Iberall, A.S., A.M. Schindler. On the physical basis of a theory of human thermoregulation. *J. Dyn. Sys., Measurement & Control, ASME* 95:68-75, 1973.

On the Physical Basis of a Theory of Human Thermoregulation[1]

A. S. IBERALL
Fellow ASME.

A. M. SCHINDLER
General Technical Services, Inc., Upper Darby, Pa.

This paper is concerned with the basis for thermoregulation under conditions of thermodynamic equilibrium. The thermoregulatory system can in principle only be understood through an appropriate integration of its physical, chemical, physiological, and neurological aspects. The common description of thermoregulation simply as a hypothalamic "feedback" control of a servomechanical "set point" has not led to an adequate self-contained model. Review of steady-state data (nude, resting human, low wind and humidity, 5–45 deg C ambient temperature T_a) indicates that, at the very least, there are physical-physiological aspects of thermoregulation not explained by a hypothalamic set point model. Rather, we can point to a wide spectrum of cyclic "thermodynamic engine" processes which regulate the various subsystems of the body [1].[2] The cooperative orchestration of all these processes produces a dynamic regulation of temperature, essentially as a self-regulation. This dynamic regulation produces an overall thermal equilibrium for the entire body.

To illustrate some unresolved equilibrium problems: Reported mean skin temperatures $\bar{T}_s$ versus T_a are incompatible with the physically determined skin-to-air transfer coefficient of about 7 kcal/m²/hr/deg C. At low T_a, either mean metabolism M must be higher or $\bar{T}_s$ must be lower, or the physical conductance must be rejected. One experimental test suggests that $\bar{T}_s$ is lower than commonly quoted (e.g., $\bar{T}_s$ = 26 deg C instead of 30 deg C at T_a = 20 deg C). Such discrepancies may arise if free convective transfer is suppressed or if experiments are not carried on to equilibrium. Change in metabolism, heat storage, and tissue temperature may be significant for several hours, requiring at least 3 hours of sample data for accurate equilibrium temperature measurements. At low T_a, there is no solid evidence for metabolic regulation; in the cold, equilibrium M rises only 20–30 percent, not 200–300 percent as proposed by some workers. At high T_a, the usual definition of mean tissue conductance [$\bar{C} = M/(T_c - \bar{T}_s)$] leads to nonphysically large $\bar{C}$ as $\bar{T}_s$ approaches deep body temperature T_c.

This paper is restricted to the physiological-physical modeling of the regulation of a variety of coupled fluxes (e.g., oxidative metabolism, evaporative flux, free and forced convective flux, fluid heat exchange) and potentials (e.g., internal and surface temperatures, evaporative phase changes). To resolve the foregoing difficulties we offer two hypotheses: (a) the body autoregulates a vital core; peripheral regions cool, e.g., extremity T_s drops toward ambient, the regulated core "contracts" longitudinally; (b) a significant portion of the evaporative heat loss may occur below the skin's surface. Adjustment of two parameters emerging from these hypotheses allows a consistent modeling of steady-state thermoregulation. We suggest that hypothalamic control is one component of regulation and operates at higher frequency (with 7-min period) than steady-state autoregulation (3-hr period).

Introduction

In this paper we undertake an examination of the physical factors that may lead toward a theory of human thermoregulation. We shall show discrepancies between generally accepted theory and results of generally accepted experiments. Our objective is to identify those discrepancies, explain

Contributed by the Automatic Control Division for publication (without presentation) in the JOURNAL OF DYNAMIC SYSTEMS, MEASUREMENT, AND CONTROL. Manuscript received at ASME Headquarters, December 19, 1972. Paper No. 73-Aut-J.

[1]Work supported by Army Research Office and NASA under Contract NASW-1815.

[2]Numbers in brackets designate Reference at end of paper.

Copies will be available until August, 1974.

Discussion on this paper will be accepted at ASME Headquarters until December 17, 1973

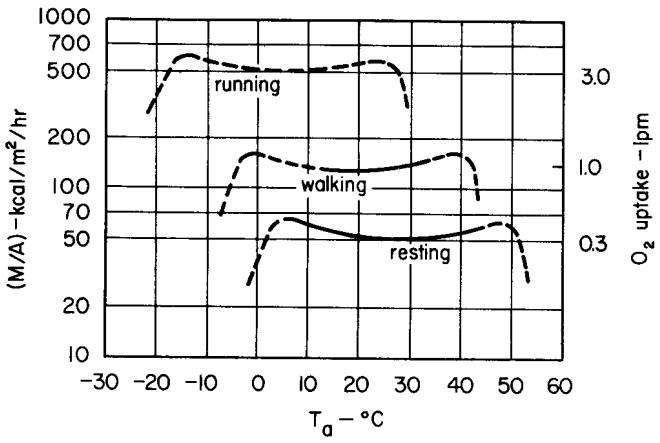

Fig. 1 Metabolism and oxygen uptake versus ambient temperature for various activity levels. The dotted portions represent reasoned guesses at the limits of survivability

why they have arisen, and propose ideas for their resolution. To make our major points as clearly as possible, attention is restricted to the *steady-state* (*equilibrium*) characteristics of the *nude, resting* human. This represents the simplest real case in which all relevant physical facets of the problem can be examined.

We will regard a physical steady-state to be achieved after it has existed for a few multiples of 7 min and can be sustained for more than 4 hr with only negligible variation in all time-averaged parameters. Experimentally, over a T_a range of 15 C to 35 deg C, at rest or at exercise, any change in conditions (e.g., ambient temperature or activity level) leads to an initial body transient which dominates other effects for the first 7 min. Even after the first 20 min, a system is still under the influence of the initial transient. At all times there is a high frequency cycle (2-min period) in metabolism and a low frequency cycle (7-min period). After 20 min the average metabolism is determinable with an accuracy of about 10–15 percent by taking 2–10 min averages of sample data. There is little subsequent change in metabolism, O_2 uptake, blood flow, pH and CO_2 levels. However, sample data averages are required for the next 3–5 hr to determine the average metabolism to 1–2 percent accuracy. For body temperatures (or "storage") a much slower transient exists. There are lower frequency cycles (30 min and $3^1/_2$ hr periods), and about 60–90 min are required for the initial transient to die out. After that the mean temperature at any station would not change very much, but the cycles would continue. A *minimum* of 4 hr is needed to achieve equlibrium in temperature and to avoid storage [1].

