cause excessive skin wettedness ($>100\%$) (57). Both studies suggest

that heat acclimation alters the central thermoregulatory drive in some definitive manner. Uncertain in both studies was whether the human eccrine sweat gland develops the ability to be more responsive to acetylcholine or whether the neuroglandular junction actually releases additional transmitter. Sato and Sato (131) first studied isolated eccrine sweat glands from human subjects who listed themselves as heavy sweaters; they had eccrine sweat glands exhibiting larger volumes, longer secretory tubules, and enhanced sensitivity to methacholine (MCh) compared to subjects who listed themselves as poor sweaters. Sato et al. (130) subsequently showed in the patas monkey that heat acclimation definitely resulted in an increase in size and an enhanced sweat secretion with increasing dosage of MCh of isolated eccrine sweat glands.

Kraning et al. (84) measured the dose–response attributes of human forearm sweat glands to MCh and the extent of active gland density following a heat acclimation routine that elevated core temperature by 1.5°C each day (Fig. 4.14). Their results and those of Sato et al. (130) strongly confirm the hypothesis that heat acclimation modifies sweating activity principally through a peripheral mechanism that augments the secretory response of the eccrine gland to the neurotransmitter.

In general, multiple studies have also documented that the close relationships of arm VOP:T_{cor} and sweating rate:T_{cor} are maintained during fever states, heat acclimation, changes in menstrual cycle of women, and hypohydration and that these relationships are maintained despite wide perturbations of time of day (79, 103, 125, 133).

Table 4.4 gives a summary of some of these experimental findings, illustrating that both heat loss components related to sudomotor drive and skin blood flow are coupled functionally to equivalent efferent thermal drive from the CNS.

Exchange by Conduction–Convection and Adaptive Properties. In heavily furred animals or when humans are clothed, heat exchange is not necessarily directed to the environment, except at exposed skin sites. Sensible heat transfer [that which is associated with a temperature difference due to dry (nonevaporative) radiative plus convective heat exchange] often arises by a cascade of axial/radial conductive–convective mechanisms from internal heat transported through skeletal muscle and the fat layer to the skin and from there to the environment (81, 82).

Dry heat exchange between the skin and the environment in humans is best described by operative temperature (T_o), which has been shown to integrate resident heat transfer coefficients at the skin boundary layer with the thermal environment (49). This rational concept has been extended for use in a wide variety of other homeothermic species by Bakken (3) and others (151), who have experimentally validated that the operative temperature (called T_E by the authors) also serves to unify sensible heat loss in complex thermal environments for many animals besides humans.

Convective and Respiratory Heat Transfer and Adaptive Response. With each respiration, air is introduced into the respiratory tract at the given ambient conditions, but upon reaching the alveoli the air becomes 100%
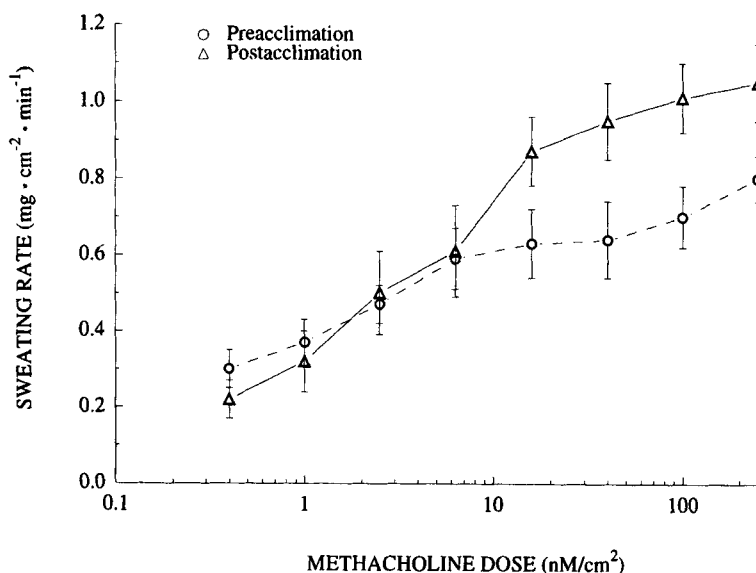

FIG. 4.14. Effect of heat acclimation on the sweating response to increasing doses of methacholine in six subjects (±SEM). From Kraning et al. (84).

TABLE 4.4. *Responses of Thermoregulatory Skin Blood Flow (SkBF) and Sweating ($\dot{m}_s$) to Specific Factors*

	Primary Controls	Secondary Controls	Modifying Effects	Independent Factors
Arm and finger SkBF	(1) Sympathetic vasoconstrictor drives and central (T_c) and peripheral temperatures ($\overline{T}_{sk}$) (2) Active vasodilation is highly T_c-dependent: relative importance of T_c and T_{sk} coefficients range from 8:1 to 12:1	(1) Local skin temperature, rate of change of T_{sk} ($\delta\overline{T}_{sk}/\delta t$) on vasoconstriction (2) Several metabolic, humoral products associated with $\dot{m}_s$ [PGE_1, co-release of acetylcholine (ACH) and VIP?]	(1) Multiplicative (local T_{sk}) with additive components (2) Coordinated with sweating at $\overline{T}_{sk} = \geq$ 34°C, but not at all skin sites	Effect of work activity to a great extent
Sweating: whole body (E_{rsw}) and local ($\dot{m}_s$)	Additive central integration of core and peripheral temperatures. Relative ratio of core:skin ranges from 5:1 to 10:1	Local skin temperature in a multiplier; ($\delta\overline{T}_{sk}/\delta T$) is more influential than local skin warming; skin hydration (local wettedness)	Strongly multiplicative	Work factors

Compiled from Gonzalez (52); new data from refs. (78–80, 128).

saturated and is considered at deep body temperature. In many mammals, expired air varies little from the temperature or humidity of the alveolar air. Small mammals (135) have adapted the means to recover heat or cool vapor. Rate of convective loss (watts) is equal to the product of the mass flow rate of air through the respiratory tract, specific heat of air and temperature difference of the inspired ($T_{in,a}$), and expired ($T_{ex,a}$) air. Mitchell (100) demonstrated that $C_{res} = 0.02\dot{V}_{ESTPD}(T_{ex,a} - T_{in,a})$, where 0.02 is a factor incorporating mean density and specific heat of the air and $\dot{V}_{ESTPD}$ is the ventilatory minute volume (liter/min). According to this equation, Mitchell estimated that a person exercising at a $\dot{V}_{O_2}$ of 1.5 liter/min in a cold environment ($T_a = -30°C$) with a $\dot{V}e$ of 35 liter/min would exhibit a C_{res} loss of only about 45 W, or 10% of M.

Ventilatory rate affects both dry (C_{res}) and respired evaporative heat loss, but the significance is most remarkable in nonsweating animals compared to sweating ones (see Fig. 4.15). Respiratory heat loss caused by an efficient panting mechanism in humans (and other homeotherms with eccrine sweat glands) is a minor avenue used to regulate internal body temperature. Several accounts (67, 100) show that only 20%–30% of total water loss occurs by obligatory increases in respiratory volume and that this process is only minimally associated with increases in rectal temperature. In homeotherms which rely heavily on sweating as a method for dispelling heat in warm environments, respiratory heat loss is minor and can be adequately estimated by the classical equations assumed in the perfect gas laws. For humans C_{res} and E_{res} in sea-level environments can be estimated using Fanger's equations (Eqs. 4.9 and 4.10) (41, 150).

