DEVELOPMENT OF MATHEMATICAL MODELS

OF ENVIRONMENTAL PHYSIOLOGY

NAS-9-9531

Final Report - B

Jan A. J. Stolwijk
John W. Mitchell
Ethan R. Nadel

PRICES SUBJECT TO CHANGE

John B. Pierce Foundation
290 Congress Avenue
New Haven, Connecticut 06519

Part I. Cardiopulmonary system and its response to environmental factors

Introduction

In the course of the development of a mathematical model of human thermoregulation which was described in a previous report (1), it was decided to simplify the linkage between thermoregulation and the cardiopulmonary system by assuming that the latter system responded instantaneously and perfectly to any challenge it encountered. As was already pointed out at that time, this simplification introduced severe errors under certain circumstances. Especially at the onset and at the end of severe exercise and in a cold environment during light exercise or moderate shivering, the model was a poor predictor of thermoregulatory and cardiopulmonary system responses. Some of the discrepancies between model prediction and experimentally observed responses were most likely to be due to the over-simplification of the cardio-vascular and respiratory system responses. As a result it seemed appropriate to evaluate the possibility of developing a more adequate description of the cardiovascular and respiratory systems with their control characteristics.

Existing literature

As a first step in the evaluation of cardio-vascular and respiratory system models, a very thorough survey of the existing literature was carried out with the help of research librarians at the Kline Science Library at Yale University. The library provided copies of all abstracts in Biological Abstracts which had combinations of words in the title indicating that the paper dealt with theoretical

or mathematical models or simulations of physiological control systems with special reference to respiration or circulation. After review of the abstracts a number were eliminated as being inappropriate and the remainder of the papers were secured in the form of xerox copies.

The results were uniformly disappointing. Circulation models often dealt with the hydrodynamics associated with the heart beat in which case there was no control loop at all; or in the case of models with control characteristics built in, there was a great deal of attention devoted to shock and hemorrhage, but there was no accounting of exercise. Similarly, in models of the respiratory control system the modeling effort was concentrated largely on hyperventilation due to CO_2 inhalation, with almost no efforts to account for increased ventilation in exercise.

In the absence of generally accepted frameworks for respiratory and cardio-vascular systems we had no alternative but to develop a new model, especially since it was essential that the new models interface with a well-developed model of the thermoregulatory system. Many features of the passive system for the thermoregulatory system are identical with those of the circulatory system.

The original plan of writing a comprehensive review of the existing models of circulation and respiration had to be abandoned in view of the considerable effort such a review would have required with very little useful yield to be anticipated for the current project.

The literature survey produced a general review which was delivered at the I.E.E.E. convention in New York City in 1971 as one of the invited papers in a symposium on the role of bio-engineering in medicine. The text of this paper follows below.

THE DEVELOPMENT AND USE OF SIMULATION MODELS IN MEDICINE

Jan A. J. Stolwijk, Ph. D.

John B. Pierce Foundation and Department
of Epidemiology and Public Health, Yale
University School of Medicine, New Haven,
Connecticut   06510.

Any time we think about some aspect of physiology or medicine, we form a mental image which is an abstraction of the real world. In this mental image we relate our observations to those of others and to a conceptual scheme which attempts to organize such observations in such a way as to explain them in a consistent manner. If we are to communicate this mental image, or test its implications against the behavior of the real-world system, this image must be expressed and it then becomes a hypothesis or model. Typically, we abstract the complex systems of the living world into much simplified conceptual models. We do not seem to think effectively about systems with more than three or four simultaneous and related variables, and in our experimental work we prefer to design the experiment in such a way that only a few variables are involved, keeping everything else as constant as possible. The sciences basic to medicine, such as physiology and biochemistry, produce large amounts of new observations on simpler and simpler subsystems. As Peterson (1965) pointed out, these observations and the conceptual models of their simple systems need to be reassembled into a much more complex model before we can derive the full benefit for the medical sciences responsible for clinical applications. Text books of medical physiology have to present such complex models and usually such texts will present a complex system as a collection of verbally and pictorially expressed sub-systems, with more verbal descriptions of the inter-relationships between such sub-systems.

Verbally expressed conceptual models of complex systems can still be very valuable. When Claude Bernard (1865) wrote his "Introduction à l'étude

de la médicine expérimentale", he provided an extremely important concept.
The constancy of the internal conditions with the implied regulation clarified
what was a puzzling and meaningless complexity of observations.

Usually we find that the implications of a conceptual model are most
precisely and unambiguously given by expressing the model in mathematical
language. A classical example of such a formulation was published by Otto
Frank (1899) in the form of a cardiovascular model. It is interesting to quote
from Frank's introduction:

> "In order to perform a quantitative analysis it is neces-
> sary to adopt a series of simplifications in the cardio-
> vascular system, without any alteration of essential
> characteristics. First of all we will ignore everything
> except the purely mechanical phenomena, and even these
> we wish to simplify somewhat. We will exclude all re-
> flected waves and assume that the pressure is the same
> in all parts of the arterial system...
>
> It goes without saying that it will be necessary to ex-
> perimentally prove that the real system behaves as this
> system and whether and to what extent the mathematical
> development of this system is applicable to the animal
> body. Perhaps it is not superfluous to emphasize that
> the procedure of simplification of the conditions is ap-
> plied very generally wherever natural laws are to be form-
> ulated. It is necessary to forego consideration of details
> and phenomena which are superfluous to the matter at
> hand, whenever one wants to express a law of nature
> in mathematical form. In other words one only studies
> the abstract mathematical model. The advantage of such
> a model over a normal physical model (which can be con-
> structed from pipes and tubing) is immediately apparent.
> It consists of the fact that the relationships between in-
> dividual properties and values are always clearly visible
> and can be expressed in simple sentences and that one
> only needs to change a constant in order to study another
> condition whereas this would necessitate rebuilding of the
> physical model..."

Frank's model has one important characteristic which is historically important: it deals with a linear problem with a readily available analytical solution so that his model was easy to evaluate. Unfortunately, almost any biological system one tackles has serious and essential non-linearities and general solutions are not available. Although numerical solutions can be obtained for particular cases, the amount of effort required was very discouraging.

With the advent of modern high speed computers and the increase in biologically-oriented people who have effective access to such machines, there has been a rapid increase in the appearange and use of mathematical models of biological systems.

An illustration of this is found in Figure 1 which attempts to measure the increase in the use of mathematical models in biological and medical research. Before 1955, the number of titles of papers abstracted in Biological Abstracts which referred to models was relatively constant at about one per ten thousand. By 1970, this had increased to more than 50 per ten thousand, with an approximately linear increase between 1960 and 1970.

A substantial part of this increase in mathematical modeling seems to be associated with the study of complex biological control systems, in the hope that control system theory as developed in the decades of the forties and fifties would contribute to a much improved insight into complex biological systems. A number of text books appeared from 1963 onward starting with texts by Grodins (1963) and Riggs (1963). In 1966, two more texts appeared by Milsum (1966) and by Milhorn (1966). Yamamoto and Brobeck (1965) and
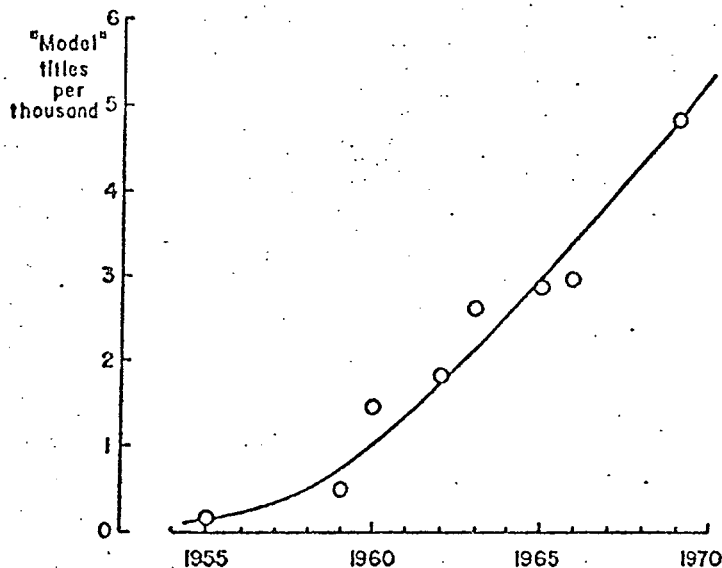
Figure 1 – Increase in the use of mathematical models in biology
and medicine, measured as the ratio of papers using the word
"model" in the title, to all papers listed in Biological Abstracts.

Kalmus (1966) edited texts on physiological regulatory systems and regulatory

systems in biology, respectively. Grodins (1963) provided an introduction to

the elements of control theory, and a mathematical description of two physio-

logical systems. He was unable to supply a strong link between the two halves

of his book. He was hopeful when he said:

> "Most biological control systems still await application of
>
> the conceptual approaches and analytic tools of the math-
>
> ematician, physicist and control engineer."

On the same page he tempers this enthusiasm:

> "Nevertheless, there is a certain feeling of dis-
> appointment in not finding a sufficiently general theory
> of non-linear systems available for our use. The na-
> ture of the beast is such that we may have to relax
> our expectations of ever finding such a theory."

Bellman (1962) as well as Grodins (1970) refer to what Bellman calls "math-

ematical experimentation" and what Grodins calls the "science servo system".

In either case they imply the need for continuous iteration between model and

experiment, the mathematical simulation of experiments. The implications of

these statements are very important for the status and the role of mathematical

simulation in physiology.

Without a usable general theory of complex non-linear systems the

contribution of mathematical modeling will not be essentially different from

what we have seen so far, although there will undoubtedly be further develop-

ment in quantity, sophistication and performance of such models. It is, how-

ever, unlikely that there will be a quantum jump in the significance of math-

ematical modeling in physiology.

How can we then assess the present role of mathematical simulation in

physiology, and can we identify newly developing or potential roles?

It would be very gratifying indeed if we could point to one or more ex-

amples of cases in which the application of mathematical simulation by itself

directly advanced the state of our knowledge of a given system, but this re-

viewer is unable to point out such a case. Given that the use of the simulation

approach is growing in absolute terms as well as relative to the total research

effort in the biological sciences (Figure 1), its proponents must perceive

benefits which are indirect. It is likely that an important part of the bene-

fit is highly personal. There are many authors, including this reviewer, who,

in describing the first simulation model they have arrived at, add a paragraph

which relates with some degree of enthusiasm the merits of the simulation ap-

proach and the new perspective acquired in the process of formulating the sim-

ulation model. As Adolph (1968) points out, personal intellectual development

is an important benefit of research and thus it can be a powerful motivational

force, and temporarily it could be the only motivation. Adolph makes another

point which is noteworthy in our context:

> "How did I know that my work on equilibrations and
> physiological regulations was not illusory?...I think
> my courage derived from an acute realization that in
> my new endeavor I was learning more than in previous
> periods of concentrated research; that I was now plan-
> ning broadly; that I gained much, first by virtue of the
> fact that there were few models to follow...My chief
> point here is that I clearly reaped a personal benefit
> even before my new effort could be recognized to be a
> contribution to physiology..."