In its simplest form the problem of equilibrium thermoregulation can be formulated as follows: There are essentially two independent variables, a maintainable activity level of the subject (e.g., resting, walking) characterized by his metabolism M, and a measure of the environmental conditions, an effective "ambient" temperature T_a. We shall consider only the resting state of the nude human. The effective T_a is a combination of the air temperature, the radiative wall temperature, and the relative humidity. Air and radiative temperatures are here taken to be the same. Relative humidity plays a significant role only at high temperatures [2].

Given the independent variables, one seeks to model the observable dependent variables: deep-body or "core" temperature T_r, mean (over the body) skin temperature $\bar{T}_s$, and evaporative heat loss E. The essential physical requirement for equilibrium is that the metabolic energy produced within the body must be conducted or convected to the body's surface and thence conducted, convected, or radiated to the environment. There may be heat storage during the (transient) approach to equilibrium, but in the steady-state the storage must be zero. Failure to satisfy this condition implies continuing changes in time - averaged body temperatures, and thus a nonequilibrium situation.

Much published data relevant to thermoregulation are actually taken over short time scales (less than 1 hr). Many investigators, using such dynamic data, have proposed or implicitly assumed that thermoregulation originates in the hypothalamus, with that structure acting essentially as a thermostat with a specified "set-point" [3–8]. While this proposition may be valid for short-term regulation, one must keep an open mind as to possible roles for the hypothalamus or other structures in long-term equilibrium regulation [9].

One must search the literature rather carefully to find true equilibrium data. A brief summary of data [1c, 4, 10–17], which must be accounted for by a model of steady-state human thermoregulation, is provided in Figs. 1–3.

1 Deep-body temperature T_r is essentially constant, being almost independent of both T_a and M. For a resting nude human, it is near 37 deg C and varies only about 1 deg C as T_a varies from 0 C to 45 deg C. The variation of T_r at or near equilibrium is described by Nielsen [18, 19] and Lind [20]. There has been much discussion of the relative merits of various measures of deep-body temperature. The experimental data of Nielsen [19] and Saltin [21] show little difference in results for esophageal and rectal temperatures, and tympanic membrane temperature differs little [22, 23]. We make no distinction and simply refer to a deep-body or core temperature T_r.

Does the near constancy of T_r represent autoregulation of the system or the existence of a set-point temperature to which some feedback controller continuously adjusts the system? Hardy [5] noted our 1960 view [1*a*] in his statement that "the equations for heat transfer from the body and for body temperature equilibrium together with the control systems equations should provide the necessary material for a complete systems analysis of the physiologic temperature regulator. However, there is not sufficient information concerning the details of the regulator [of the set-point] to make this a worthwhile effort at this time" [24].

2 At a given activity level, M is nearly constant over most of the ambient temperature range compatible with life (Fig. 1). At either end of that range there is an approximately 20–30 percent rise in metabolism and then an abrupt drop, leading to death. We interpret those rises as energetic charges placed on the cardiovascular system, to provide the blood flow in a peripherally vasoconstricted state at low temperatures or to maintain profusely discharging sweat glands in a highly vasodilated state at high temperatures. Increase in activity level shifts this curve upwards and toward lower temperature.

We emphasize that a 20–30 percent change in M is likely the most that is found in the steady-state in cold environments. Larger transient changes and continuing cyclic changes occur, but the time-averaged mean value of M stays within about 30 percent of its value at "thermal neutrality" (i.e., minimum M

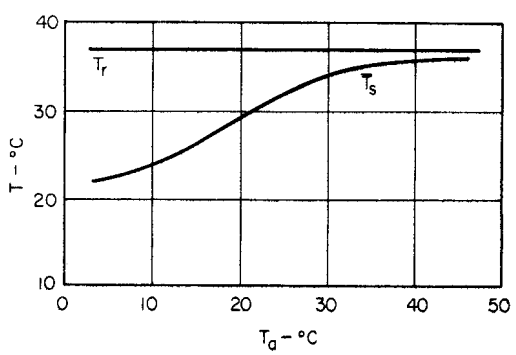

Fig. 2 Deep-body (T_r) and mean skin ($\bar{T}_s$) temperatures versus ambient temperature for a resting individual

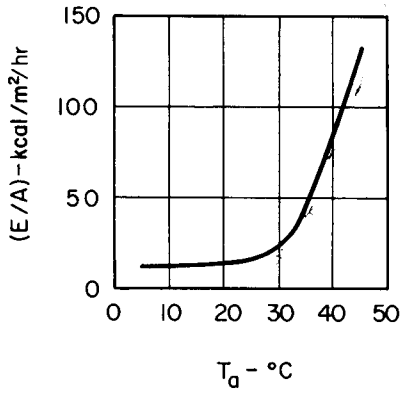

Fig. 3 Evaporative heat loss versus ambient temperature for a resting human (low humidity and low wind)

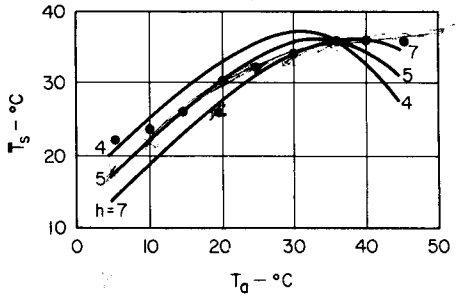

Fig. 4 Computed values of $\bar{T}_s$ (solid curves) compared to experimental values (dots) versus ambient temperature for various values of h (in kcal/m²/hr/deg C)

for a given activity level). Such limitations in variations of M have been measured by us [1c] and others [4, 14, 17]. A study by Raven, et al. [25] suggests that metabolism might have "leveled off" to an equilibrium value three to five times greater at 5 deg C than at 28 deg C. However, the study can be faulted for having been carried on for only two hours. Clearly there remains need for further experimental study to determine true equilibrium results.

Hardy and DuBois [12] left the impression that the body shivers in cold and perhaps doubles its metabolism. In fact, at *all* temperatures, metabolism continually cycles by a factor of nearly 2:1 [1c, 26, 27]. In the cold there may be transient shivering and continued bouts of shivering, but measurements show the same harmonic spectrum of metabolism. In the time-average, taken over a minimum of $3^1/_2$ hr, metabolism does not change much after the first 20 min, and shows only a moderate rise for the same activity (e.g., rest) at 0 deg C as compared to 30 deg C (5-hr data [1c]; 10-hr data [17]).