However, in birds and mammals (with variable or no eccrine sweat glands), panting becomes the principal

means of body temperature regulation (17, 67, 135). Animals which employ both panting and sweating (such as the goat) apparently adapt the most efficacious avenue necessary for control of hyperthermia. For example, when resting goats are hypohdyrated and exposed to

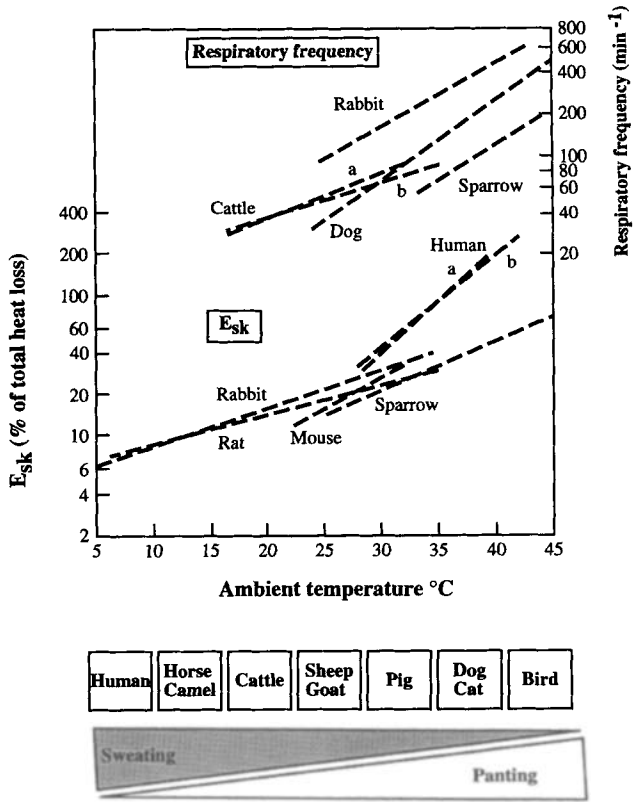

FIG. 4.15. Respiratory frequency and evaporative heat loss plotted vs. ambient temperature in various species *(top panel)*. Bottom panel shows the relative reciprocity of sweating and panting in various animals. Adapted from Hensel et al. (67).

excessively warm environments, sweat secretion is reduced but an increase in panting is facilitated by changes in core temperature (109). During exercise, both the respiratory heat loss and the sweating rate of well hydrated goats were shown to be linearly related to central blood temperature (109). Nijland and Baker (109) showed, however, that the respiratory heat loss to central blood temperature relationship was unaltered by hypohydration (upper body weight decreases of 8.6%) when the goats exercised in a 35°C dry ambient. However, there occurred an increase in the central blood temperature threshold for initiation of sweating with an unaltered slope.

Boulant and Gonzalez (20) showed in rabbits that the panting response stimulated by a thermal clamping of preoptic temperature was modified by the skin and core temperature levels. Warm skin and colonic temperatures in the hyperthermic range (37°–40°C skin and 40°C colonic) decreased the hypothalamic thermosensitivity (the unit change of response for each degree change in hypothalamic temperature) for panting. In moderate body temperature zones (skin temperatures between 33° and 36°C and colonic temperatures between 38.6° and 39.6°C), preoptic heating increased the hypothalamic thermosensitivity for panting. These results suggest that warm skin receptors (presumably affecting warm-sensitive neurons) have a blunted role in stimulation of the heat loss response (19).

Evaporative Heat Loss and Adaptive Response. As defined, skin wettedness is the fraction of the body surface wet with sweat (45). If the level of evaporation remains constant as ambient water vapor rises, skin wettedness will increase (55, 57). The result is that the skin surface area becomes increasingly larger to facilitate evaporation of water.

Mole (96) considered the human skin surface as a continuum affected primarily by a patchwork of wet and dry areas. Skin relative humidity (Φ_{sk}) and total skin saturation vapor pressure ($\Phi_{sk} \cdot P_{s,sk}$) are the critical factors that describe the moisture concentration gradient to the ambient. Kerslake reconciled differences between the concepts of skin wettedness and skin relative humidity and showed that an equivalency in both occurs when the ambient water vapor pressure (P_w) is 0 and the skin surface is 100% wet (75). However, even if there is no active sweating, water continues to be lost from the skin by diffusion by an amount equivalent to about $0.05 \cdot E_{max}$ (49, 75). Development of a small resistance-type dew point sensor (58) gives the direct measurement of $\Phi \cdot P_{s,sk}$. This has allowed the combining of the two concepts, by which calculation of regional water loss (E, $g \cdot m^{-2} \cdot h^{-1}$) from specific skin areas may be described as:

$$E = h_e(\Phi \cdot P_{s,sk} - P_w) \qquad \text{(Eq. 4.80)}$$

$$E = w \cdot h_e(P_{s,sk} - P_w) \qquad \text{(Eq. 4.80')}$$

where h_e is the evaporative heat transfer coefficient, which includes the boundary layers between skin, clothing, and ambient. By solving both equations for skin wettedness,

$$w = (\Phi \cdot P_{s,sk} - P_w)/(P_{s,sk} - P_w) \qquad \text{(Eq. 4.81)}$$

Berglund (13) and others (54) used miniature humidity sensors to measure both $\Phi \cdot P_{s,sk}$ and P_w utilizing Eq. 4.81 to calculate skin wettedness under clothing during steady-state and transient conditions.

Evidence that skin wettedness is physiologically adjusted to provide the required rate of evaporation has been shown by many others (80). In the study by Kolka et al. (80), atropine (2 mg, im) blocked the sweating response and local skin wettedness accompanied the appropriate decreases in sweating rate. However, during control warm experiments local skin wettedness was not shown to be limited by E_{max} (as long as $P_{s,sk} > P_w$) but by the rate of sweat secretion tightly associated with internal core temperature drive (104), as originally suggested by Kerslake (75) and others (49). Thus the actual fluid output (by thermoregulatory sweating) is dependent on a sudomotor signal and available fluid delivery to the eccrine sweat gland that is evaluated by local skin wettedness. There is also some evidence that properties affecting skin relative humidity are associated with changes in the periglandular flow of blood in the eccrine sweat gland (131).

Physiological Responses of Heat Loss by Thermal Radiation and Adaptive Response

All homeotherms (as well as all materials) are absorbers and emitters of thermal radiation. The second law of thermodynamics requires that, for a given wavelength, each substance's emissivity and absorptance be the same. Emissivity is defined as the ratio, for a given wavelength interval, of the energy emitted per unit area divided by the energy emitted by a blackbody of the same area. For solid materials, the actual absorptance is a fraction of the wavelength. A *blackbody* is defined as a body or surface that absorbs all wavelengths. Such a cavity, held at a given temperature, will generate radiant energy according to a function of wavelength (λ_m) (62). Infrared radiation (beyond the red end of the visible spectrum) includes wavelengths from 0.4 μm to about 150 μm (far infrared) (95, 143).

Thermal radiation is the "radiant energy emitted by

a medium that is due solely to the temperature of the medium" (143). The temperature of the medium governs the actual emission of thermal radiation. Of concern to human and other animal skin structural properties and thermal exchanges are the two distinct bands: solar (shortwave) radiation, occurring in the range of 0.3 μm, and longer waves occurring at about 0.7–150 μm.

Human skin displays a variable thickness over different parts of the body and is therefore heterogenous to incident radiation. In the spectral region, from 0.3 μm to 3 μm, there is a maximum reflectivity for white and black skin at about 0.8–1.2 μm, roughly the λ_{max} of maximum radiation for most infrared heaters. From 3 μm to 20 μm, reflectance becomes independent of skin pigmentation and is quite low. Skin pigmentation is an important variable at short wavelengths near 0.4–0.7 μm; a darkly pigmented person will become markedly more heated by direct radiation $\geq 2,500°K$ than would one of lighter skin. For humans, the wearing of clothing, however, lessens the above differences (1, 5, 6). By decreasing the emissivity of clothing, a reduction in radiative heat loss adds an additional protection against heat stress. Emissivity (ε_s) of human skin varies with spectral region, but skin emits in the infrared spectrum (5 μm to 20 μm). It is the emissivity value in this range that is most important in human heat exchange.

In the presence of clear days with desert heat, clothing also decreases heat transfer by thermal radiation but affects the evaporative process through the garment. Typical decreases in the amount of radiant heat on the skin can be about 30%–40% with ordinary working clothes near industrial furnaces (5). Early work by Adolph et al. (1) showed that the effective solar load on seminude men walking in the desert was about 233 W. When clothing of light color was worn, demand on evaporative (by thermoregulatory sweating) heat transfer was lessened by some 116 W (roughly 50% of solar load), which would tend to drop the effective air and globe temperatures by 4°–5°C (5). The thick, loose-fitting, light-colored clothing used by natives in the hot dry zones of the Middle East offers a guard for thermal extremes, spanning excessive heat by solar gain in the day and rapid cold spells at night (5). In coastal areas of such zones, ventilation of clothing by activity allows some evaporation, but in humid zones, where air temperature stays about 32°–33°C with humidities in the 90% rh range, clothing is more of an impediment since the effective ambient water vapor pressure gradient is so small (6, 47).