He refers here to a change in his perspective to a conceptual systems approach

which occurred for him more than thirty years ago. If the benefits of mathe-

matical simulation of complex physiological systems are related to those en-

joyed by Adolph, then there are several important implications. The first of

these is quite obvious: a change in perspective will not by itself produce ad-

vances, but it will make such advances possible only for those who are actively

engaged in physiological research. A second consequence is also very important

and reduces the potential overall benefit of simulation applications: the experience

and the new perspective are difficult to transfer and they seem most effective as a personal experience. Substantial numbers of graduate students are now being exposed to courses in application of control theory to biological systems and it is reasonable to expect that they will contribute materially to the growth of simulation of biological systems. Their performance may answer the question whether this approach can be taught or must be experienced as a self-educational process.

Bellman (1962) sees mathematical experimentation as a promising enterprise in which very complex hypotheses can be tested via computer-aided solutions of mathematical models and tested against laboratory observations on the real system. Grodins (1970) stresses the same iteration between mathematical experiment and laboratory experiment where the mathematical model is constructed from the concepts abstracted from laboratory experiments, and where the implications of the model are obtained through the computer simulation of the same experiments. Comparisons will then suggest changes in system concepts as well as the most effective experimental challenge.

For the moment this mathematical experimentation in connection with simultaneous physiological research is the most important contribution of mathematical models perceived by this reviewer. There are, however, other applications which are beginning to develop as complex models become more sophisticated and more accurately represent a wide range of responses. Such models become a repository of accurately defined concepts combined with proven and experimentally verified constants and coefficients. When subjected to a definite set of conditions such a model will produce a reliable dynamic representation

of physiologic responses. At this point such models will produce benefits for others than the investigator who formulated and validated it. The Manned Spacecraft Center of NASA uses a model of physiological body temperature regulation (Stolwijk (1970, 1971)) as a part of their overall Apollo simulation program which evaluates interaction between man and his life support systems for various mission profiles and contingencies. It is likely that others with applied physiological problems can benefit similarly. Through preliminary evaluations with simulation models it should prove possible to reduce the number of applied experimental studies which tend to be conducted to evaluate the responses to particular environmental conditions.

As an example, the thermoregulation model will indicate the physiological strain resulting from exposure to a heat wave. It is beginning to be apparent that in California, for example, the number of excess deaths during a heat wave exceeds the number of deaths for any other single natural disaster in that state. It should be possible to warn the elderly population with cardio-vascular impairments who are mostly at risk and improve their survival rate.

It is likely that advance or real time simulation of surgical anesthesia based on body weight and fat contant would provide benefits in induction, maintenance and recovery of surgical patients.

I do not consider it likely that the inverse problem using simulation models will prove tractable. It is not likely that simulation will be very helpful in diagnosis of abnormal conditions in medical practice. The heuristic value of simulation models seems limited to conditions where interaction between model and experiment is close and iterative.

Efforts are under way in several laboratories to make use of mathematical models simulation physiological systems in combination with computer accessible literature data files. Such developments would be very helpful in assessing the mutual compatibility of new experimental data, data in the literature and computer predictions from mathematical models. Yamamoto and Raub (1967) proposed such a concept in order to exploit the archival characteristics of simulation models. Walters, et. al (1970) describe an information support system of a somewhat different nature which they are in the process of implementing. Their system is centered around a computer data file which allows retrieval of abstracted literature data for specifiable sets of conditions. This file can also accept locally acquired experimental data from a mobile computer controlled data acquisition system and the file can also produce data for simulation studies, and help in the comparison of new experimental data, literature data, and simulation results. These authors also mention the use of their system in a course on biological control systems.

References

1. Adolph, E.F. Prefatory Chapter: Research provides self-education. ~Ann. Rev. Physiol. 30: 1-14, (1968).

2. Bernard, Claude. Introduction à l'étude de la médicine expérimentale. Ballière, Paris (1865).

3. Bellman, Richard. Mathematical experimentation and biological research. Fed. Proc. 21: 109-111 (1962).

4. Frank, Otto. Die Grundforn des arteriellen Pulses. Z. f. Biol. 37: 483-526 (1899).

5. Grodins, F.S. Control Theory and Biological Systems. Columbia Univ. Press, N.Y. (1963).

6. Grodins, Fred S. Theories and models in regulatory biology. In: Physiological and Behavioral Temperature Regulation. edited by Hardy, J.D., Gagge, A. Pharo, and Stolwijk, J.A.J., Springfield, Ill., (1970), pp. 722-726.

7. Kalmus, H. ed. Regulation and Control in Living Systems. John Wiley and Sons, New York (1966).

8. Milhorn, H.T., Jr. The Application of Control Theory to Physiological Systems. W.B. Saunders, Co, Philadelphia, Pa. (1966).

9. Milsum, J.H. Biological Control System Analysis. McGraw-Hill, New York, (1966).

10. Peterson, L.H. Current advances and limitations in applying Claude Bernard's concepts to cardiovascular control and regulation. In: Les concepts de Claude Bernard sur le milieu interieur. Masson et Cie, Paris (1965) pp. 191-212.

11. Riggs, D.S. The Mathematical Approach to Physiological Problems. Williams and Wilkins, Baltimore (1963).

12. Stolwijk, J.A.J. Mathematical model of thermoregulation. In: Physiological and Behavioral Thermoregulation. edited by Hardy, J.D., Gagge, A. Pharo, and Stolwijk, J.A.J. Charles C. Thomas, Springfield, Ill., (1970) pp. 703-721.

13. Stolwijk, J.A.J. A Mathematical Model of Physiological Temperature Regulation in Man. National Aeronautics and Space Administration NAS 9-9531 Final Report (1971).

14. Walters, R.F., K.P. Brin, F. Roth, J.T. Morrison and C.R.G. Renoud. Information support systems for experimental investigation. Comput. Biol. Med. 1: 75-86 (1970).

15. Yamamoto, W.S. and J.R. Brobeck. ed. Physiological Controls and Regulation. W.B. Saunders Co., Philadelphia (1965).

16. Yamamoto, William S. and William F. Raub. Models of the regulation of external respiration in mammals: problems and promises. Computers and Biomed. Res. 1: 65-104 (1967).

A model of cardio-vascular adjustments during exercise

During the report year a number of experimental and theoretical studies were carried out in an effort to arrive at a description of what quickly appeared to be an extremely crucial and poorly understood link between the physiological regulation of body temperature, cardiac output and exercise: the control of peripheral resistance in the muscle at the onset of exercise. Change of the peripheral resistance in working muscle is the most important and the most common challenge to the cardiovascular control system. As in most complex control systems, it is very difficult to elucidate system structure from steady state results. On the other hand, most measurements of cardiac output or local blood flow require steady state conditions for useful results.

Our results so far are best presented in the form of the draft paper which follows. Our main reservation at the present time is that we have not yet adequately explored alternative formulations of the control system. In a later version we anticipate that this local control system will be able to present a well understood and physiologically plausible load to the blood pressure regulating system. Since the "time constants" are of the order of one minute or longer it was not deemed desirable to represent the individual heart beat, with all the computational complexity this would introduce into the simulation.

Muscle blood flow and oxygen uptake during

exercise transients - a theoretical study.

John W. Mitchell
Jan A. J. Stolwijk
Ethan R. Nadel

John B. Pierce Foundation Laboratory
and
Yale University School of Medicine
New Haven, Connecticut 06519

During the transition from rest to a steady level of exercise, many metabolic and circulatory adjustments occur. Oxygen uptake and muscle blood flow increase, inducing corresponding changes in cardiac output, heart rate and stroke volume. Increased pulmonary ventilation serves to maintain arterial oxygen tension essentially constant, and because of increased extraction of oxygen in the working tissues, oxygen tension in the veins draining the muscles is decreased. In the transition period less oxygen is taken into the body than is required by the working muscles. The energy necessary for muscular contraction during this period of oxygen deficit is obtained from oxygen stores, muscle phosphagen stores and by glycolysis leading to lactic acid formation (anaerobic metabolism). The relationship between oxygen uptake, cardiac output, muscle blood flow, anaerobiosis, etc. are rather well known during the steady state of exercise, but only to a limited degree in the transient. At present, there is no general theory available to quantitatively relate these terms in the transient. In this paper, such a model is developed for predicting the circulatory and metabolic responses during work.

Various investigators have attempted to describe the time course of oxygen consumption and heart rate during exercise transients in terms of exponential and similar functions. Henry (7) postulated that the increase in oxygen consumption in tissue with exercise proceeds as a first-order reaction. This leads to an exponential rise in oxygen consumption with time and a single characteristic time constant for muscle. The experimental results for oxygen consumption have been analyzed using curve-fitting techniques to determine the time constants (2,4,7). Cardus and Ziegler (3) have arbitrarily postulated weighted first order reactions and stochastic processes for oxygen consumption and developed

complex exponential relationships for the response. None of these models have been based on known physiological mechanisms.

Gilbert et al. (5,6) formulated a model in which the inflow, outflow, and change in storage of oxygen in a working muscle were related by the conservation of matter principle. The anaerobic conversion of chemical energy into work was not included, and there were no control or feedback equations relating blood flow or capillary resistance to oxygen uptake. Consequently, the variation in blood flow with time must be specified before oxygen uptake can be computed.

MODEL FORMULATION

In the development of the present model for a working muscle, a "standard" man is assumed, and the anthropomorphic and physiological data are given in Table 1. These values are consistent with the model developed by Stolwijk (15), with additions, as needed, to include cardiovascular variables.

. The model for muscle is based on the conservation of matter principle applied to the oxygen in a unit mass of working muscle under transient exercise. This principle relates the inflow of oxygen carried with the blood to the outflow, change in stores, and consumption.

$$\dot{m}_{bl} x_a = \dot{m}_{bl} x_v + dx_m/dt + \dot{V}_{O_2,m} \tag{1}$$

where $\dot{m}_{bl}$ is the blood flow rate (ml/min-100g_m), x_a and x_v are the arterial and venous oxygen concentrations in the blood flowing through the muscle (ml O_2/100 ml_{bl} or vol %), x_m is the oxygen stored in the muscle myoglobin (ml O_2/100 g_m), $\dot{V}_{O_2,m}$ is the oxygen consumed in the muscle by metabolic processes (ml O_2/min-100 g_m), and the subscript m denotes muscle.