3 Mean skin temperature (an area weighted average) $\bar{T}_s$ versus ambient temperature is shown in Fig. 2 for a nude resting human. It ranges from $\bar{T}_s = 22$ deg C at $T_a = 5$ deg C to $\bar{T}_s = 37$ deg C at $\bar{T}_a = 45$ deg C.

4 At (local) T_s above 35 deg C sweating is profuse. Evaporative heat loss by a resting human (low humidity, low wind) is shown in Fig. 3. At rest, E ranges from 10 kcal/m²/hr at $T_a = 25$ deg C to perhaps 130 kcal/m²/hr at $T_a = 45$ deg C.

Thermoregulation is generally described [4, 5, 8] by three distinct physiological mechanisms and approximate temperature regimes: a "metabolic control" for $\bar{T}_s < 32$ deg C, a "vasomotor control" (dilation or constriction of the vasculature) for 32 deg C $< \bar{T}_s <$ 35 deg, and a "sudomotor control" (i.e., sweating) for $\bar{T}_s > 35$ deg C.

We can formulate the thermoregulation problem in a way which separates its physical from its physiological aspects. The function of the thermoregulatory system is to maintain a near constant T_r. In the steady-state the transfer of heat produced within the body takes place in two stages: transfer from a core, where most of the heat is generated, to the surface, and then transfer from the surface to the environment. The first stage requires determining physical heat transfer coefficients in terms of physiological mechanisms, for example, blood flow, its local distribution, sweat mechanisms, etc. The second transfer is an essentially physical process, with transfer coefficients depending on geometric factors, surface and ambient temperatures, properties of air, wind velocity, and whether the surface is wetted.

Surface-to-Air Transfer—A Physical Problem

Heat is transferred from the surface to the environment by radiation, conduction, free (i.e., no wind) or forced (i.e., a wind) convection. The total heat transfer coefficient h is some combination of the coefficients for these separate processes. For the *steady-state*, h is defined by

$$M - E = hA(\bar{T}_s - T_a) \tag{1}$$

where A is the total surface (DuBois) area. (About 10 percent of the metabolism and of the evaporative heat loss are lost by evaporation in the breath. Thus M and E in the foregoing (and henceforth) approximately represent $(M - 0.1M)$ and $(E - 0.1E)$. Only the net transfer across the skin determines the coefficient h.) Rewriting (1) gives

$$\bar{T}_s = T_a + \frac{M - E}{Ah} \tag{2}$$

In Fig. 4, $\bar{T}_s$ versus T_a is plotted for various values of h and compared with data (dots). (Here $A = 1.8m^2$, a typical value.) From Fig. 4, empirical values of h vary from about 7 kcal/m^2/hr/deg C at $T_a = 45$ deg C to about 3–4 kcal/m²/hr/deg C at $T_a = 5$ deg C.

As a prerequisite for a satisfactory theory of human thermoregulation, one must develop a satisfactory theory for h in order to correctly predict $\bar{T}_s$ as a function of T_a. We and others have done this before [4, 28, 29, 30]. However, here special attention is paid to some important details about the components of h which were overlooked in previous evaluations of the theoretical results.

Radiation [31, 32]. The skin is assumed to be a perfect black body—its emissivity is 0.99 [4]. According to Stefan's law, the body receives $s\,\tau_a^4$ kcal/m²/hr ($s = 4.94 \times 10^{-8}$ kcal/m²/hr/(deg K)⁴ and τ absolute temperature deg K) and emits $s\,\tau_s^4$ kcal/m²/hr. The effective radiating surface is about 3/4 of the DuBois area [4], and the net heat radiated from the body is

$$H_r = 0.75sA\,(\tau_s^4 - \tau_a^4).$$

Defining h_r via $H_r = h_r A\,(\bar{T}_s - T_a)$ and converting to deg C

$$h_r \approx 3.0\,[1 + 0.0055\,(T_a + \bar{T}_s)]\ \text{kcal/m}^2\text{/hr/deg C} \tag{3}$$

With the data of Fig. 2, h_r can be plotted (Fig. 5); it is in the range 3.3 to 4.4 kcal/m²/hr/deg C for 5 deg $< T_a <$ 50 deg C.

Free Convection [31, 32]. The heat transfer coefficient for free convection depends on the temperature difference between skin and air and on geometrical factors (e.g., size, shape, and orientation of the individual, and size of the chamber in which the experiment is performed).

Only the most simple free convection problems have been treated. However, there exists (cf. the bibliography in reference [31]) a large body of correlations which summarize results of free convection experiments in both laminar and turbulent flow regimes. Three dimensionless numbers are used in correlating such data.

The Nusselt number Nu compares the convective energy transport to the energy that would be transferred by simple

conduction. For a body characterized by a dimension D transferring heat into a medium of conductivity k, for which a heat transfer coefficient h is measured, Nu is defined by hD/k. If one can determine Nu, one can calculate h.

The Prandtl number Pr combines several gas properties. For air, Pr is essentially a constant (Pr = $c_p\mu/k$ = 0.71, where μ = viscosity, c_p = specific heat).

The Grashof number Gr represents the ratio of the buoyant force to the viscous force on an air "element," Gr = $gD^3\Delta T/\nu^2\tau$, where the acceleration of gravity g = 980 cm/sec^2, the kinematic viscosity ν = 0.14 cm^2/sec (air), $\Delta T = |\bar{T}_s - T_a|$, τ is absolute temperature, and D is the same characteristic dimension as in Nu. An important point is that if the individual is situated such that distances between him and the walls are smaller than D, then one must replace D in the Grashof number by that smaller dimension (reference [32], p. 181). Since wall proximity tends to decrease the buoyant force with respect to the inertial forces, the free convection may be substantially suppressed.

Dimensionless analysis of the fundamental transfer equations (reference [31], chapter 23) shows that Nu is a function only of the product Gr Pr. The form of the relationship depends on the geometry of the particular situation. We shall represent a person by a cylinder 30 cm in diameter and 180 cm in height.

Evaluating the Grashof number with τ = 300 deg K and Pr = 0.71 gives

$$\text{Gr Pr} = 3.4 \times 10^6\,\Delta T \quad D = 30 \text{ cm (horizontal position)}$$

and

$$\text{Gr Pr} = 7.3 \times 10^8\,\Delta T \quad D = 180 \text{ cm (vertical position)}$$

ΔT will generally be in the range 2 C to 10 deg C.