The two ways of measuring emissivity of skin, analysis of reflecting power and analysis of absorbing power in the above ranges, are technically complex. Hardy (61), in a study of the emitting power of the skin, analyzed skin temperature accurately with an infrared radi-

ometer and compared this with the temperature of a "perfect blackbody" using the Stefan-Boltzmann law. Hardy found $\varepsilon_s = 0.98 \pm 1\%$, a value constant for >0.4 μm. The ε_s can be considered, therefore, very nearly 1.0 (61, 101).

To an extent, the color of clothing affects its reflective properties to solar radiation. However, Breckenridge and Pratt (25) showed little effectiveness of color on overall heat exchange. Others have shown some effectiveness of dyed fabrics as reflectors of solar radiation (spanning wavelengths from 0.7 μm to 0.9 μm) (32). To a great extent, this effect is color-dependent. For similar samples (cotton poplin) treated with various shades of black, blue, khaki, and white, or coated with aluminum paint, the expected highest reflection coefficients were with white and aluminum and the most absorptive with black cloth. Aluminized suits (with reflective fibers) are also attractive embellishments in ensemble construction, especially for use during exercise outdoors (23). However, such low-emissivity fabrics generally have poor moisture permeation properties (23). One study by Berglund et al. (16) showed that color and weave also influence absorbed radiation. Their subjects, exercising in skin-tight, form-fitted garments with varied wicking material, increased rate of weight loss differentially as a direct result of garment color in respect to incident direct radiation. Rate of body weight loss (by thermoregulatory sweating) for a given increase of radiation was highest with blue knits followed by black, white, nude (shorts), and a silver-colored body suit (90% polypropylene, 10% lycra). Extrapolation of the results in terms of extent of hypohydration, based on loss of body water, shows that the higher reflectance of the silver-colored suit was water-sparing for up to 5 h compared to rate of water loss apparent in studies with nude skin in a 27°C environment with simulated solar intensity of about 847 W · m^{-2}.

Additional information concerning mechanisms of radiative heat exchange between the clothed human body, various animal coat colors and thicknesses, and the ambient can be found in other reviews (9, 10, 22, 24, 48, 49, 95, 151, 152). For other animal species, radiative heat loss and heat gain is associated strongly with type, color, and depth of fur, plumage, and/or pelt. Cena and Clark (32) reviewed the literature and pointed out that, in general, reflectivity of animal coats in the short-wave spectrum is augmented from the blue end to the near infrared, regardless of coat color. An implicit assumption is that animals with dark-colored coats (having greater absorptivity to short-wave radiation) will gain more from solar loads than animals with light-colored coats (97, 152).

Walsberg et al. (152) found that the relationship between coat color and radiative heat gain was variable, depending on the natural ambient environment's wind

speed. In a study in which pigeons with white or black plumages, maximally erected, were exposed to 900 W $\cdot$ m^{-2} of simulated solar radiation, Walsberg et al. (152) showed that below a threshold of about 3 m/s wind velocity, the radiative heat gain of pigeons with black plumages was much higher (from about 100 to 150 W $\cdot$ m^{-2}) than that of pigeons with white plumages (radiative heat load from 75 to 87 W $\cdot$ m^{-2}). However, at wind speeds from 3 m/s to 9 m/s, solar heat load was equivalent in pigeons with black plumages at a threshold of 3 m/s but decreased as wind speed increased (from 75 to 25 W $\cdot$ m^{-2}).

Skuldt et al. (142) found that in artificial fur, fur conduction and radiation through the hairs were almost of equal magnitude in deep fur, but radiation increased as fur density decreased.

HEAT LOSS IN SPECIAL ENVIRONMENTS

Heat loss mechanisms active in disparate environments [hypobaric (altitude), hyperbaric, and aquatic] are uniquely tied to both systemic physiological changes and the skin–environment interface (4, 73, 86, 122, 153, 156).

Hypobaric Environments

The effect of high altitude (hypoxic–hypobaric stress) on heat loss specifically alters the boundary layer insulation (I_a) between the skin and air. This is even more prominent with high wind speeds (28). At the other extreme, the lower the wind speed and the atmospheric pressure, the greater is the value of the I_a, or the lower is the heat conductance. To maintain a given metabolic rate without increasing sensible heat flux when wind speed is elevated also requires an appropriate increase in total clothing insulation. Gagge and Nishi (49) clearly demonstrated that the heat and mass transfer properties that govern exchange at the skin–environment interface have certain relationships affected by barometric pressure: (1) the heat transfer coefficient (h_c) in still air, which is dependent on metabolic activity or forced convection, varies as a factor of $P_B/760$; (2) the effective Lewis relation based on the ratio of the evaporative heat transfer coefficient (h_e^*) to h_c^* is also elevated with hypobaric environments, due to the effect of P_B on density and mass diffusivity of the fluid on the surface properties; (3) these latter properties also alter the extent of possible convective heat exchange (C) and the thermal efficiency factor of clothing, as discussed in the section on Psychrometrics of the Human Heat Balance Equation. The effect of hypobaric environments on the Lewis relation can be described as $2.2 \cdot (760/P_B)^{0.45}$, °C/Torr,

which would increase the maximum evaporative power of the environment.

Characteristically, as skin-sensible heat loss drops with hypobaric environments, skin diffusion increases (31). One possible mechanism for this effect could occur via augmented interstitial fluid pressure to the skin (103). These mechanisms can have an impact on climbers in high terrestrial areas, particularly with the augmented intensity of solar radiation at high altitude (5, 47) resulting in both hypohydration and increased transepidermal water loss.

The hypoxia that is prevalent in hypobaric environments also affects general heat exchange and the control of thermoregulatory sweating and skin blood flow in an as yet unknown fashion (80). Rowell et al. (129), in a hypoxic breathing study, were not able to document any alterations in the relationship of cutaneous blood flow to T_{es} response in subjects exercising in sea-level environments. Kolka et al. (80) however, studied four men and four women (in the follicular phase of the menstrual cycle) who exercised at 60% $\dot{V}_{O_2}$ peak at sea level and in two hypobaric environments (P_B at 552 and 428 Torr). They found a lower core temperature reference point (T_{es}) for vasodilation in both hypobaric exposures; however, the slope in the arm blood flow to T_{es} relationship during constant exercise was significantly lower than that observed in the sea-level experiments at equivalent local T_{sk} (34°–35°C). Alternatively, although there occurred no change in the reference core temperature for initiation of local sweating, the slopes of the sweating rate to core temperature were decreased by hypobaria. Applying Boulant's hypothalamic neuronal model for thermoregulation (19) to these results reveals some interesting hypotheses. It is possible that the cutaneous warm receptors, sensing increases in skin temperatures during exercise, have little effect on the hypothalamic control of skin blood flow in hypoxia-hypobaria, but acute hypoxia may alter the local hypothalamic thermosensitivity for skin blood flow and sweating response. Additionally, it is possible that at the constant skin temperatures observed (33°–35°C), cutaneous cold receptors, which inhibit central neurons responsible for increasing skin blood flow, may become active.

Hyperbaric Environments

Special problems exist in heat loss mechanisms during acute and continual hyperbaric exposure, which have been detailed in several reports (4, 122, 156). In humans, several basic properties affecting heat exchange occur when a comfortably clothed person at sea level (for example, ≈ 1 ATA) is exposed to deep mines or pressurized tunnelling cabins where a gas mixture of helium and oxygen (HELIOX) or other gas mixtures (nitrogen, etc.) are used as the ambient envi-

ronment. Convection in the air layer is governed by the Nusselt number, which in laminar, free convection is associated with the Grashof number and the Prandtl number (114). The only variable strongly dependent on P_B is density. When air velocity is constant, h_c increases as a function of $P_B^{0.52}$ to $P_B^{0.62}$ (156). Rate of heat transfer by thermal radiation is not affected by atmospheric pressure. The mass transfer coefficient, however, increases with increasing pressure as h_c increases. Sensible heat loss (R + C) increases with elevated barometric pressure, but with increasing ambient temperature, there may be a frank heat gain on the individual. Since air velocity decreases with increasing pressure, the rate of transfer by C alone increases less rapidly than the increase in P_B. The effectiveness of thermoregulatory sweating and its evaporation, especially in warm hyperbaric chambers, diminishes with the increase in atmospheric pressure, and the moisture accumulates on the skin surface, increasing its film density. To date, there have been no studies quantifying the above mechanisms of control of sweating as a function of internal body temperature drive in hyperbaric environments.