The conservation of energy principle applied to the same muscle mass is

$$\dot{V}_{O_2,m} k = M_o + M + de_m/dt \tag{2}$$

Table 1

Anthropometric data for model man

Total body mass $= 74$ kg

Total muscle mass $= 32$ kg

Whole body	Upright Rest	Upright Maximal Exercise
$\dot{V}_{O_2}$ (L/min)	0.3	4.0
CO (L/min)	5.0	22.5
HR (beat/min)	60	180
SV (ml/beat)	83	124
x_a (vol %)	20	20
blood flow to body excluding muscle (L/min)	4.2	4.2

Muscle mass		
$\dot{V}_{O_2}$ (ml/min-100 g)	0.1	11.5
muscle blood flow rate (ml/min-100 g)	2.5	57.5

20

where k is a constant relating the conversion of oxygen to heat and work (assumed to be 4.85 cal/ml O_2), M_o is the basal metabolic rate (cal/min-100 g_m), M is the increase in metabolism due to work, and e_m is the chemical energy stored in the muscle that is converted anaerobically to heat and work during exercise.

Equations (1) and (2) can be combined to eliminate $\dot{V}_{O_2,m}$ and yield the controlling equation for the muscle processes

$$\dot{m}_{bl} x_a = \dot{m}_{bl} x_v + dx_m/dt + de_m/dt + (M_o + M)/k \quad . \tag{3}$$

Equation (3) describes the processes occuring in the working muscle mass. The whole body cardiovascular response to the changes in muscle blood flow during work is given by the following equation

$$CO = 4.2 \text{ L/min} + \dot{m}_{bl} \text{ (32 kg)} \tag{4}$$

where CO is the cardiac output (L/min), the muscle mass is 32 kg (see Table 1), and it is assumed in first approximation that the blood flow to the rest of the body excluding the muscles stays essentially constant at 4.2 L/min during exercise. In fact, shifts in the distribution of cardiac output do occur between rest and exercise, but these are small in comparison to changes in muscle blood flow and, for the purpose of this analysis, will be ignored.

The whole body oxygen uptake ($\dot{V}_{O_2}$) is given by

$$\dot{V}_{O_2} = 0.3 \text{ L/min} + \dot{m}_{bl}(x_a - x_v) \text{ (32 kg)} \tag{5}$$

where 0.3 L/min is the basal metabolic rate for the rest of the body excluding the muscle mass. The mixed arterio-venous oxygen concentration difference (A-V difference) is given by

$$\text{A-V diff} = \dot{V}_{O_2}/CO \quad . \tag{6}$$

The heart rate HR is given by

$$HR = CO/SV \quad . \tag{7}$$

where SV is the stroke volume (L). At rest, the stroke volume is taken as .083 L, and is assumed to increase to .124 L for all levels of exercise. These values and the constant value during exercise are consistent with the observations of several investigators (1,9,14,15).

Additional relationships among the variables in Eq. (3) are needed before solutions may be obtained. The oxygen concentrations in the hemoglobin and myoglobin are dependent on the oxygen tension (mm Hg), and these oxygen dissociation curves are given in Fig. 1 (from ref. 8). It is assumed that in the capillaries the flow is slow enough to allow complete equilibration of oxygen tension in the blood and the tissue so that the oxygen tension in the venous blood equals that in the muscle. This assumption couples the blood and muscle oxygen concentrations. The arterial oxygen tension and concentration are assumed to remain constant at all work levels at values of 95 mm Hg and 20 vol % respectively (21).

A control equation for the regulation of blood flow during exercise is postulated based on physiological observations. During rest, the overall arterio-venous difference is about 4 vol %, and the corresponding oxygen tension is about 50 mm Hg as indicated in Fig. 1, assuming that the A-V difference across the muscle is equal to the mixed A-V difference. The A-V difference increases rather rapidly to 10 vol % at low levels of exercise, and then rather slowly increases as the exercise level increases (14,15,21). It will be assumed that a P_{O_2} of about 25 mm Hg in the muscle capillaries, corresponding to an oxy-hemoglobin concentration of 10 vol %, is a value at which local control of muscle blood flow initiates.

The exact mechanisms by which peripheral vascular resistance in the working muscle is regulated during the rapid transient of exercise are not known at
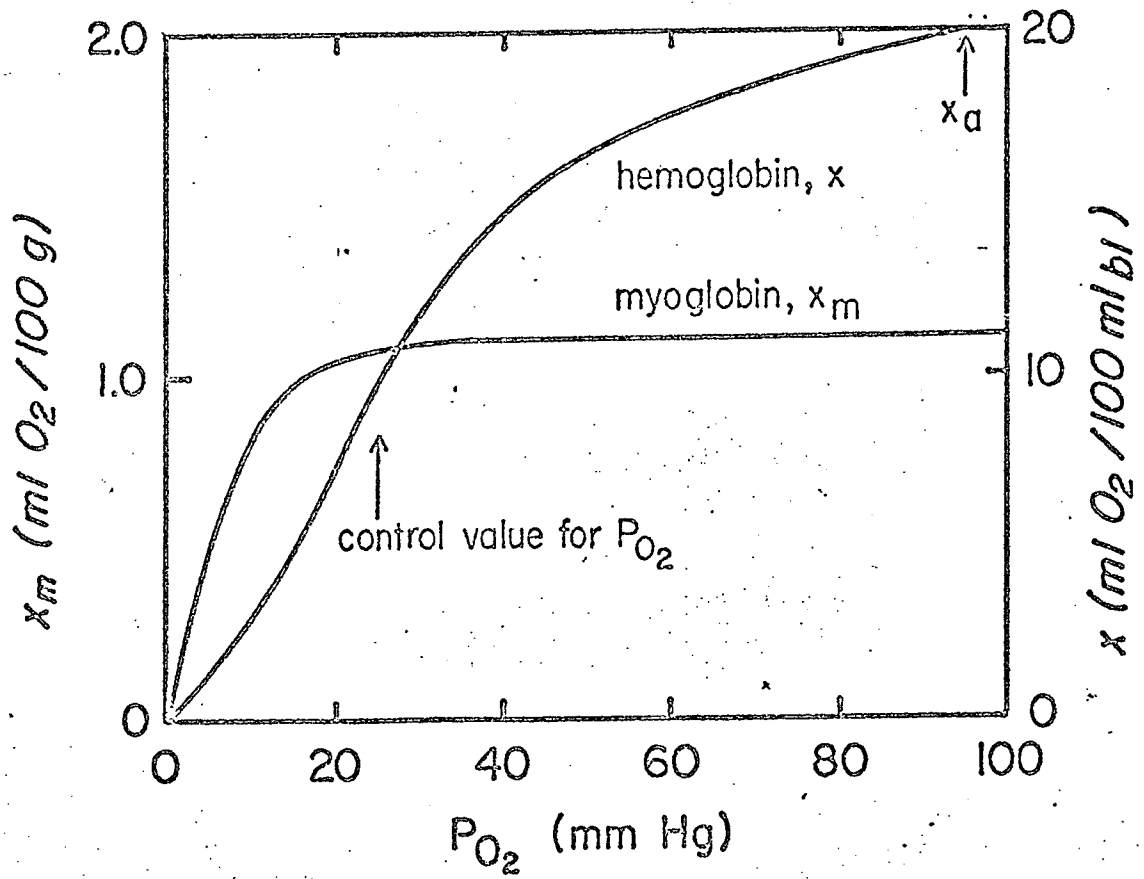
Figure 1. Oxygen dissociation curves for
hemoglobin and myoglobin.

present. However, it is known that the level of lactic acid concentration, P_{CO_2}, and pH as well as P_{O_2} can exert individual effects upon local blood flow. Since all of these quantities are metabolically related, and all are correlated during exercise (21), there is justification for using almost any of these as the control variable in a model. In this model, P_{O_2} is used as the control variable, although this should not imply that P_{O_2} alone acts to signal the need for increased oxygen supply.

Based upon these considerations, a proportional control equation for blood flow rate is assumed in which the blood flow rate increases in direct proportion to the decrease in P_{O_2} below the control value of 25 mm Hg

$$\dot{m}_{bl} - \dot{m}_{bl,o} = -A(P_{O_2} - 25 \text{ mm Hg}) \tag{8}$$

where $\dot{m}_{bl,o}$ is the blood flow rate in the resting muscle and A is the proportional control constant. The metabolic rate for resting muscle is taken to be 0.5 cal/min-100 g_m, and the corresponding oxygen consumption is 0.1 ml O_2/min-100 g_m (8,18). The blood flow rate in the resting muscle needed to sustain this metabolic rate at an A-V difference of 4 vol % is $\dot{m}_{bl,o} = 2.5$ ml/100 g_m.

The maximum blood flow rate in the working muscle is taken to be 57.5 ml/min 100 g_m, which corresponds to a $\dot{V}_{O_2}$ max of 4 L/min for the whole man. It is assumed that at this maximum work rate, the venous oxygen tension in the blood leaving the muscle is 0 mm Hg. With these assumptions, the relationship between blood flow rate and P_{O_2} given by Eq. (8) becomes

$$\dot{m}_{bl} = 2.5 \text{ ml/min-100 } g_m + \frac{2.2 \text{ ml}}{\text{min-100 } g_m\text{-mm Hg}} (25 - P_{O_2}) \tag{9}$$

This relation between $\dot{m}_{bl}$ and P_{O_2} is given in Fig. 2.