We are interested in both horizontal and vertical positions. For horizontal cylinders, the experimental correlations (reference [32], pp. 177, 180) for h_{free} in (kcal/m^2/hr/deg C) are

$$h_{free} = 1.57\,(\Delta T)^{1/4} \tag{4a}$$

for $10^3 <$ Gr Pr $< 10^9$, laminar flow, $\bar{T}_s > T_a$ and

$$h_{free} = 0.7\,(\Delta T)^{1/4} \tag{4b}$$

for $3 \times 10^5 <$ Gr Pr $< 3 \times 10^{10}$, laminar flow $\bar{T}_s < T_a$. For vertical cylinders, with D = 180 cm, the appropriate experimental correlation (reference [32], p. 173) is

$$h_{free} = 1.16\,(\Delta T)^{1/3} \tag{5}$$

for $10^9 <$ Gr Pr $< 10^{12}$, turbulent flow; $\bar{T}_s > T_a$. Qualifications on these expressions are given in [32].

Since the position of the subject is often not stated, or a variety of positions is allowed during the same experiment, the best we can do is to indicate likely values limiting the range of h_{free}. The largest values of h_{free} come from the horizontal laminar results, while the smallest come from the vertical turbulent results. When the skin surface is cooler than the environment, we have only the one horizontal laminar result. The values of h_{free} for the vertical and horizontal cylinders do not differ much, as a consequence of a nominal length to diameter ratio of about 6:1. Thus since two "extreme" orientations yield nearly the same values, those values of h_{free} should be fairly good for any orientation. The value of h_{free} averaged between the vertical (5) and horizontal (4a) results are used for $\bar{T}_s < T_a$ and the value of h_{free} from the horizontal result (4b) is used for $\bar{T}_s > T_a$ (Fig. 5).

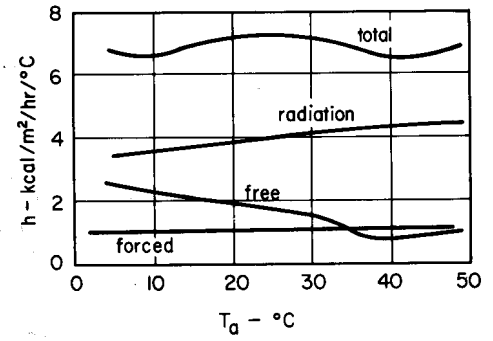

Fig. 5 Contributions to h from radiation, free convection, and forced convection as functions of ambient temperature

Forced Convection. Forced convection occurs when the air near the subject is driven by some potential gradient. For such cases Nu is correlated with the dimensionless Reynolds number, Re = DV/ν, where V is the air velocity. With the body represented by a cylinder 30 cm in diameter, Re is

$$\text{Re} = 215\,V \qquad (V \text{ in cm/sec}).$$

There is little difference between correlations for flow perpendicular or parallel to the cylinder axis. The result for perpendicular flow at low forced convection velocities, often prescribed in experiments, is

$$h_{forced} = 0.46\,V^{0.466},\ 0.2 < V < 20 \text{ cm/sec} \tag{6}$$

This is illustrated in Fig. 5 for V = 7.5 cm/sec. When both free and forced convections occur simultaneously, McAdams recommends (reference [32], p. 258) that the higher value be used. However, when h_{forced} and h_{free} are of the same order, as occurs in this problem, we prefer a "vector" addition, writing the total convection coefficient h_c

$$h_c = \sqrt{h^2_{forced} + h^2_{free}}$$

The total heat transfer coefficient h is the arithmetic sum of the radiative and convective contributions (Fig. 5). For low forced convective velocities ($V \approx$ 7.5 cm/sec), h nearly equals 6–7 kcal/m^2/hr/deg C across the entire ambient temperature range, regardless of the orientation of the subject.

These results are consistent with those of previous investigations. Colin, et al. [30], using $V \approx$ 35 cm/sec, obtained a forced convective contribution approximately twice ours and a total h = 10 kcal/m^2/hr/deg C. For lower wind conditions, other workers have also computed h = 6–7 kcal/m^2/hr/deg C.

Although these theoretical results may be generally accepted, they are apparently inconsistent with the experimental data obtained in thermoregulation experiments. In particular, Fig. 4 shows that h should vary between 3 kcal/m^2/hr/deg C at T_a = 5 deg C and 7 kcal/m^2/hr/deg C at T_a = 45 deg C. There is a clear discrepancy between the predicted h and the measured h. (Colin et al. [30] also noted that, in the cold, they measured h = 7 kcal/m^2/hr/deg C instead of an expected h = 10 kcal/m^2/hr/deg C [33].)

Actually, the theory presented here does permit values $h \approx$ 3 kcal/m^2/hr/deg C, but only under somewhat artificial conditions. If the free convection were suppressed by closely confining the subject, and if the wind velocity were "zero," and if the radiative wall temperatures were adjusted to reduce radiation, then h could equal 3 kcal/m^2/hr/deg C. However, in most experiments, major efforts were apparently made to maintain the temperature close to the nominal value and to maintain specified nonzero wind velocities. Still, there remains a variety of subtle ways in which the measured h can be affected.

If an experiment in a cool environment is not carried out over a long enough time, mean skin temperature may not have fallen to its final equilibrium value. The value of h will then appear to be smaller than it really is. If the subject is partly or lightly clothed, or if he is allowed to shield himself or protect himself by curling up, or if the space around him is insufficient to allow a free convective flow, then the measured value of h will be less than the calculated value. We suspect that the last possibility is the principal reason that experimental values of h are low compared to predicted values. In reviewing the experimental ar-