During cold stress in hyperbaric environments, the gas property having the greatest influence on thermal insulation is thermal conductivity, which is independent of atmospheric pressure. In humans, the effect of increased sensible heat loss with hyperbaria in the cold reduces overall thermal insulation, and heat transfer is elevated from such exposed skin sites as the face, legs, and hands. Increasing insulation by clothing to compensate for these effects results in encumbrance and inhibition of evaporation especially during moderate activity (49, 156). The thermal insulative value of a thin garment gives enough protection against cold environments with increasing atmospheric pressures, but such a garment creates an effective barrier for insensible heat loss. Gagge and Nishi (49) demonstrated by rational equations that in a "thermally neutral" environment at 30 ATA, in the unclothed state (0.06 clo) the evaporative heat transfer coefficient would be decreased to about 20%–30% of its sea-level value. In the clothed state the effective evaporative heat transfer coefficient, which includes the clothing conductance factors, is only about 8%–10% of the sea-level value. Thus in a hyperbaric environment a normally porous garment used at sea level has the same physiological effect as a vapor-impermeable garment (159). Measurements of the regional skin wettedness properties in hyperbaric environments have yet to be done.

Aquatic Environments

Generally, physiological responses that affect thermal exchange during water immersion are also tempered by the specific insulation of the tissues. Heat exchange during partial or complete water immersion follows the biophysical principles covered in earlier sections dealing with Radiation, Convective & Evaporative Heat Exchanges. With complete water immersion, the radiative and evaporative components are absent during exercise, but the forcing drive of increased internal body temperature undoubtedly stimulates sweat glands. Eccrine sweat gland secretion and mass flux are mixed with the boundary layer flow resident within the free or turbulent convection of ambient water flow. However, little or no data exist addressing these properties.

A major obstacle to an adequate description of thermal exchange in water immersion is the determination of convective heat loss (49, 105, 158). Heat loss by this component is many times greater than in air because of the higher thermal capacity of water. Theoretically determined values (114, 120, 121, 158) are subject to assumptions of body shape and water motion. In humans, heat transfer equations derived by conventional analyses of the surface-fluid assumptions indicate that sensitivity is based on the skin to water temperature differences, which offer little thermal resistance (158). For example, consider the $h_{c,a}$ of a horizontal cylinder with a 0.02 m outer diameter in atmospheric air (1 ATA). With free convection at $\Delta T = 25$ K, the combined coefficient ($h_c + h_r$) is about 8 W · m^{-2} · K^{-1}. In water, however, $h_{c,w}$ is about 741 W · m^{-2} · K^{-1}, or about 93 times greater. A nude human in comparable circumstances would have an $h_{c,a}$ of about 3 W · m^{-2} · K^{-1}, but estimates of $h_{c,w}$ can range from 100 to 230 W · m^{-2} · K^{-1}, generally on point with theory, or 77 times greater, depending on the precision of partitional calorimetry or heat flow transducer methods (105, 158). This coefficient has been estimated by heat flow transducers in a precise swimming flume at water velocities from still to 0.97 m · s^{-1} impinging on a swimming person. The $h_{c,w}$ values varied for the unclothed subjects from a value of 230 W · m^{-2} · K^{-1} at rest in still water to 460 W · m^{-2} · K^{-1} at rest in moving water to 580 W · m^{-2} · K^{-1} while actively swimming (105).

With partial immersion outdoors R from direct solar load (that is, head and neck exposed), respiratory evaporative and convective ($E_{res} + C_{res}$) heat loss must be integrated with the net heat flux arriving at the skin surface as a function of the metabolic drive [$H_{sk} = M - (E_{res} + C_{res})$] in any heat balance.

For the nude, completely immersed human body, heat balance in a water environment is (49):

$$\pm S = H_{sk} - h_{c,w}(T_{sk} - T_w) \qquad \text{(Eq. 4.82)}$$

Estimates of rate of heat storage (S) in warm water (+S) or heat debt (−S) due to cold water immersion, lower than a comfortable $\overline{T}_{sk}$, are tempered by metabolic drive, specific thermal resistance of tissues, percent body fat, age, and physical fitness, and S becomes intimately

associated with percent body fat and the convective heat transfer coefficient from the exposed body surface to the water (146). Heat balance for other swimming mammals can also be estimated by Eq. 4.82, but there are sparse experimental data on the $h_{c,w}$ and metabolic components of the equation.

In humans, during complete water immersion when the body is covered with various protective garments, the effective heat transfer coefficient in Eq. 4.82 becomes $h_w{}^*$, which includes modification by a thermal efficiency factor ($F_{cl,w} \cdot h_{c,w}$), as demonstrated by Gagge and Nishi (49). The factor $F_{cl,w}$ is dependent on the ratio of the boundary–water resistance to the total insulation of the suit ($I_w/I_{T,w}$), in clo units. The total clothing insulation ($I_{T,w}$) is the sum of the skin-surface insulation of water I_w, in clo units, and the intrinsic insulation of the garment in water. Calculations of these factors are most accurately done with heated manikins, indoors in a water tank, where total H_{sk} (by direct power input to the manikin to maintain a constant skin-surface temperature) is a known variable. Since no radiative loads occur, the ratio $(I_w)^{-1}/0.1547$ can be used to estimate $h_{c,w}$. For example, a skin-free boundary water insulation of about 0.028 clo is estimated by this ratio as 230 W $\cdot$ m^{-2} $\cdot$ K^{-1}, the value found in one study (105). Reasonable values of total insulation during water immersion also have been obtained by physiological means using indirect calorimetry (72). By this classical method, total insulation and tissue insulation (a sum of the insulation of fat, muscle, and skin layers) was estimated in studies on Korean women breath-hold divers (72). Tissue insulation is first found by estimation of the deep core to skin temperature gradient. Total insulation from the "shell" (Fig. 4.9) to water (T_w) is:

$$I_T = (T_c - T_w)/H_{sk} \qquad [\text{m}^2 \cdot \text{K} \cdot \text{W}^{-1}]$$
(Eq. 4.83)

where T_c is deep core temperature (°C), T_w is water temperature, and H_{sk} is in W/m^2.

The challenge of predicting human endurance by quantifying heat loss in aquatic environments is directly associated to the development of suitable clothing for water immersion that allows extension of the critical thermoneutral zone by substitution of adequate insulation which humans lack. This focus has produced immersion suits that are "wet," with variable thicknesses of foam materials, or suits that are "dry," therefore operable in air but necessary during involuntary immersion (89). Dry suits often become too easily compressed in water and lose their air-measured thermal resistance. Nevertheless, serious hyperthermia occurs above 15°C water temperature (18) unless auxiliary aids to dissipate heat are designed into the suit (zippers, open hood vents, etc.). One thermal manikin study (18) quantitatively estimated upper environmental limits and min-

imum water temperatures for a wide assortment of protective suits. Analysis was done by manipulation of the basic sensible heat transfer equation during separate air and water immersion.

Across animal species there are examples of wet and dry suits (31) resident in respective species pelts. In seals and polar bears, for example, the uncompressed water layer of 2–10 mm (about 0.32–1.6 clo) juxtaposed to the skin acts as an effective thermal insulator permitting deep dives. Alternatively, the penguin's feather pelt acts as a suitable dry suit with a very efficient air insulator. Interestingly, the outcome of the use of a wet suit or a cotton suit by Korean breath-hold divers is that thermoregulatory mechanisms previously showing changes in response as a consequence of explicit cold acclimation in humans are not wholly functional anymore (72). In the past, these individuals developed cold acclimation by massive vasoconstrictor output during multiple seminude dives when they exposed themselves to cold water. They also relied on adequate compensation by altering intrinsic tissue insulation, which undoubtedly expanded the thermoneutral zone during cold-water immersion. A clear adaptation to the cold environment in humans has been replaced by behavioral adjustments.