The amount of chemical energy stored in the muscle e_m is deduced from experimental measurements of the oxygen deficit in man during the transition from
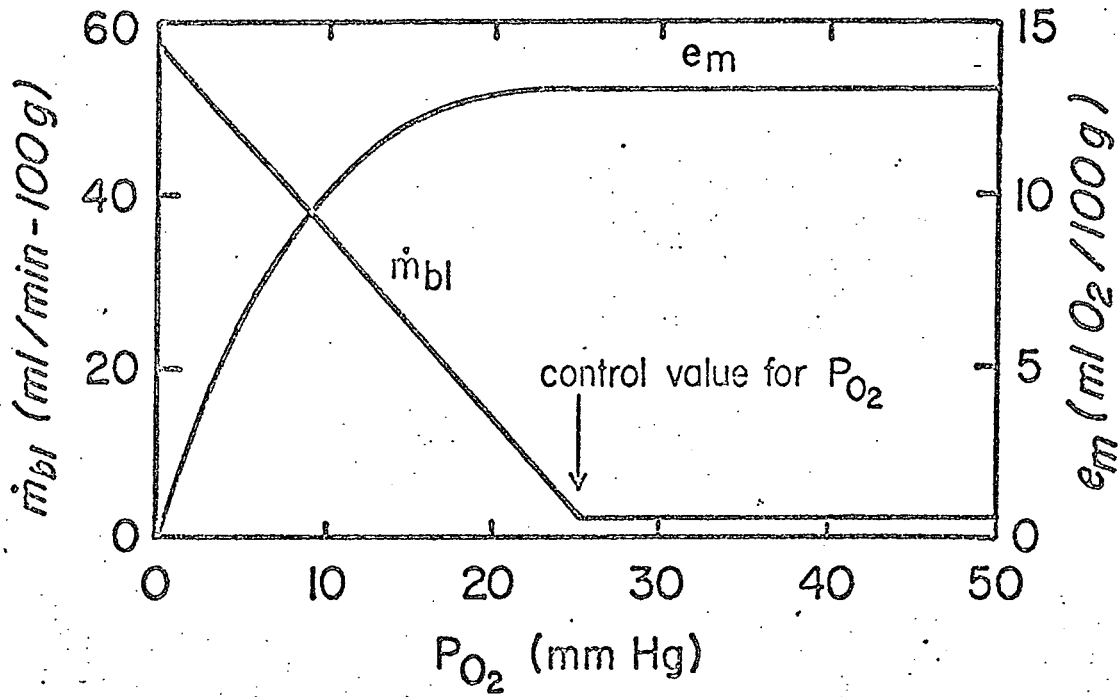
Control Relations

Figure 2. Muscle blood flow rate ($\dot{m}_{bl}$) and remaining oxygen
equivalent stores (e_m) as a function of tissue
oxygen tension.

rest to exercise. The values of oxygen deficit at various work loads as determined by several investigators (10,11,12,19,21,22) and by the present authors in another study (13) are given in Fig. 3. The original data were converted from a measured deficit in liters of oxygen to a per 100 g_m basis by making the simplifying assumption that the oxygen deficit was distributed uniformly over the muscle mass of the subject. A smooth curve was then drawn through these data as shown in Fig. 3.

The total oxygen deficit involves the depletion of oxygen stored in the venous blood and in the muscle myoglobin, the utilization of phosphagen stores and the production of lactate. The separate contributions of the myoglobin and blood oxygen stores were determined using the following relations

$$\text{(Myoglobin Deficit)} = (1.2 \text{ ml}/100 \text{ } g_m - x_m) \tag{10}$$

where x_m is a function of P_{O_2}, which in turn is a function of blood flow rate and oxygen uptake as given by Eq. (5) and (9). The computed myoglobin deficit is shown in Fig. 3.

The oxygen depletion between arterial and venous blood was computed assuming a systemic venous blood volume of 3.5 L. This deficit is then

$$\text{(Blood Deficit)} = \frac{3.5 \text{ L}}{32 \text{ kg}} (16 \text{ vol \%} - x_v) \tag{11}$$

where x_v is a function of P_{O_2}, blood flow rate, and oxygen uptake. This relation is also shown in Fig. 3.

The energy obtained from anaerobic sources was computed as the difference between the total oxygen deficit and the sum of the deficits from myoglobin and blood. This difference, which is e_m, is plotted in Fig. 2 as a function of P_{O_2}. In the model, it is assumed that the chemical energy release occurs so rapidly that the instantaneous energy level e_m is given by the instantaneous value of P_{O_2};
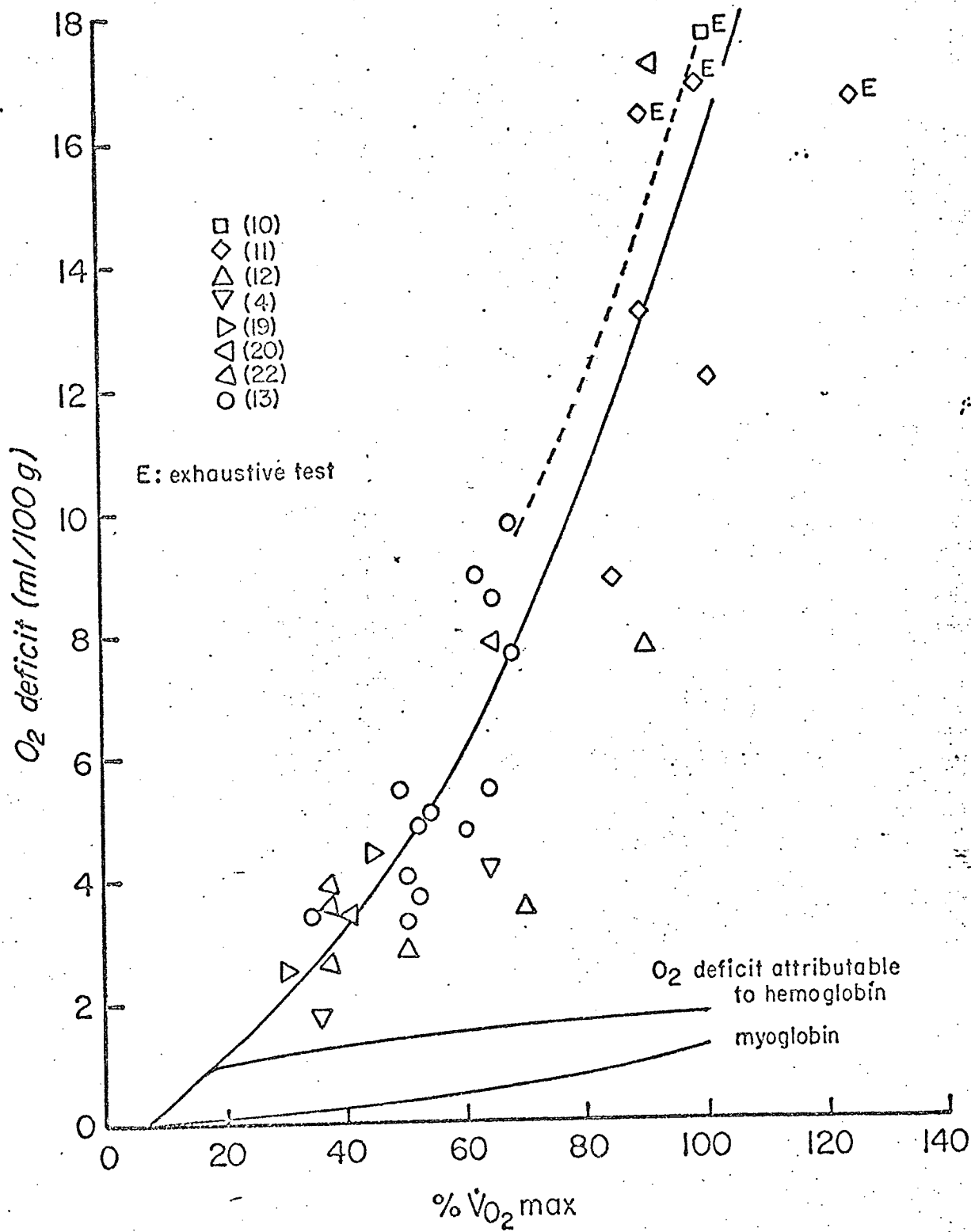
Figure 3. Relation between total, hemoglobin and myoglobin oxygen deficits, and percentage of maximum aerobic capacity.

the relation in Fig. 2 is assumed to be valid at any instant of time. This is consistent with observations of markedly increased lactate levels at the start of exercise (10,11,21). The calculations by Wasserman (21) also support the assumption that lactate production contributes directly to the deficit during the early phases of exercise.

In summary, Eq. (3) and (5) represent the governing equations for the local muscle processes during exercise. It is assumed that blood flow rate, oxygen concentration in the hemoglobin and myoglobin, and the chemical energy stored in the muscle are functions of the instantaneous oxygen tension as presented graphically in Fig. 1 and 2. Oxygen tension has been arbitrarily chosen as the indicator of the chemical state of the muscle. The whole body cardiovascular and metabolic responses to exercise are given by Eqs. (4), (5), (6) and (7).

Equations (3) and (5) cannot be solved explicitly because of the difficulty in obtaining experimental values for the functions $\dot{m}_{bl}$, x_m, and e_m. Solutions for various work levels were obtained by rewriting Eq. (3) in finite difference form and rearranging

$$-\dot{m}_{bl}(x_a - \bar{x}_v) + \frac{x_m' - x_m}{\Delta t} + \frac{e_m' - e_m}{\Delta t} + (M_o + M)/k = 0 \qquad (12)$$

where the primes denote the future values for the time step and the bars denote the average value over the time step. To determine the future values, Eq. (12) was written as

$$R = (x_m' - x_m) + (e_m' - e_m) - \Delta t\, \bar{\dot{m}}_{bl}\, (x_a - \bar{x}_v) \qquad (13)$$

where R is the residual and must be equal to zero for each time step. A computer program was written to solve Eq. (13) by iteration for each time step.

RESULTS

1.   Steady State Results:

The general model equations developed in the previous section are first solved for the steady state by setting the time derivatives in Eq. (3) equal to zero. Equation (3) becomes the steady state energy balance equation

$$\dot{m}_{bl}(x_a - x_v) = (M_o + M)/k \quad . \tag{14}$$

Equation (8) for blood flow rate control and Fig. 1 for the oxygen concentrations are employed to solve Eq. (14) for different metabolic rates. Equations (4), (5) and (7) are used to compute the total CO, $\dot{V}_{O_2}$ and HR. The muscle and total oxygen consumption are shown in Fig. 4 as functions of P_{O_2}. These results, together with the relations in Fig. 1 and 2, show that increased blood flow is the major factor in facilitating the increase in oxygen consumption. The A-V difference can only increase by a factor of 5 at most over resting values, while flow can increase up to a factor of 20. The total oxygen consumption reflects the increase in muscle oxygen consumption.

The steady state cardiac output and heart rate as predicted by the model are presented in Fig. 5 in comparison with the experimental results of several investigators. The measured cardiac outputs include not only muscle blood flow but an increased skin blood flow over resting levels as a response to increased body temperatures in exericse. The skin blood flow is probably on the order of 10 to 15 percent of the total cardiac output. The present model results do not take the increased skin blood flow into account, but only predict the increased flow to the working muscles. The model results agree with the observations within this difference.