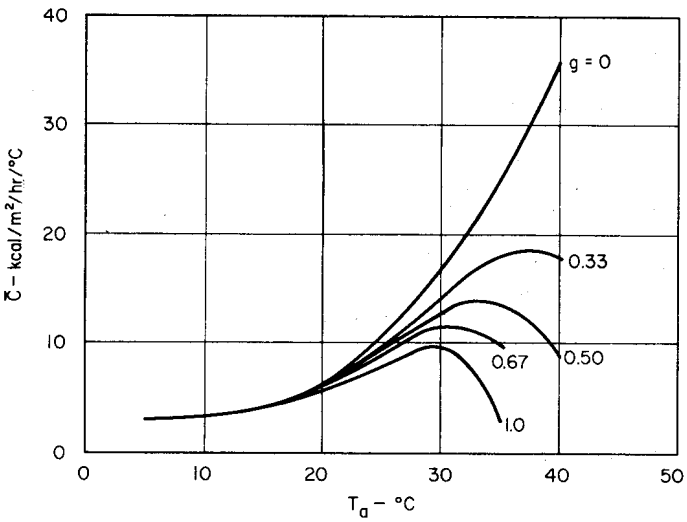

Fig. 6 Mean tissue conductance versus ambient temperature for different fractions g of subsurface evaporative loss

rangement of our own 1964 study, we now realize that we were not and could not have been making measurements with a fully established free convective flow. That requires 7 m of room height. Instead, vertical free convection was suppressed and we measured $h = 6.0$ kcal/m²/hr/deg C at $T_a = 29$ deg C and $h = 4.3$ kcal/m²/hr/deg C at $T_a = 20.5$ deg C. From what we know of others' work, it seems likely that they also suppressed free convection. Suppressing free convection reduces h by approximately 3 kcal/m²/hr/deg C in the cold (Fig. 5).

There is no similar discrepancy in warm environments because h_{free} becomes small (it depends on $T_a - \bar{T}_s$) and so, even if it is suppressed, only a slight difference is made in the total h.

We believe that this analysis provides reconciliation of theory with experiment. To test this one specific issue, we performed an experiment at $T_a = 19$ deg C, in an open (shaded) space, with a male subject wearing only a brief nylon bathing suit. After four hours a mean skin temperature of 26–27 deg C was recorded. This is about 3 deg C less than the value in Fig. 2 and it lies nearly on the line $h = 7$ kcal/m²/hr/deg C in Fig. 4. This datum strongly suggests that the theory of h established directly from physical principles is consistent with equilibrium measurements. There remains a need for further measurements of mean skin temperatures in ambient conditions below 20 deg C. They should be made under the following conditions: (1) nude or nearly-nude fully extended subjects, (2) a minimum 3–4 hour exposure, (3) low wind velocity (less than 5 cm/sec), (4) open space (i.e., distances to walls should be about 7 metres) around the subject. Then we could be confident that the data are suitable for comparison with the theory.

Heat Transfer Through the Skin—A Physical-Physiological Problem—Some New Ideas

The approximate linear associative dependence of mean body temperature $\bar{T}_b$ on $\bar{T}_s$ and T_r, at any operative equilibrium, namely

$$\bar{T}_b = aT_r + b\bar{T}_s, \tag{7}$$

and the gross linearity of tissue temperature with depth (see Bazett and McGlone in [4]) are generally regarded as sufficient basis for describing a peripheral body zone by a tissue conductance.

The mean tissue transfer coefficient, in the steady-state, is defined as [4, 16, 34]

$$\bar{C} = \frac{M/A}{T_r - \bar{T}_s} \tag{8}$$

This definition is inappropriate as $\bar{T}_s$ approaches T_r in a hot environment. Physically $\bar{C}$ cannot become too large. To avoid incompatibility with the second law of thermodynamics, some other energetic process must intervene. The conductivity of tissue should vary from about 18 kcal-cm/m²/hr/deg C when vasoconstricted (i.e., the conductivity of fat) to about 50 kcal-cm/m²/hr/deg C when vasodilated (i.e., the conductivity of water, or blood). Tissue conductivity is an active physiological parameter. With an average tissue thickness (from the surface to the middle of the muscle layer) on the order of 2 cm [4] the mean conductance $\bar{C}$ would then be expected to vary from 9 to 25 kcal/m²/hr/deg C. (In a warm environment there might be an additional increment to C arising from the countercurrent heat exchange of warm blood flowing near the surface and cooling there. An overestimated upper limit to this contribution is about 15 kcal/m²/hr/deg C [35]. The effect is not very important and will be neglected.)

Values of C computed from the data of Figs. 1–3 and found in the literature are considerably in excess of 25 kcal/m²/hr/deg C at high T_a [4, 36]. Thus the definition in (8) is inadequate. Some other mechanism must contribute to a consistent determination of $\bar{C}$.

In seeking such a mechanism, we first note that there are pools of fluid beneath the surface of the skin, e.g., in the sweat glands. We hypothesize that at least part, and under some conditions, all of the evaporative heat loss may take place beneath the skin's surface. (That is, the subcutaneous tissues may be able to function as a refrigerator. Whether this hypothesized function is identifiable with the sweat glands or some other functional unit remains to be determined.) The evaporated water vapor passes out through pores. When the water vapor partial pressure in the pores exceeds the saturation pressure, sweat seeps to the surface and evaporation takes place there. Gagge's concept of a variable wetted surface [4] then becomes applicable.

The subsurface evaporation proposed here is different from the water vapor diffusion suggested by Buettner [37] to account for part of the insensible evaporative loss which contributes only about 10 percent of E. We are suggesting that there is a mechanism which can generate considerably greater subsurface losses. Whether that process is a simple diffusion or some more complex mechanism is not clear.

Thus, we propose that in the definition of $\bar{C}$, M should be reduced by that fraction g of the evaporation E which takes place beneath the surface. (That is, for $g = 1$, all evaporation is subsurface and the skin is dry; for $g = 0$, all evaporation is from the surface, perhaps indicating a completely wetted surface.) Then

$$\bar{C} = \frac{(M - gE)/A}{T_r - \bar{T}_s} \tag{9}$$

With the data in Figs. 1–3, $\bar{C}$ versus T_a is plotted for various values of g, Fig. 6. The curve $g = 0$ corresponds to the previous definition of $\bar{C}$ and illustrates the unacceptably high values of $\bar{C}$. With g greater than zero, the values of $\bar{C}$ can be maintained within physiological-physical limits, at least at high ambient temperatures. In particular, $\bar{C}$ reaches a level near 18 kcal/m²/hr/deg C for g approximately 1/3. As a first approximation, that value for $\bar{C}$ is close enough to the expected limit of 25 to be reassuring of some *a priori* merit to the model. The physiological significance for a limiting value of $g = 1/3$ or thereabouts is not known.

The value of $\bar{C} = 3$ kcal/m²/hr/deg C measured at low T_a (Fig. 6) differs from the expected physiological lower limit of 9 kcal/m²/hr/deg C. The value of g plays an insignificant role in determining the value of $\bar{C}$ at low T_a since E is considerably smaller than M. Thus this new difficulty is not related to our hypothesis in the following.