Heat Exchange in Spacecraft

Heat exchange occurring in spacecraft and in various planetary surface environments where there is an absence of gravity is a particularly interesting concern because in such surroundings there also exists no free convection. Table 4.5 is a theoretical summary of responses suggested by the rational analysis of Hardy (60). The table shows variable heat loads present on an astronaut walking (3 mph) during a lunar day dressed in various ensembles in shaded surroundings or unshaded from the sun. The outer clothing temperature necessarily equals the ambient temperature. It is evident that the heat produced by activity must be balanced by heat loss to the environment (by evaporation and radiation) and gain from the sun. Several scenarios are possible, depending on the type of suit worn (38). With a dark suit it is obvious that the outer temperature would heat up to 81°C. Shielding the astronaut from direct solar load would be an improvement, but the reflected solar radiation from the lunar surface would still increase the outer temperature to 51°C. If sufficient evaporation of sweat were possible, the person could spend a few minutes at this temperature, but the physiological heat strain would still become too intense, particularly since the space suit is vapor-impermeable. If the suit were highly polished (for example, to reflect 90% of the sun's heat), the person would be hampered more so than with a black suit because without convective heat loss (due to air motion) and lack of radiative

TABLE **4.5**. *Heat Balance and Ambient Temperature of a Typical Person Walking on the Surface of the Moon, Lunar Day*

Exposure	Ambient Temperature (°C)	Metabolism (M)	Direct Radiation from Sun (Q_h)	Reflected Radiation from Moon Surface (Q_p)	Re-emitted from Moon Surface (Q_{LI})	Heat Loss by Radiation to Space ($Q\infty$)
Walking 3 mph; unshaded dark suit; $\varepsilon = \alpha_z = 1$	81	279	858	277	360	1,775
Walking 3 mph; unshaded reflective suit; $\varepsilon = \alpha_z = 0.1$	134	279	86	28	−85	308
Walking 3 mph; unshaded white suit; $\varepsilon = 0.8; \alpha_z = 0.2$	59	279	172	56	587	1,094

Heat loss values are all in $W \cdot m^{-2}$; calculated from Hardy (60).

heat loss, the outer temperature of the suit would approach 134°C. The type of suit best applicable, as seen in Table 4.5, is a pure white suit, which has high reflectance in the visible spectrum but serves as a blackbody and radiates adequately in the infrared.

REFERENCES

1. Adolph, E. F., H. Rahn, R. E. Gosselin, et al. *Physiology of Man in the Desert.* New York: J. Wiley & Son, 1947.
2. Aschoff, J., and R. Wever. Kern and schale im warmehauschalt des menshen. *Naturwissenchaften,* 45: 477–485, 1958.
3. Bakken, G. S. Measurement and application of operative and standard operative temperatures in ecology. *Am. Zool.* 32: 194–216, 1992.
4. Behnke, A. R. Temperature and comfort in pressurized environments. In: *Human Factors 1970,* Bulletin SF-70-3. New York: ASHRAE, 1970, p. 7–10.
5. Belding, H. S. Protection against dry cold. In *Physiology of Heat Regulation and Science of Clothing,* edited by L. H. Newburgh. Philadelphia: Saunders, 1949, p. 351–367.
6. Belding, H. S. Resistance to heat in man and other homeothermic animals. In: *Thermobiology,* edited by A. H. Rose. New York: Academic, 1967, p. 479–510.
7. Belding, H. S., and T. F. Hatch. Index for evaluating heat stress in terms of resulting physiological strain. *Heating, Piping, Air Cond.* 27: 129–136, 1955.
8. Belding, H. S., and E. Kamon. Evaporative coefficients for prediction of safe limits in prolonged exposures to work under hot conditions. *Federation Proc.* 32: 1598–1601, 1973.
9. Belding, H. S., H. D. Russell, R. C. Darling, and G. E. Folk. Thermal responses and efficiency of sweating when men are dressed in Arctic clothing and exposed to extreme cold. *Am. J. Physiol.* 149: 204–222, 1947.
10. Belding, H. S., H. D. Russell, R. C. Darling, and G. E. Folk. Analysis of factors concerned in maintaining energy balance for dressed men in extreme cold; effects of activity on the protective value and comfort of an arctic uniform. *Am. J. Physiol.* 149: 223–239, 1947.
11. Bennett, A. F., and J. A. Ruben. Endothermy and activity in vertebrates. *Science* 206: 649–653, 1979.
12. Benzinger, T. H., and C. Kitzinger. Direct calorimetry by means of the gradient principle. *Rev. Sci. Instrum.* 20: 849–860, 1949.

13. Berglund, L. G. Moisture measurements with a miniature resistance dew point sensor. In: *Moisture and Humidity: Measurements and Control in Science and Industry.* Research Triangle Park, NC: Instruments Society of America, 1985, p. 759–766.
14. Berglund, L. G., and R. R. Gonzalez. Evaporation of sweat from sedentary man in humid environments. *J. Appl. Physiol.* 42: 767–772, 1977.
15. Berglund, L. G., and R. R. Gonzalez. Mass transfer characteristics of the sweating human. *Special Technical Publication ASME.* New York: Amer. Society of Mechanical Engineers, 1978, p. 173–180.
16. Berglund, L. G., D. Fashena, X. Su, and A. Gwosdow. Absorbed solar radiation from measured sweat rate. *Proc. 13th Ann. Northeast Bioenerg. Conf.* New York: IEEE, 1987, p. 507–510.
17. Bligh, J. and T. E. Allen. A comparative consideration of the modes of evaporative heat loss from mammals. In: *Physiological and Behavioral Temperature Regulation,* edited by J. D. Hardy, A. P. Gagge, and J. A. J. Stolwijk. Springfield, IL: Thomas, 1970, p. 97–107.
18. Bogart, J. E., J. R. Breckenridge, and R. F. Goldman. Thermal protection of commercial dry suit diving systems. *USARIEM Technical Report T1/81.* Natick, MA: US Army Res. Inst. Env. Med., 1981.
19. Boulant, J. A. A hypothalamic neuronal model for thermoregulation. In: *Selected Topics in Environmental Biology,* edited by B. Bhatia, G. S. Chhina, and B. Singh. New Delhi: Interprint, 1976, p. 41–44.
20. Boulant, J. A., and R. R. Gonzalez. The effect of skin temperature on the hypothalamic control of heat loss and heat production. *Brain Res.* 120: 367–372, 1977.
21. Brebner, D. F., D. McK. Kerslake, and J. L. Waddel. The diffusion of water vapor through human skin. *J. Physiol. (Lond.)* 132: 225–231, 1955.
22. Breckenridge, J. R. Effects of body motion on convective and evaporative heat exchanges through clothing. In: *Clothing Comfort: Interaction of Thermal, Ventilation, Construction, and Assessment Factors,* edited by N. R. S. Hollies and R. F. Goldman. Ann Arbor, MI: Fiber Society, 1977, p. 153–166.
23. Breckenridge, J. R. Insulating effectiveness of metallized reflective layers in cold weather clothing systems. *USARIEM Technical Report T2/78.* Natick, MA: US Army Res. Inst. Env. Med., 1978.
24. Breckenridge, J. R., and R. F. Goldman. Human solar heat load. *ASHRAE Trans.* 78: 110–119, 1972.
25. Breckenridge, J. R., and R. L. Pratt. Effect of clothing color on solar heat load. *USARIEM Technical Report EP-155,* Natick, MA: US Army Res. Inst. Env. Med., 1961.
26. Buono, M. J., D. Dyar, R. Bulbulian, A. A. Sucec, J. Heaney, and G. R. Banta. The relationship between vasoactive intestinal

polypeptide and whole-body sweat rate during exercise. *Naval Health Res. Center Tech. Rep. No. 90-36,* San Diego, CA: Naval Health Center, 1990, p. 1–9.

27. Burton, A. C. Human calorimetry: II. The average temperature of the tissues of the body. *J. Nutr.* 9: 261–268, 1935.

28. Burton, A. C., and O. G. Edholm. *Man in a Cold Environment: Physiology and Pathological Effects of Exposure to Low Temperatures.* London: Arnold, 1955.

29. Carlson, L. D., and A. C. L. Hsieh. *Control of Energy Exchange.* New York: Macmillan, 1970, p. 151.

30. Carnochan, P., R. J. Dickinson, and M. C. Joiner. The practical use of thermocouples for temperature measurement in clinical hyperthermia. *Int. J. Hypertherm.* 2: 1–19, 1986.

31. Cena, K., and J. A. Clark. Thermal resistance units. *J. Therm. Biol.* 3: 173–174, 1978.

32. Cena, K., and J. A. Clark (Eds). *Bioengineering, Thermal Physiology and Comfort,* edited by K. Cena and J. A. Clark. New York: Elsevier, 1981, p. 289.