2.   Transient Results:

The model results for the time course of blood flow rate and oxygen tension

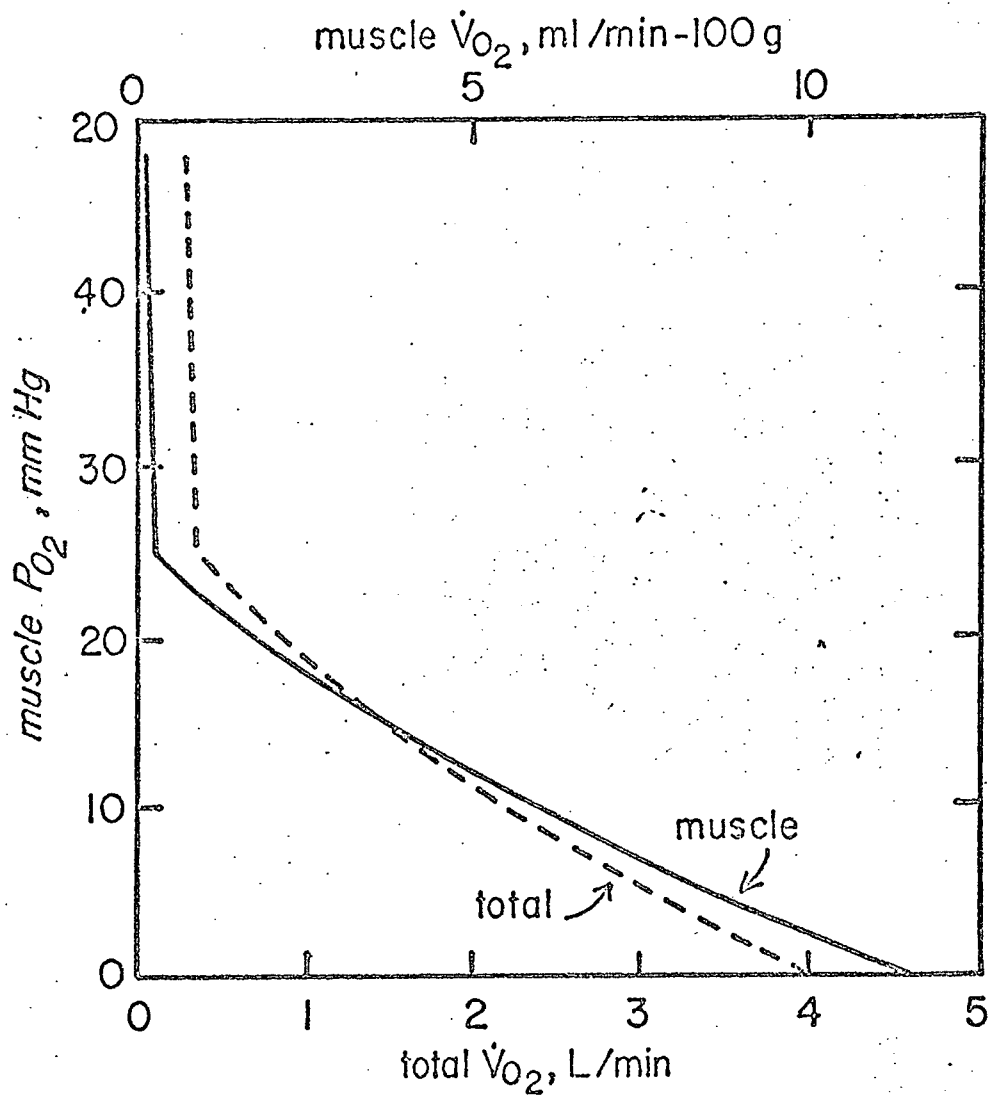# Steady State Results for Oxygen Consumption

Figure 4. Tissue oxygen tension in the muscle as a function of total oxygen uptake, and of muscle metabolism.
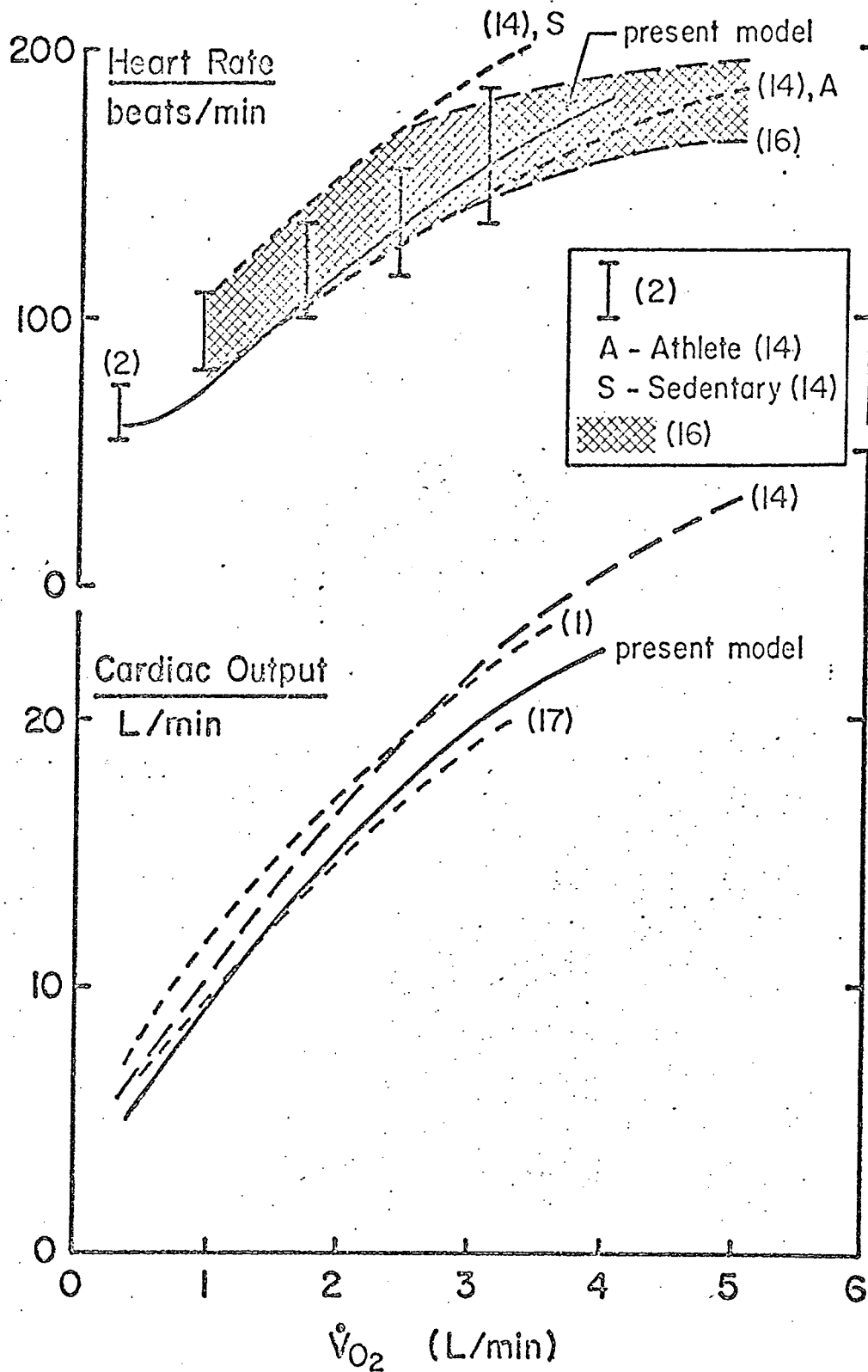
Figure 5. Steady state relationships between cardiac output, heart rate and rate of oxygen uptake.

at several metabolic rates are presented in Figs. 6 and 7. Blood flow rate and P_{O_2} change very rapidly during the transient. For low work levels (M less than about 2 ml/min-100 g_m or 20 M_o), equilibrium values are reached in less than one minute. At higher work levels, most of the transient is over in the first minute, but a steady state is not reached for several minutes (up to 8 minutes at a work level of 10 ml/min-100 g_m). The metabolic rate of 11.5 ml/min-100 g_m represents the maximum steady state level for the muscle, in the present model formulation.

There are no complete data available for a comparison with the predicted results of Fig. 6 and 7. The muscle responses have been extended to predict heart rates, and these are compared with measurements made during the transient onset of exercise in Figs. 8, 9, 10 and 11. The measured values were obtained from an electrocardiogram (13), and represent heart rate values averaged over 6 second time periods. Predicted rates were also computed using a higher stroke volume (.15 L) than given in Table 1. This higher value is representative of athletes (14) and appears to be more appropriate for subjects JM, EN, and BS than the lower value. The model and experimental results agree within about 10% during the first minute.

In Fig. 12, the model results are compared to the measurements of Jones et al. (9), who calculated cardiac outputs from instantaneous arterial pressure measurements. The model results predict a slightly faster response than measured. Their experimental values taken at rest appear to be high (7.5 to 9 L/min), which suggests that some artifact has been introduced by the experimental technique. The model and data disagree by less than 10 percent, which is about one standard deviation for the data.