The usual response to this problem is that, due to a "metabolic response" such as shivering, M increases enough in the cold so

that computed values of $\bar{C}$ would be at least 9 kcal/m²/hr/deg C. Clearly, M would have to increase by about a factor of 3 (cf. (8) or (9)) to raise $\bar{C}$ from 3 to 9 kcal/m²/hr/deg C. However, for a given activity level, M does not change more than about 30 percent over the survivable temperature range (Fig. 1). A more realistic mechanism is required.

It is found that after 2 or 3 hours in the cold, one's extremities (arms halfway to the elbow, legs halfway to the knee) blanch and their temperatures drop toward the ambient temperature (1*b*, *c*). Some longer time scale regulatory mechanism has responded to the cold and redirected blood flows, "abandoning" the extremities in order to maintain regulation over a vital core. Much less heat is then transferred to the environment across these unregulated areas (the temperature difference approaches zero, evaporative loss is negligible). To represent this idea in the heat transfer equations, let f be the fraction of the total area which remains regulated. Equations (1) and (9) become

$$M - E = fhA(T_s - T_a) \tag{10}$$

$$M - gE = fCA(T_r - T_s) \tag{11}$$

where T_s is the mean temperature over the regulated regions and C is the tissue conductance corresponding to T_s. The mean temperature over the whole surface is

$$\bar{T}_s = fT_s + (1 - f)T_a \tag{12}$$

Equations (10), (11), and (12) are proposed as representing steady-state thermoregulation of the body. The coefficient h is a physical factor with a value near 7 kcal/m²/hr/deg C. The coefficient C represents an active physical-physiological mechanism. The parameters f and g are physiological-physical parameters which characterize complex physiological mechanisms, whose effects are summarized quantitatively in this crude way but whose detailed physics and physiology are not yet understood.

To model the physical-physiological behavior of C, we note that the transition in tissue conductivity from 18 to 50 kcal-cm/m²/hr/deg C [35] takes place essentially linearly between local skin temperatures T_s of 28 C to 34 deg C (inferred in part from reference [4], p. 205, and reference [1*f*]). With the temperature increasing nearly linearly over the approximately 2 cm from the surface (T_s) to the middle of the muscle layer (T_r) (reference [4], p. 132) values of C can be derived (Table 1) [38].

Table 1 Peripheral tissue conductance versus skin temperature

T_s(°C)	C(kcal/m²/hr/°C)	T_s(°C)	C(kcal/m²/hr/°C)
25	13	31	20
27	16	33	22
29	19	35	24

With these values of C and with $h = 7$ kcal/m²/hr/deg C, one can determine the course of f and g in the range 5 deg C $< T_a <$ 45 deg C. Combining (10) and (11) to eliminate T_s gives

$$f = \frac{(C + h)(M - E)}{hC(T_c - T_a)} + \frac{E(1 - g)}{C(T_r - T_a)} \tag{13}$$

With M and E as known functions of T_a from Figs. 1 and 3, f versus g is a straight line for a given T_a. A family of such straight lines is shown in Fig. 7. The dashed line indicates the logical likely course of f and g between $T_a = 5$ deg C and $T_a =$ 45 deg C. (There is some uncertainty in the indicated course because the computation in the region $T_a > 30$ deg C involves the taking of small differences ($M - E$) between large quantities (M and E).) Fig. 7 shows clearly why it is not possible to construct a rational model with $g = 0$ (i.e., no subsurface evaporation) over the entire range of ambient temperatures. With $g = 0$, above $T_a \approx$ 30–31 deg C the body has no mechanism to maintain regulation; after all, f cannot exceed unity.

This model is a considerable oversimplification of the real system. Yet all that is required in principle to "complete" the problem is an equation describing how the blood is redistributed to different parts of the body, thus indicating which regions are regulated and which are abandoned. That relation would represent a physiological-physical theory for the parameter f.

Others have attempted more complex modeling of thermoregulation [39, 40, 41]. In general, the approach has been to assign values for the blood flow to the various parts of the body. The net result is to effectively assign a value for our parameter f.

We can also model $\bar{T}_b$

$$\bar{T}_b = aT_r + b\bar{T}_s \tag{7}$$

which is of considerable interest in the study of thermoregulation in the cold [6, 42]. Proposed empirical values of (a, b) range from (0.5, 0.5) to (0.9, 0.1), depending on T_a. Consider the body to be a cylinder with a diameter d_2 of about 30 cm, an unspecified length, a core of diameter d_1, and a core-surface thickness [$t = (d_2 - d_1)/2$] of about 2 cm. Assume that a fraction f of both core and surface length is regulated at T_c and T_s, respectively, and that the unregulated fraction $(1 - f)$ is near T_a. Then the volume-averaged $\bar{T}_b$ is easily calculated (assuming a linear temperature gradient between core and surface) to be approximately

$$\bar{T}_b = fT_r\left(1 - \frac{2t}{d}\right) + \bar{T}_s\frac{2t}{d} + (1 - f)T_a\left(1 - \frac{2t}{d}\right)$$

Here we have used $d_1 \approx d_2 = d$, $d_2^2 - d_1^2 \approx 4\,dt$, $d_2^2 + d_1^2 \approx 2d^2 + 4\,dt$, and $\bar{T}_s = fT_s + (1 - f)T_a$. Using numerical values in the foregoing gives

$$\bar{T}_b = 0.87\,fT_r + 0.13\,\bar{T}_s + 0.87\,(1 - f)T_a$$

Thus we can identify

$$a = 0.87\,f.$$

From (2), with $M/A \approx 50$ kcal/m²/hr, $E \approx 0$, and $h \approx 7$ kcal/m²/hr/deg C, $\bar{T}_s \approx T_a + 7$, so

$$b = 0.87\,(1 - f) + 0.13 = 1 - a$$

(There is a minor adjustment, neglected here, to these values because $\bar{T}_s = T_a + 7$.) From Fig. 7 we can get f versus T_a and then tabulate a and b versus T_a (Table 2).