33. Cooney, D. O. *Biomedical Engineering Principles.* New York: Marcel Dekker, 1976, p. 1–458.

34. Chilton, T. H., and A. P. Colburn. Mass-transfer (absorption) coefficients: prediction from data on heat transfer and fluid friction. *Indust. Eng. Chem.* 26: 1183–1187, 1934.

35. Clark, R. P. Human skin temperature and convective heat loss: In: *Bioengineering, Thermal Physiology and Comfort,* edited by K. Cena and J. A. Clark. New York: Elsevier, 1981, p. 57–76.

36. Colin, J., and Y. Houdas. Experimental determination of coefficient of heat exchange by convection of the human body. *J. Appl. Physiol.* 22: 31–38, 1967.

37. Crawford, E. C., Jr., and B. J. Barber. Effects of core, skin, and brain temperature on panting in the lizard *Sauromalus obesus. Am. J. Physiol.* 226: 569–573, 1974.

38. Drake, R. M., J. E. Funk, and J. B. Moegling. Sensible heat transfer in the Gemini and Apollo space suits. In: *Thermal Problems in Biotechnology,* New York: Am. Soc. Mech. Eng., 1968, p. 96–112.

39. Ducharme, M. B., J. Frim, and P. Tikuisis. Errors in heat flux measurements due to the thermal resistance of heat flux disks. *J. Appl. Physiol.* 69: 776–784, 1990.

40. Elizondo, R. S., and G. S. Johnson. Peripheral effector mechanisms of temperature regulation: the regulation of sweating activities in primates. In: *Advances in Physiological Sciences, Vol 32: Contributions to Thermal Physiology,* edited by Z. Szelenyi and M. Szekely. Budapest: Pergamon, 1981, p. 397–408.

41. Fanger, P. O. *Thermal Comfort* (2nd ed.). New York: McGraw-Hill, 1972.

42. Farlow, J. O., C. V. Thompson, and D. E. Rosner. Plates of the dinosaur *Stegosaurus:* forced convection heat loss fins. *Science* 192: 1123–1125, 1976.

43. Flavahan, N. A. The role of vascular α_2-adrenoceptors as cutaneous thermosensors. *NIPS* 6: 251–255, 1991.

44. Fobelts, A., and A. P. Gagge. Rationalization of the effective temperature, ET*, as a measure of the enthalpy of the human indoor environment. *ASHRAE Trans.,* 94: 12–28, 1988.

45. Gagge, A. P. A new physiological variable associated with sensible and insensible perspiration. *Am. J. Physiol.* 120: 277–287, 1937.

46. Gagge, A. P. Effective radiant flux: an independent variable that describes thermal radiation in man. In: *Physiological and Behavioral Temperature Regulation.* edited by J. D. Hardy, J. Stolwijk, and A. P. Gagge. Springfield, IL: Thomas, 1970, p. 34.

47. Gagge, A. P. Partitional calorimetry in the desert. *Physiological Adaptations: Desert and Mountain.* New York: Academic, 1972, p. 23–51.

48. Gagge, A. P., and J. D. Hardy. Thermal radiation exchange of the human by partitional calorimetry. *Am. J. Physiol.* 23: 248–258, 1967.

49. Gagge, A. P., and Y. Nishi. Heat exchange between the human skin surface and thermal environment. *Handbook of Physiology: Reactions to Environmental Agents,* edited by D. H. K. Lee, H. L. Falk, and S. D. Murphy. Bethesda, MD: Am. Physiol. Soc., 1977, p. 69–92.

50. Gagge, A. P., A. C. Burton, and H. C. Bazett. A practical system of units for the description of the heat exchange of man with his environment. *Science* 94: 428–429, 1941.

51. Gagge, A. P., J. A. J. Stolwijk, and Y. Nishi. An effective temperature scale based on a simple model of human physiological regulatory response. *ASHRAE Trans.* 77: 247–262, 1971.

52. Gonzalez, R. R. Exercise physiology and sensory responses. In: *Bioengineering, Thermal Physiology and Comfort,* edited by K. Cena and J. A. Clark. New York: Elsevier, 1981, p. 123–144.

53. Gonzalez, R. R. Work in the north: physiological aspects. *Arct. Med. Res.* 44: 7–17, 1986.

54. Gonzalez, R. R., and K. Cena. Evaluation of vapor permeation through garments during exercise. *J. Appl. Physiol.* 58: 928–935, 1985.

55. Gonzalez, R. R., L. A. Berglund, and A. P. Gagge. Indices of thermoregulatory strain for moderate exercise in the heat. *J. Appl. Physiol.* 44: 889–899, 1978.

56. Gonzalez, R. R., M. J. Kluger, and J. D. Hardy. Partitional calorimetry of the New Zealand white rabbit at temperatures 5–35°C. *J. Appl. Physiol.* 31: 728–734, 1971.

57. Gonzalez, R. R., K. B. Pandolf, and A. P. Gagge. Heat acclimation and decline in sweating during humidity transients. *J. Appl. Physiol.* 36: 419–425, 1974.

58. Graichen, H., R. Rascati, and R. R. Gonzalez. Automatic dew point temperature sensor. *J. Appl. Physiol.* 52: 1658–1660, 1982.

59. Hammel, H. T. Regulation of internal body temperature. *Ann. Rev. Physiol.* 30: 641–710, 1968.

60. Hardy, J. D. *Physiological Problems in Space Exploration,* edited by J. D. Hardy. Springfield, IL: Thomas, 1964.

61. Hardy, J. D. Heat transfer. In: *Physiology of Heat Regulation and the Science of Clothing,* edited by L. H. Newburgh. Philadelphia: Saunders, 1949, p. 78–109.

62. Hardy, J. D. and E. F. Dubois. The technique of measuring radiation and convection. *J. Nutr.* 15: 461–475, 1938.

63. Hardy, J. D., A. T. Milhorat, and E. F. Dubois. Basal metabolism and heat loss of young women at temperatures from 22 degrees to 35 degrees Celsius. *J. Nutr.* 21: 383–404, 1941.

64. Havenith, G., R. Heus, and W. A. Lotens. Clothing ventilation, vapour resistance and permeability index: changes due to posture, movement, and wind. *Ergonomics* 33: 989–1005, 1990.

65. Heath, J. E. Reptilian thermoregulation: evaluation of field studies. *Science* 146: 784–785, 1964.

66. Henane, R. Acclimatization to heat in man: giant or windmill—a critical reappraisal. In: *Advances in Physiological Sciences, Vol 32: Contributions to Thermal Physiology,* edited by Z. Szelenyi and M. Szekely. Budapest: Pergamon, 1981, p. 275–284.

67. Hensel, H., K. Bruck, and P. Raths. Homeothermic organisms: heat exchange with the environment. In: *Temperature and Life,* edited by H. Precht, J. Christophersen, H. Hensel, and W. Larcher. New York: Springer-Verlag, 1973, p. 545–564.

68. Holbrook, K. A., F. Parker, G. A. Holloway, Jr., and K. K. Kraning. Structure and function of the skin. In: *Allergy Principles and Practice,* edited by E. Middleton, Jr., C. E. Reed, E. F. Ellis, et al., St. Louis: Mosby, 1988, p. 592–613.

69. Hynson, J., D. I. Sessler, B. Glosten, and J. McGuire. Thermal balance and tremor patterns during epidural anesthesia. *Anesthesiology* 74: 680–690, 1991.

70. Jessen, C., and G. Kuhnen. No evidence for brain stem cooling

during face fanning in humans. *J. Appl. Physiol.* 72: 664–669, 1992.

71. Johnson, J. M., G. L. Brengelmann, J. R. S. Hales, P. M. Vanhotte, and C. B. Wenger. Regulation of the cutaneous circulation. *Federation Proc.* 45: 2841–2850, 1986.

72. Kang, D. H., Y. S. Park, Y. D. Park, I. S. Lee, D. S. Yeon, S. H. Lee, S. Y. Hong, D. W. Rennie, and S. K. Hong. Energetics of wet-suit diving in Korean women breath-hold divers. *J. Appl. Physiol: Respir. Environ. Exercise Physiol.* 54: 1702–1707, 1983.

73. Keatinge, W. R. *Survival in Cold Water.* Oxford: Blackwell, 1969.

74. Keatinge, W. R. Direct effects of temperature on blood vessels: their roles in cold vasodilation. In: *Physiological and Behavioral Temperature Regulation,* edited by J. D. Hardy, A. P. Gagge, and J. A. J. Stolwijk. Springfield, IL: Thomas, 1970, p. 231–236.