The model was used to predict the response during the transient from one
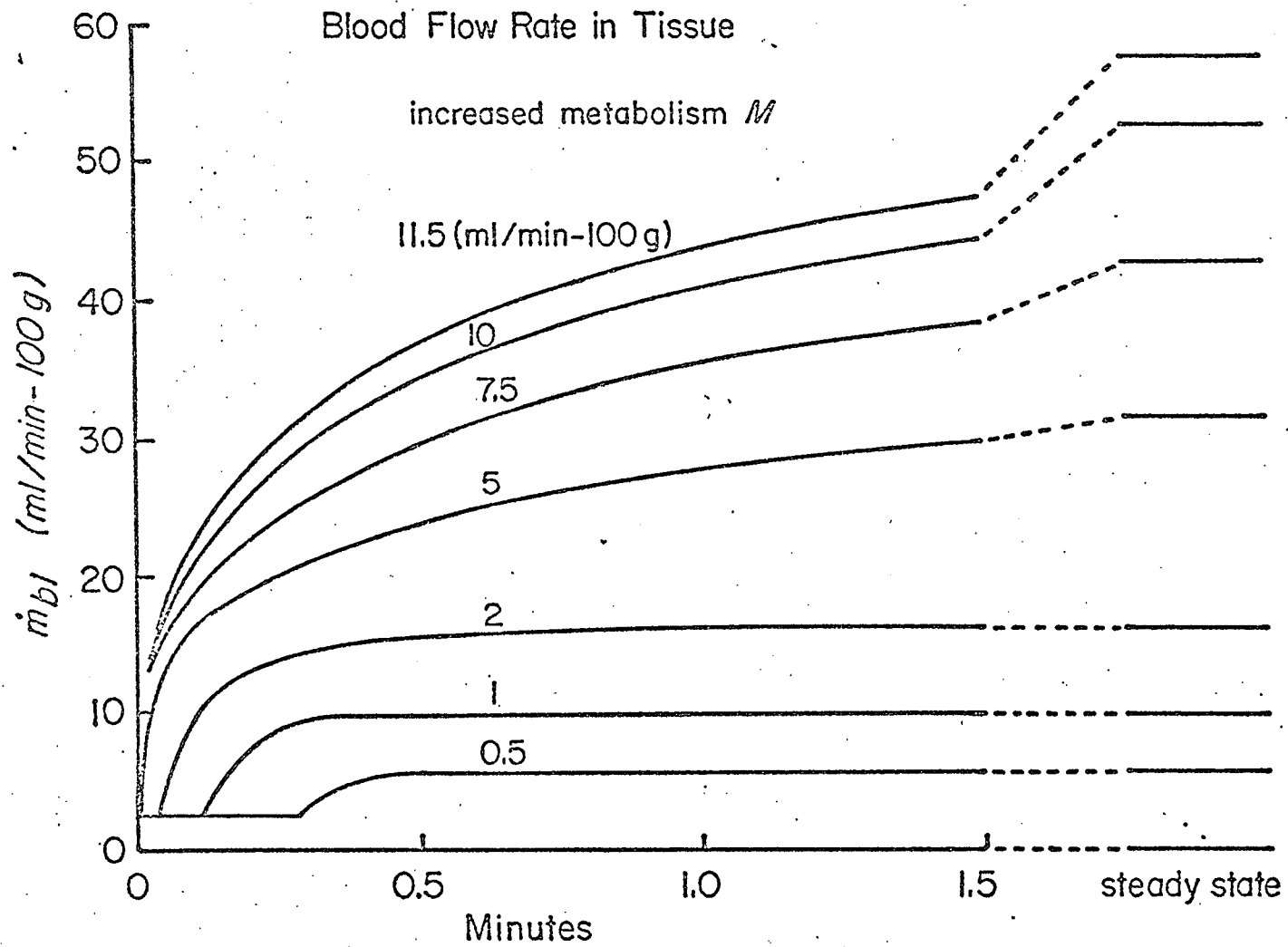
Figure 6. Model predictions of muscle blood flow rates after onset of exercise at various levels.
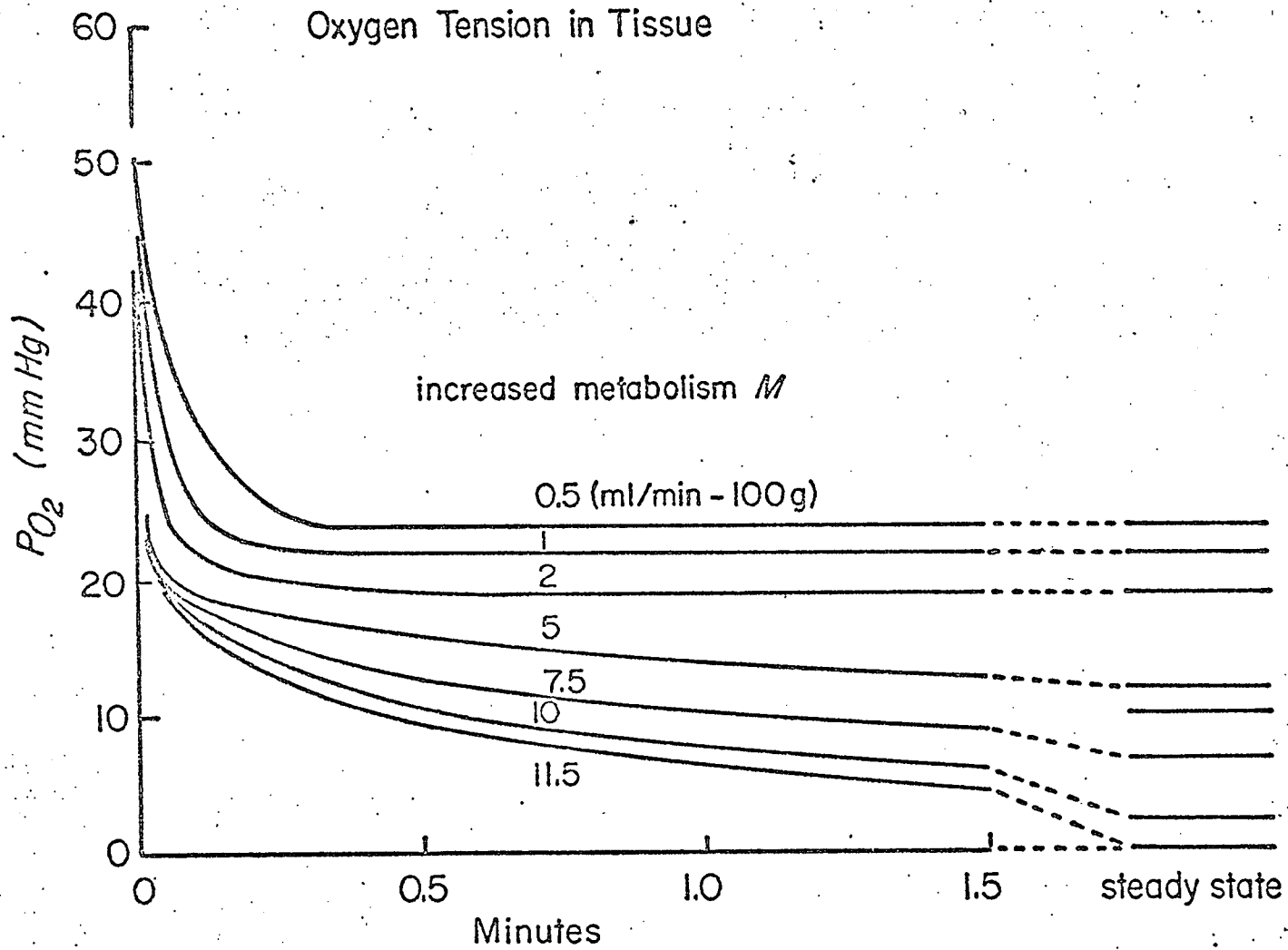
Figure 7. Model predictions of muscle tissue oxygen tension after onset of exercise at various levels.
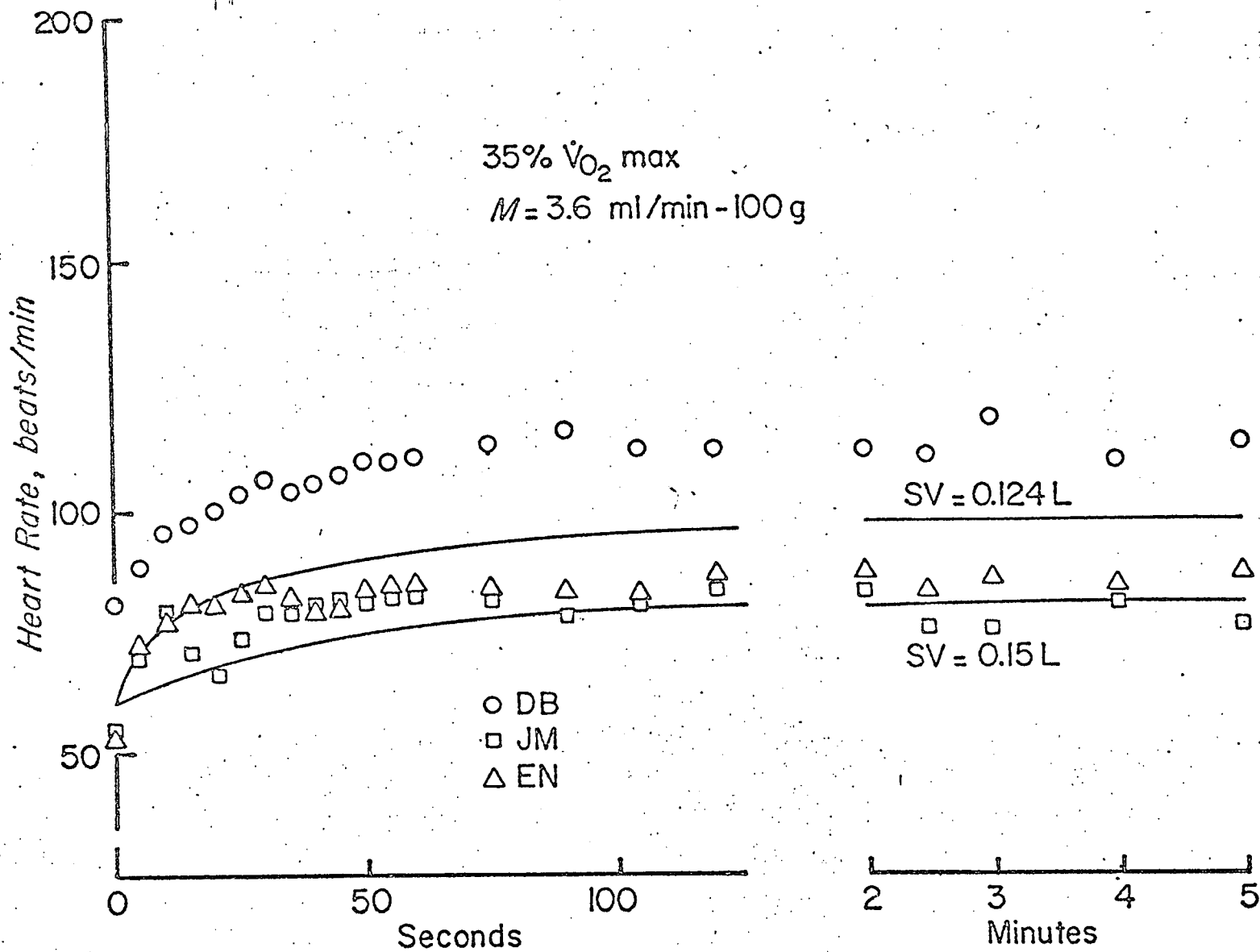
Figure 8. Model predictions and observed experimental values for heart rate after onset of exercise at 35% of maximum aerobic capacity.
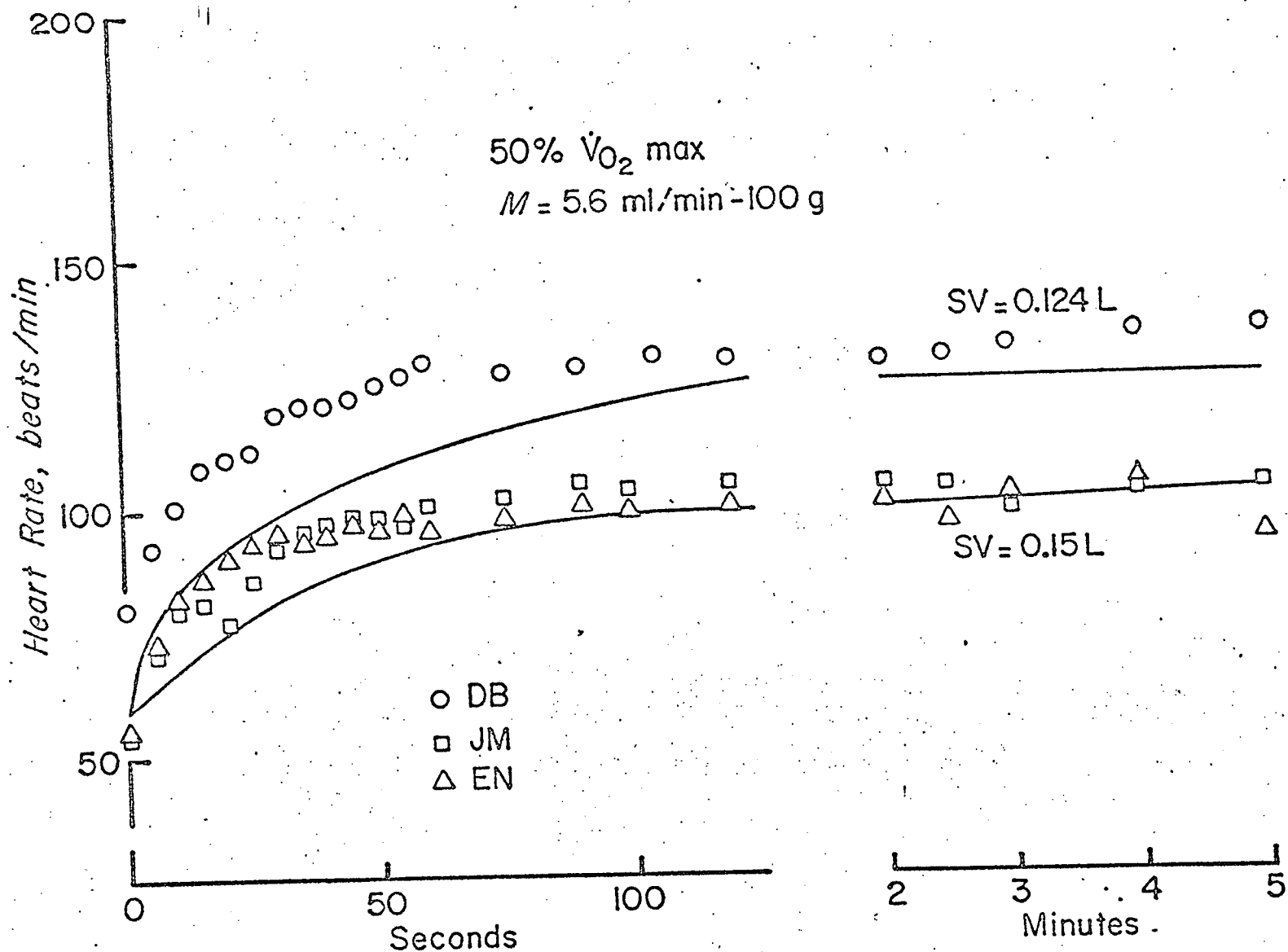