Table 2 Coefficients *a* and *b* versus ambient temperature

T_a(°C)	f	a	b
> 33	1	0.87	0.13
30	0.82	0.72	0.28
25	0.65	0.57	0.43
20	0.58	0.52	0.48
15	0.50	0.44	0.56

In warm environments ($T_a >$ 32–33 deg C, $f = 1$) the limiting values of (a, b) are (0.87, 0.13) while in the cold the values change to (0.5, 0.5) at $T_a = 20$ deg C, $f = 0.6$. These theoretical values are in reasonable agreement with known empirical values.

In warm environments there is a shift in regulation from subsurface cooling to surface cooling, with a physical psychrometric process determining the ultimate limit to a capability for evaporative cooling. Above $T_a \approx 40$ deg C, the skin is completely wetted and evaporative losses are determined by physical and geometrical factors. The simplest derivation of evaporative loss is based on the similarity of the heat and mass transfer equation sets when those transfers take place in the same hydrodynamic fields, that is, when the process is psychrometric [31, 43]. The heat transfer coefficient for convection h_c (i.e., heat transfer = $h_c \times$ temperature difference) and the mass transfer coefficient

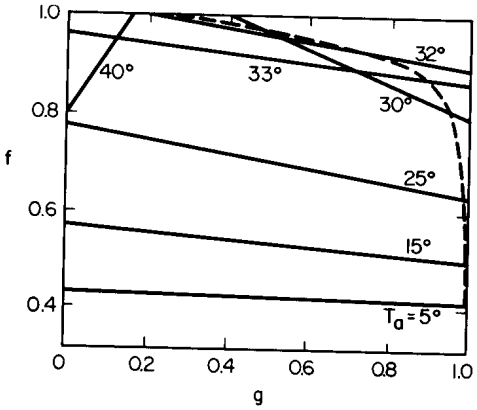

Fig. 7 Likely progression (dashed line) of f (fraction of body area regulated) and g (fraction of subsurface evaporative loss) versus ambient temperature

h_m (i.e., mass transfer = h_m × water vapor partial pressure difference) are related in the indicated units by

$$h_m \text{ (kcal/m}^2\text{/hr/mm Hg)} \approx 2\ h_c \text{ (kcal/m}^2\text{/hr/deg C)}$$

Fig. 5 shows that, for small wind velocity V = 7.5 cm/sec, h_c = 2 kcal/m²/hr/deg C and thus the expected value of h_m is 4 kcal/m²/hr/mm Hg [44, 45]. The maximum possible evaporative heat loss from a completely wetted surface depends on the water vapor partial pressure difference between skin and environment, determined by the T_a, T_s, and relative humidity. Demands on the body for heat loss in excess of this limit cannot be met, and the result is heat prostration.

More background discussion and details of this model can be found in [46].

References

1 Our earlier work in which dynamic time scales were investigated and discussed can be found in:

(*a*) Iberall, A., *Journal of Basic Engineering*, TRANS. ASME, Series D, Vol. 82, No. 1, Mar. 1960, pp. 96, 103; No. 3, Sept. 1960, p. 513.

(*b*) Iberall, A., Cardon, S., NASA Contractor's Rpt. CR-141, Jan. 1965.

(*c*) Iberall, A., Cardon, S., NASA Contractor's Rpt. CR-219, May 1965.

(*d*) Iberall, A., NASA Contractor's Rpt. CR-129, Oct. 1964.

(*e*) Bloch, E., Cardon, S., Iberall, A., Jacobowitz, D., Kornacker, K., Lipetz, L., McCulloch, W., Urquhart, J., Weinberg, M., Yates, F., NASA Contractor's Rpt. CR-1720, Feb. 1971.

(*f*) Iberall, A., *Curr. in Modern Biology*, Vol. 2, 1968 p. 140.

2 A more precise definition of our "ambient" temperature is not relevant to the point of this paper. For more details, see papers by A. Gagge at the 1939 and 1970 Temperature Symposia, in references [3 and 16].

3 *Temperature—Its Measurement and Control in Science and Industry*, Reinhold, New York, 1941.

4 Newburgh, L., ed., *Physiology of Heat Regulation and the Science of Clothing*, Saunders, Philadelphia, Pa. 1949.

5 Hardy, J., *Physiological Review*, Vol. 41, 1961, p. 521.

6 *Temperature—Its Measurement and Control in Science and Industry*, Vol. 3, Reinhold, New York, 1963.

7 Hammel, H., *Ann. Rev. Physiol.*, Vol. 30, 1968, p. 641.

8 Benzinger, T., *Physiol. Rev.*, Vol. 49, 1969, p. 671.

9 There is an analogous case in pressure regulation in the cardiovascular system. While the carotid baroreceptor may be involved in short term 5–15 sec regulation of blood pressure, it is not the determinant of long-term pressure regulation.

10 Winslow, C., Herrington, L., and Gagge, A., *American Journal of Physiology*, Vol. 120, 1937, p. 1.

11 Gagge, A., Winslow, C., and Herrington, L., *American Journal of Physiology*, Vol. 124, 1938, p. 30.

12 Hardy, J., and DuBois, E., *Journal of Nutrition*, Vol. 15, 1938, p. 477.

13 Adolph, E., and Molnar, G., *American Journal of Physiology*, Vol. 146, 1946, p. 507.

14 Scholander, P., Hammel, H., Anderson, K., and Loyning, Y., *Journal of Applied Physiology*, Vol. 12, 1958, p. 1.

15 Gagge, A., Stolwijk, J., and Saltin, B., *Environmental Research*, Vol. 2, 1969, p. 209.

16 Hardy, J., Gagge, A., and Stolwijk, J., eds., *Physiological and Behavioral Temperature Regulation*, Thomas, Springfield, Ill., 1970.

17 Irving, L., Andersen, K., Bolstad, A., Eisner, R., Hildes, J., Loyning, Y., Delms, J., Peyton, J., and Whaley, R., *Journal of Applied Physiology*, Vol. 15, 1960, p. 635.