75. Kerslake, D. McK. *The Stress of Hot Environments.* Cambridge: Cambridge University Press, 1972.

76. Kleiber, M. Trophic responses to cold. *Federation Proc.* 22: 772–774, 1963.

77. Kluger, M. J., R. R. Gonzalez, and J. A. J. Stolwijk. Temperature regulation in the exercising rabbit. *Am. J. Physiol.* 224: 130–135, 1973.

78. Kolka, M. A., and L. A. Stephenson. Cutaneous blood flow and local sweating after systemic atropine administration. *Pflugers Arch.* 410: 524–529, 1987.

79. Kolka, M. A., and L. A. Stephanson. Control of sweating during the human menstrual cycle. *Eur. J. Appl. Physiol.* 58: 890–895, 1989.

80. Kolka, M. A., L. A. Stephanson, P. B. Rock, and R. R. Gonzalez. Local sweating and cutaneous blood flow during exercise in hypobaric environments. *J. Appl. Physiol.* 62: 2224–2229, 1987.

81. Kraning, K. K. A computer simulation for predicting the time course of thermal and cardiovascular responses to various combinations of heat stress, clothing and exercise. In: *USARIEM Technical Report T13-91.* Natick, MA: U.S. Army Res. Inst. Env. Med., 1991, p. 1–49.

82. Kraning, K. K. Temperature regulation and the skin. In: *Physiology, Biochemistry, and Molecular Biology of the Skin,* edited by L. A. Goldsmith. New York: Oxford University Press, 1991, p. 1085–1095.

83. Kraning, K. K., and R. R. Gonzalez. Physiological consequences of intermittent exercise during compensable and uncompensable heat stress. *J. Appl. Physiol.* 71: 2138–2145, 1991.

84. Kraning, K. K., P. A. Lehman, R. G. Gano, and T. S. Weller. A non-invasive dose-response assay of sweat gland function and its application in studies of gender comparison, heat acclimation, and anticholinergic potency. In: *Thermal Physiology 1989,* edited by J. B. Mercer. Amsterdam: Elsevier, 1989, p. 301–307.

85. Kuhnen, G., and C. Jessen. Threshold and slope of selective brain cooling. *Pflugers Arch.* 418: 176–183, 1991.

86. Lambertsen, C. J. (Ed). *Underwater Physiology. Proceedings on the 4th Symposium on Underwater Physiology.* New York: Academic, 1971.

87. Lasker, G. W. Human biological adaptability. *Science* 166: 1480–1486, 1969.

88. Lewis, W. K. The evaporative of a liquid into a gas. *ASME Trans.* 4: 325–335, 1922.

89. Light, I. M., A. Avery, and A. M. Grieve. Immersion suit insulation: the effect of dampening on survival estimates. *Aviat. Space Environ. Med.* 58: 964–969, 1987.

90. Lih, M. M.-S. *Transport Phenomena in Medicine and Biology.* New York: Wiley, 1975, p. 1–531.

91. Lind, A. R. Prediction of safe limits for prolonged occupational exposure to heat. *Federation Proc.* 32: 1602–1606, 1973.

92. Livingstone, S. D., R. W. Nolan, J. Frim, L. D. Reed, and R. E. Limmer. A thermographic study of the effect of body composition and ambient temperature on the accuracy of mean skin temperature calculations. *Eur. J. Appl. Physiol.* 56: 120–125, 1987.

93. Lotens, W. A., and G. Havenith. Calculation of clothing insulation and vapour resistance. *Ergonomics* 34: 233–254, 1991.

94. Lotens, W. A., and E. J. G. van de Linde. Insufficiency of current clothing descriptions. In: *Int. Conference of Biophysical and Physiological Evaluation of Protective Clothing.* Lyon, France, 4–8, July, 1983, p. 1–13.

95. McLean, J. A. Loss of heat by evaporation. In: *Heat Loss from Animals and Man,* edited by J. L. Monteith and L. E. Mount. London: Butterworth, 1977, p. 19–31.

96. Mole, R. H. The relative humidity of the skin. *J. Physiol (Lond.)* 107: 399–411, 1948.

97. Monteith, J. L. *Principles of Environmental Physics.* London: Arnold, 1973.

98. Monteith, J. L. Specification of the environment for thermal physiology. In: *Heat Loss from Animals and Man,* edited by J. L. Monteith and L. E. Mount, London: Butterworth, 1977, p. 1–17.

99. Monteith, J. L., and G. S. Campbell. Diffusion of water vapour through integuments—potential confusion. *J. Therm. Biol.* 5: 7–9, 1980.

100. Mitchell, D. Convective heat transfer from man and other animals. In: *Heat Loss from Animals and Man,* edited by J. L. Monteith and L. E. Mount, London: Butterworth, 1977, p. 59–76.

101. Mitchell, D., and A. J. Van Rensburg. Assessment of clothing insulation: the problem and its solution using direct calorimetry on exercising men. *Arch. Sci. Physiol.* 27: 149–162, 1973.

102. Mitchell, J. W. Heat transfer from spheres and other animal shapes. *Biophys. J.* 16: 561–569, 1976.

103. Nadel, E. R. Temperature regulation and hyperthermia during exercise. *Clin. Chest Med.* 5: 13–20, 1984.

104. Nadel, E. R., R. W. Bullard, and J. A. J. Stolwijk. Importance of skin temperature in the regulation of sweating. *J. Appl. Physiol.* 31: 80–87, 1971.

105. Nadel, E. R., K. B. Pandolf, M. F. Roberts, and J. A. J. Stolwijk. Mechanisms of thermal acclimation to exercise and heat. *J. Appl. Physiol.* 37: 515–520, 1974.

106. Nadel, E. R., K. B. Pandolf, M. F. Roberts, and J. A. J. Stolwijk. Mechanisms of thermal acclimation to exercise and heat. *J. Appl. Physiol.* 37: 515–520, 1974.

107. Nelson, N., L. W. Eichna, S. M. Horvath, W. B. Shelley, and T. F. Hatch. Thermal exchange of man at high temperature. *Am. J. Physiol.* 151: 626–652, 1947.

108. Newburgh, L. H. (Ed). *Physiology of Heat Regulation and the Science of Clothing.* San Francisco: Saunders, 1949.

109. Nijland, M. J. M., and M. A. Baker. Effects of hydration state on exercise thermoregulation in goats. *Am. J. Physiol.* 263 (*Regulatory Integrative Comp. Physiol.* 32): R201–R205, 1992.

110. Nishi, Y., and A. P. Gagge. Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. *J. Appl. Physiol.* 29: 830–838, 1970.

111. Nishi, Y., and A. P. Gagge. Moisture permeation of clothing—a factor governing thermal equilibrium and comfort. *ASHRAE Trans.* 76: 137–145, 1970.

112. O'Connor, M. P., and J. R. Spotila. Consider a spherical lizard: animals, models, and approximations. *Am. Zool.* 32: 179–193, 1992.

113. Oohori, T., A. P. Gagge, and L. G. Berglund. Comparison of current two parameter indices of vapor permeation of clothing—as factors governing thermal equilibrium and thermal comfort. *ASHRAE Trans.* 90: 85–101, 1985.

114. Ozisik, M. N. *Heat Transfer: A Basic Approach*. New York: McGraw-Hill, 1985.

115. Pandolf, K. B., R. W. Gange, W. A. Latzka, I. H. Blank, K. K. Kraning, and R. R. Gonzalez. Human thermoregulatory responses during heat exposure after artificially induced sunburn. *Am. J. Physiol.* 262 (*Regulatory Integrative Comp. Physiol.* 31): R610–R616, 1992.

116. Pandolf, K. B., M. F. Haisman, and R. F. Goldman. Metabolic expenditures and terrain coefficients for walking on snow. *Ergonomics* 19: 683–690, 1976.

117. Porter, W. P., and D. M. Gates. Thermodynamic equilibria of animals with environment. *Ecolog. Monogr.* 39: 227–244, 1969.

118. Prosser, C. L. Perspectives of adaptation: theoretical aspects. In: *Handbook of Physiology. Adaptation to the Environment,* edited by D. B. Dill and E. F. Adolph. Washington, DC: Am. Physiol. Soc., 1964, p. 11–25.