Figure 9. Model predictions and observed experimental values for heart rate after onset of exercise at 50% of maximum aerobic capacity.
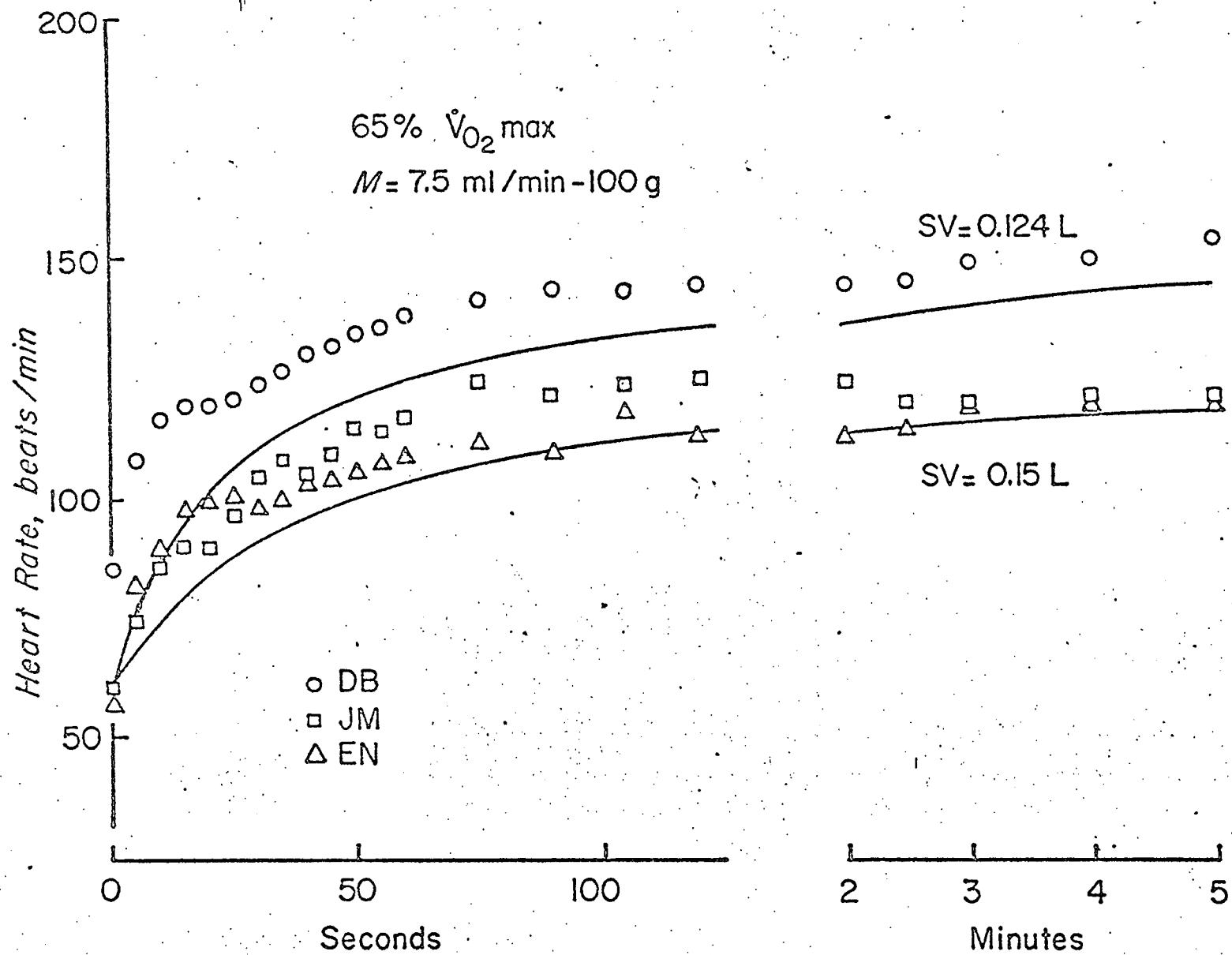
Figure 10. Model predictions and observed experimental values for heart rate after onset of exercise at 65% of maximum aerobic capacity.
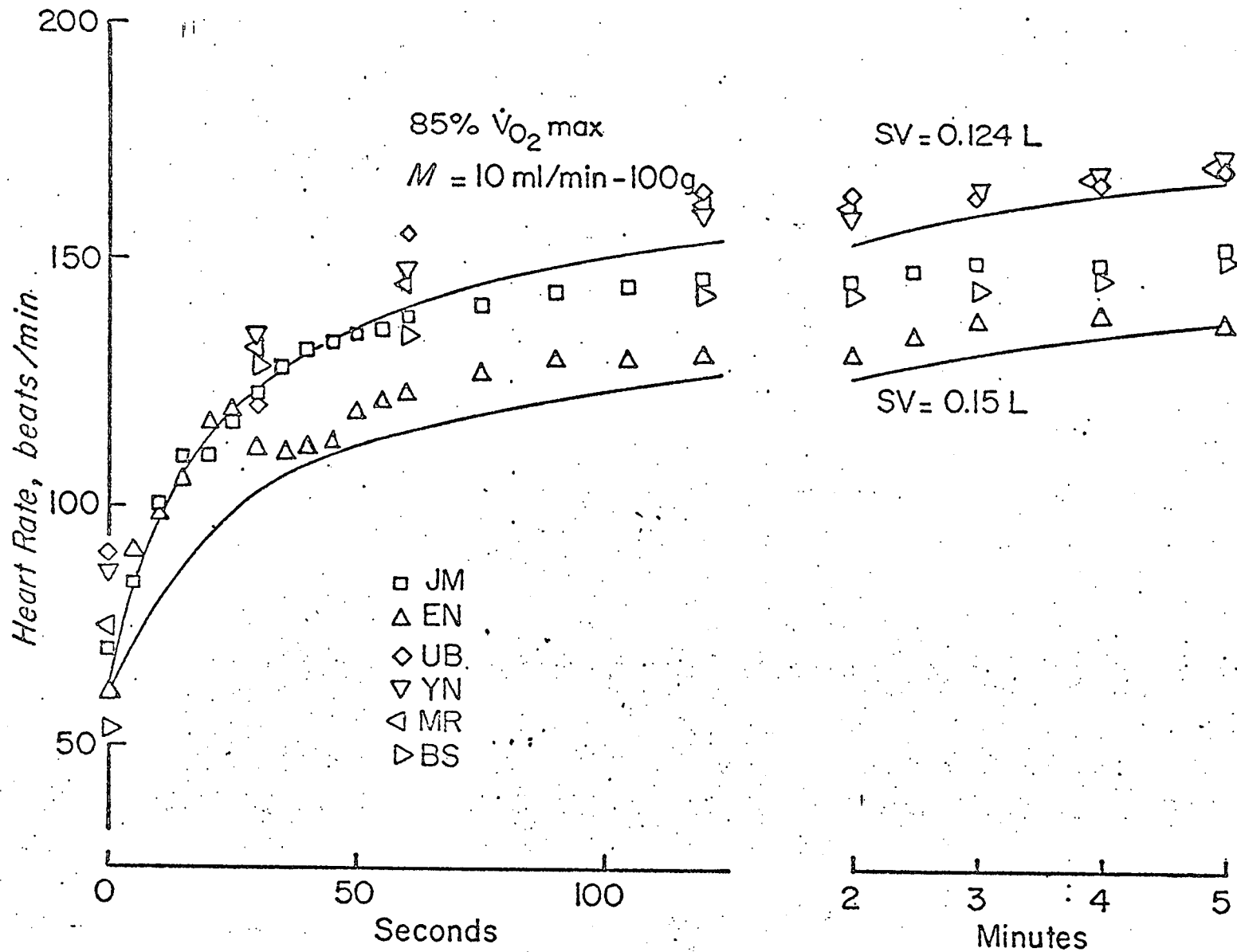
Figure 11. Model predictions and observed experimental values for heart rate after onset of exercise at 85% of maximum aerobic capacity.
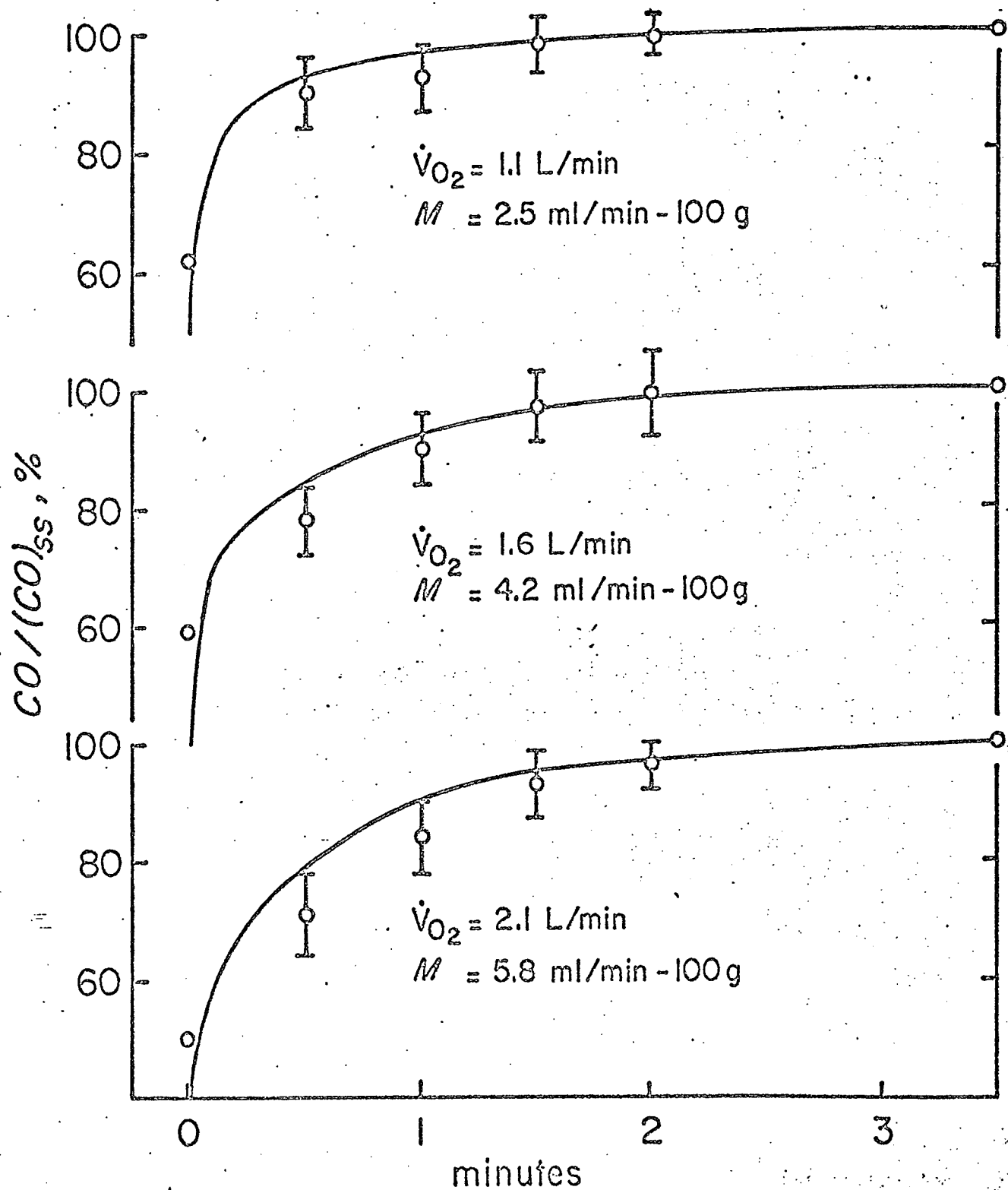
Figure 12. Predicted and experimentally observed (9) time course of increasing cardiac output after onset of exercise at three levels of oxygen uptake.