18 Nielsen, M., *Skand. Arch. Physiol.*, Vol. 79, 1938, p. 193.

19 Nielsen, B., and Nielsen, M., *Acta Physiol. Scand.*, Vol. 56, 1962, p. 120.

20 Lind, A., *Journal of Applied Physiology*, Vol. 18, 1963, p. 51.

21 Saltin, B., and Hermansen, L., *Journal of Applied Physiology*, Vol. 21, 1966, p. 1757.

22 Nielsen, B., and Nielsen, M., *Acta Physiol. Scand.*, Vol. 64, 1965, p. 323.

23 Saltin, B., Gagge, A., and Stolwijk, J., *Journal of Applied Physiology*, Vol. 25, 1966, p. 679.

24 There is a rise in T_r from near 37 deg C at rest to about 39 deg C at a peak aerobic oxygen uptake of about 3 lpm. The variation in between is essentially linear. We regard T_r as being a regulated (i.e., compensated) variable, not a feedback controlled "set-point" variable. On the other hand, there is essentially no variation in T_r with ambient temperature in the range 5–30 deg C ambient. There is a much poorer degree of regulation at higher temperature. (The flat regulation also tends to break down at lower temperature at higher work levels.)

25 Raven, P., Niki, I., Dahms, T., and Horvath, S., *Journal of Applied Physiology*, Vol. 29, 1970, p. 417. Also see Wilkerson, J., Raven, P., and Horvath, S., *Journal of Applied Physiology*, Vol. 33, 1972, p. 451.

26 Goodman, L., "On the Dynamics of Pulmonary Ventilation in Quiescent Man," Case Inst. Tech. System Research Center Report No. SRC18-L-62-1, Cleveland, Ohio, May 1962; or *IEEE Trans. Biomed. Eng.*, Vol. 11, 1964, p. 82.

27 Lenfant, C., *Journal of Applied Physiology*, Vol. 22, 1967, p. 675.

28 Iberall, A., "Life Science Engineering," Third Quarterly Rpt. to U.S. Army Medical Research and Development Command, Nov. 1961.

29 Gagge, A., and Hardy, J., *Journal of Applied Physiology*, Vol. 23, 1967, p. 248.

30 Colin, J., Timbal, J., Guien, J., Boutelier, G., and Houdas, Y., Chapter 7 in reference [16].

31 Jakob, M., *Heat Transfer*, Vol. 1, Wiley, New York, 1949.

32 McAdams, W., *Heat Transmission*, 3rd ed., McGraw-Hill, New York, 1954.

33 They put the burden on the storage term in their equation, arguing that the discrepancy in h allowed them to correct assumed values of parameters used to evaluate the mean body temperature. However, their data were taken after a two-hour exposure and thus should vary little from true equilibrium data. The storage terms are negligibly small and the real discrepancy remains.

34 Fan, L., Hsu, F., and Hwang, C., *IEEE Trans. Bio-Med. Eng.*, Vol. BME-18, 1971, p. 218.

35 To include countercurrent heat exchange one would write (7) as

$$M = A\left[\overline{C} + \frac{c_p \Delta Q_b}{2}\right]\left[T_r - \overline{T}_s\right]$$

where c_p = specific heat capacity and ΔQ_b = increased blood flow to the skin. Thus there is a parallel shunt flow of heat which increases the effective tissue conductance because the hotter core blood is cooled in its transit through the capillary bed. The coefficient 1/2 arises from taking the average temperature difference in the boundary layer.

The increased boundary layer blood flow ΔQ_b is due to the cardiovascular charge for increased metabolism in the heat. A rise of 30 percent in metabolism may be correlated with a rise of about 10 percent in blood flow (reference [1*e*]). At about 5 lpm total blood flow, it would be difficult to assume more than 0.5 lpm for the increased tissue flow. However, preferring to overestimate the effect, we will assign ΔQ_b = 1 lpm. The heat capacity is essentially that of water. Thus we could expect, at most, an added tissue conductance of 15 kcal/m²/hr/deg C from counter-current exchange.

36 Cunningham, D., Chapter 22 in reference [16]. These experiments were done on the forearm where the tissue thickness is likely less than 2 cm. If with her Figs. 22–24 we estimate 1 cm tissue thickness and assume at an application temperature of 25-30 deg C heat is transferred only by simple conduction, then we would expect at most $C \approx 50 + 15 = 65$ kcal/m²/hr/deg C (see reference [35]). However, her figure shows C as high as about 120 kcal/cm²/hr/deg C.

37 Buettner, K., *Journal of Applied Physiology*, Vol. 6, 1953, p. 229.

38 Skin temperature has a great effect on the vasomotor response of tissues. Letting the muscle thickness be t_m and the skin thickness t_s, the average local bed temperature is

$$T = T_s + \frac{1}{2}\frac{T_r - T_s}{t_s + t_m/2}\, t_s$$

where T is the average temperature over the skin thickness t_s. Writing $N = t_m/t_s$ gives

$$T = T_s \frac{N+1}{N+2} + T_r \frac{1}{N+2}$$

N represents an anatomical quantity—the ratio of muscle thickness to skin thickness. Typically N might range from 1 to 3. In any case the larger contribution (at least half) to T comes from T_s. Thus the vasomotor response is quite sensitive to T_s.

39 Stolwijk, J., and Hardy, J., *Pfluegers Archiv. Ges. Physiology*, Vol. 291, 1966, p. 129.

40 Wissler, E., Chapter 27 in reference [16].

41 Stolwijk, J., Chapter 48 in reference [16].

42 For a brief review of the subject, up to 1969, see Minard, D., Chapter 25 in reference [16].

43 Iberall, A., Schindler, A., and Shannon, J., "General Systems Science—Part III," Contractor's Report to Army Research Office, Arlington, Va., Apr. 1970.

44 This result can be compared to the experimental results of Kerslake, et al. (Kerslake, D., and Waddell, J., *Journal of Physiology*, Vol. 141, 1958, p. 156; Clifford, J., Kerslake, D., and Waddell, J., *Journal of Physiology*, Vol. 147, 1959, p. 253). They used a velocity of 25 cm/sec and so their value of h_m should be $4(25/7.5)^{1/2} = 7.3$ kcal/m²/hr/mm Hg. They actually measured 7.8 ± 0.2 kcal/m²/hr/mm Hg.

45 Rapp, G., (Chapter 6 in reference [16]), with a similar calculation, apparently overlooked the point that psychrometric calculations apply only to a completely wetted surface (where the heat and mass transfers take place in the same hydrodynamic field). Thus, he slightly overestimated h_c/h_m by assuming a mean skin temperature of 33 deg C, too low for a completely wetted surface.

46 Iberall, A., Cardon, S., Schindler, A., Yates, F., and Marsh, D., "Progress Toward the Application of Systems Science Concepts to Biology," Contractor's Rpt. to Army Research Office, Arlington, Va., AD No. 750174, Sept. 1972.

Printed in U.S.A.