119. Raman, E. R. Insulation versus counter current heat savings. In: *Proceedings of the Fifth Int. Conf. on Environmental Ergonomics,* edited by W. A. Lotens and G. Havenith. Maastricht, NL: 1992, p. 130–131.

120. Rapp, G. M. Convective mass transfer and the coefficient of evaporative heat loss from human skin. In: *Physiological and Behavioral Temperature Regulation,* edited by J. D. Hardy, A. P. Gagge, J. A. J. Stolwijk. Springfield, IL: Thomas, 1970.

121. Rapp, G. M. Convective heat transfer for nude man, cylinders and spheres at low air velocities. *ASHRAE Trans.* 79: 75–87, 1973.

122. Raymond, L. W. The thermal environment for undersea habitats. In: *Human Factors 1970,* Bulletin SF-70-3. New York: ASHRAE, 1970.

123. Riggs, D. S. Feedback, relationships, homeostasis. In: *The Mathematical Approach to Physiological Problems,* Cambridge, MA: M.I.T. Press, 1963, p. 95–107.

124. Roberts, M. F., W. P. Porter, and J. W. Mitchell. Central and peripheral components of metabolic response to cold in the hairless gerbil. *J. Appl. Physiol.* 37: 712–715, 1974.

125. Roberts, M. F., C. W. Wenger, J. A. J. Stolwijk, and E. R. Nadel. Skin blood flow and sweating changes following exercise training and heat acclimation. *J. Appl. Physiol.* 43: 133–137, 1977.

126. Robertshaw, D. Catecholamines and control of sweat glands. In: *Handbook of Physiology. Endocrinology,* edited by R. O. Greep and E. B. Astwood. Washington, DC: Am. Physiol. Soc., 1975, p. 591–603.

127. Robertshaw, D. Neuroendocrine control of sweat glands. *J. Invest. Dermatol.* 69: 121–129, 1977.

128. Roddie, I. C. Circulation to skin and adipose tissue. In: *Handbook of Physiology. The Cardiovascular System. Peripheral Circulation and Organ Blood Flow,* edited by J. T. Shephard and F. M. Abboud. Washington, DC: Am. Physiol. Soc., 1983, p. 285–317.

129. Rowell, L. B., P. R. Freund, and G. L. Brengelmann. Cutaneous vascular response to exercise and acute hypoxia. *J. Appl. Physiol.* 53: 920–924, 1982.

130. Sato, F., M. Owen, R. Matthes, K. Sato, and C. Gisolfi. Functional and morphological changes in the eccrine sweat gland with heat acclimation. *J. Appl. Physiol.* 69: 232–236, 1990.

131. Sato, K., and F. Sato. Individual variations in structure and function of human eccrine sweat gland. *Am. J. Physiol.* 245 (*Regulatory Integrative Comp. Physiol.* 14): R203–R208, 1983.

132. Satinoff, E. Neural organization and evolution of thermal regulation in mammals. *Science* 201: 16–22, 1978.

133. Sawka, M. N., R. R. Gonzalez, A. J. Young, R. C. Dennis, C. R. Valeri, and K. B. Pandolf. Control of thermoregulatory sweating during exercise in the heat. *Am. J. Physiol.* 257 (*Regulatory Integrative Comp. Physiol.* 26): R311–R316, 1989.

134. Scheuplein, R. J. Permeability of the skin. In: *Handbook of Physiology. Reactions to Environmental Agents,* edited by D. K. Lee, H. L. Falk, and S. D. Murphy. Bethesda, MD: Am. Physiol. Soc., 1977, p. 229–322.

135. Schmidt-Nielsen, K. *How Animals Work*. London: Cambridge University Press, 1972, p. 1–114.

136. Senay, L. C., D. Mitchell, and C. H. Wyndham. Acclimatization in a hot-humid environment: body fluid adjustments. *J. Appl. Physiol.* 40: 786–796, 1976.

137. Shitzer, A. Studies of bio-heat transfer in mammals. In: *Topics in Transport Phenomena,* edited by C. Gutfinger. New York: Wiley, 1975, p. 211–343.

138. Shitzer, A., L. A. Stroschein, W. R. Santee, R. R. Gonzalez, and K. B. Pandolf. Quantification of conservative endurance times in thermally insulated cold-stressed digits. *J. Appl. Physiol.* 71: 2528–2535, 1991.

139. Sibbons, J. L. H. Coefficients of evaporative heat transfer. In: *Physiological and Behavioral Temperature Regulation.* edited by J. D. Hardy, A. P. Gagge, and J. A. J. Stolwijk. Springfield, IL: Thomas, 1970, p. 108–138.

140. Simon, E. Paradigms and concepts in thermal regulation of homeotherms. *NIPS* 2: 89–93, 1987.

141. Simon, E., F.-K. Pierau, and D. C. M. Taylor. Central and peripheral thermal control of effectors in homeothermic temperature regulation. *Physiol. Rev.* 66: 235–300, 1986.

142. Skuldt, D. J., W. A. Beckman, J. W. Mitchell, and W. P. Porter. Conduction and radiation in artificial fur. In: *Ecological Monograph Series,* New York: Springer-Verlag, 1975, p. 549–558.

143. Sparrow, E. M., and R. D. Cess. *Radiation Heat Transfer*. Belmont, CA: Wadsworth, CA: 1966.

144. Stephenson, L. A., and M. A. Kolka. Cardiovascular and thermoregulatory effects of niacin. In: *Thermal Physiology,* edited by J. A. Mercer. Amsterdam: Elsevier, 1989, p. 279–284.

145. Stolwijk, J. A. J., and J. D. Hardy. Temperature regulation in man—a theoretical study. *Pflugers Arch.* 291: 129–162, 1966.

146. Tikuisis, P., and R. R. Gonzalez. Rational considerations for modelling human thermoregulation for cold water immersion. *ASHRAE Trans.* 8: 1–12, 1988.

147. Tikuisis, P., R. R. Gonzalez, R. A. Oster, and K. B. Pandolf. Role of body fat in the prediction of the metabolic response for immersion in cold water. *Undersea Biomed. Res.* 15: 123–134, 1988.

148. Van Dilla, M., R. Day, and P. Siple. Special problems of hands. In: *Physiology of Heat Regulation and the Science of Clothing,* edited by L. H. Newburgh. Philadelphia: Saunders, 1949, p. 374–386.

149. Vanggaard, L. Physiological reactions to wet-cold. *Aviat. Space Environ. Med.* 46: 33–36, 1975.

150. Varene, P. Computation of respiratory heat exchanges. *J. Appl. Physiol.* 61: 1586–1589, 1986.

151. Walsberg, G. E. Quantifying radiative heat gain in animals. *Am. Zool.* 32: 217–224, 1992.

152. Walsberg, G. E., G. S. Campbell, and J. R. King. Animal coat color and radiative heat gain: a re-evaluation. *J. Comp. Physiol.* 126: 211–222, 1978.

153. Webb, P. Body heat loss in undersea gaseous environments. *Aerospace Med.* 41: 1282–1287, 1970.

154. Winslow, C.-E. A., and L. P. Herrington. *Temperature and Human Life.* Princeton, NJ: Princeton University Press, 1949.

155. Winslow, C.-E. A., L. P. Herrington, and A. P. Gagge. Physiological reactions of the human body to varying environmental temperatures. *Am. J. Physiol.* 120: 1–22, 1937.

156. Wissler, E. H. An analysis of heat stress in hyperbaric environments. In: *Hyperbaric Diving Systems and Thermal Protection.*

edited by C. E. Johnson, M. L. Nuckols, and P. A. Clow. New York: Am. Soc. Mech. Eng., 1978, p. 53–74.

157. Wissler, E. H. Mathematical simulation of human thermal behavior using whole body models. In: *Heat Transfer in Medicine and Biology,* edited by A. Shitzer and R. C. Eberhart. New York: Plenum, 1985, p. 325–373.

158. Witherspoon, J. M., R. F. Goldman, and F. R. Breckenridge. Heat transfer coefficients of humans in cold water. *J. Physiol. Paris* 63: 459–462, 1971.

159. Woodcock, A. H. Moisture transfer in textile systems, Part I. *Text. Res. J.* 32: 628–633, 1962.

160. Yates, F. E. Dynamic regulation of mean arterial pressure: special role of renal resistances. *Federation Proc.* 42: 3143–3149, 1983.