exercise level to another. The experiments of Broman and Wigertz (2) were simulated using the model, and these results are presented in Fig. 13 and 14. The difference between the model and the data is about 8 percent during the transient from a lower to a higher level of exercise.

In addition to predictions for the increase in work level, predictions were made for a decrease in the level of work (2) using two different assumptions. In the first simulation, it was assumed that there was a continuous repayment of both the myoglobin and anaerobic deficits; the control relations for both x_m and e_m as given in Figs. 1 and 2 were used. Secondly, it was assumed that only the myoglobin deficit was repayed. As shown in Figs. 13 and 14, neither of these assumptions is acceptable; lactate repayment proceeds more slowly during the recovery phase of exercise than the rate at which it is contracted during exercise.

DISCUSSION

The control value of P_{O_2} chosen here of 25 mm Hg is the oxygen tension value at which myoglobin begins to desaturate and the release of chemical energy stores initiates (Fig. 1 and 2). This supports the idea that some change in a chemical component is needed in order to provide an error signal. This error signal initiates control mechanisms which reduce resistance in the capillary bed and increase blood flow. Concomitant alterations in levels of P_{O_2}, P_{CO_2}, pH, bicarbonate and lactate occur in tissue and blood during the first part of exercise. The magnitude of change in these variables appears to be directly correlated with the intensity of exercise (21) and thus each variable is correlated with the other. The use of P_{O_2} here as the controlling variable does not imply that the oxygen tension alone acts to signal the need for increased oxygen. Oxygen
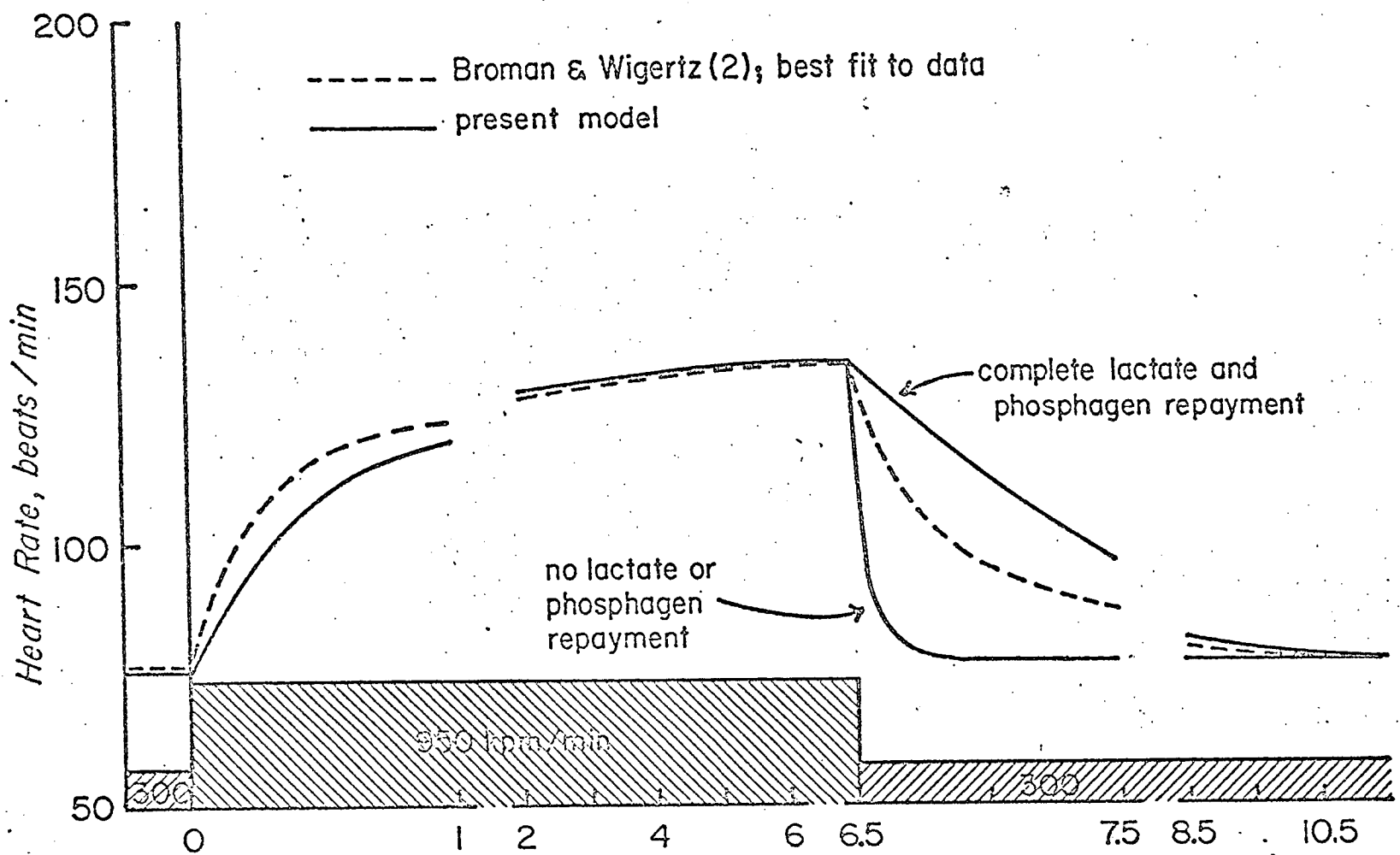
Figure 13. Observed and predicted changes in heart rate following step changes in work level.
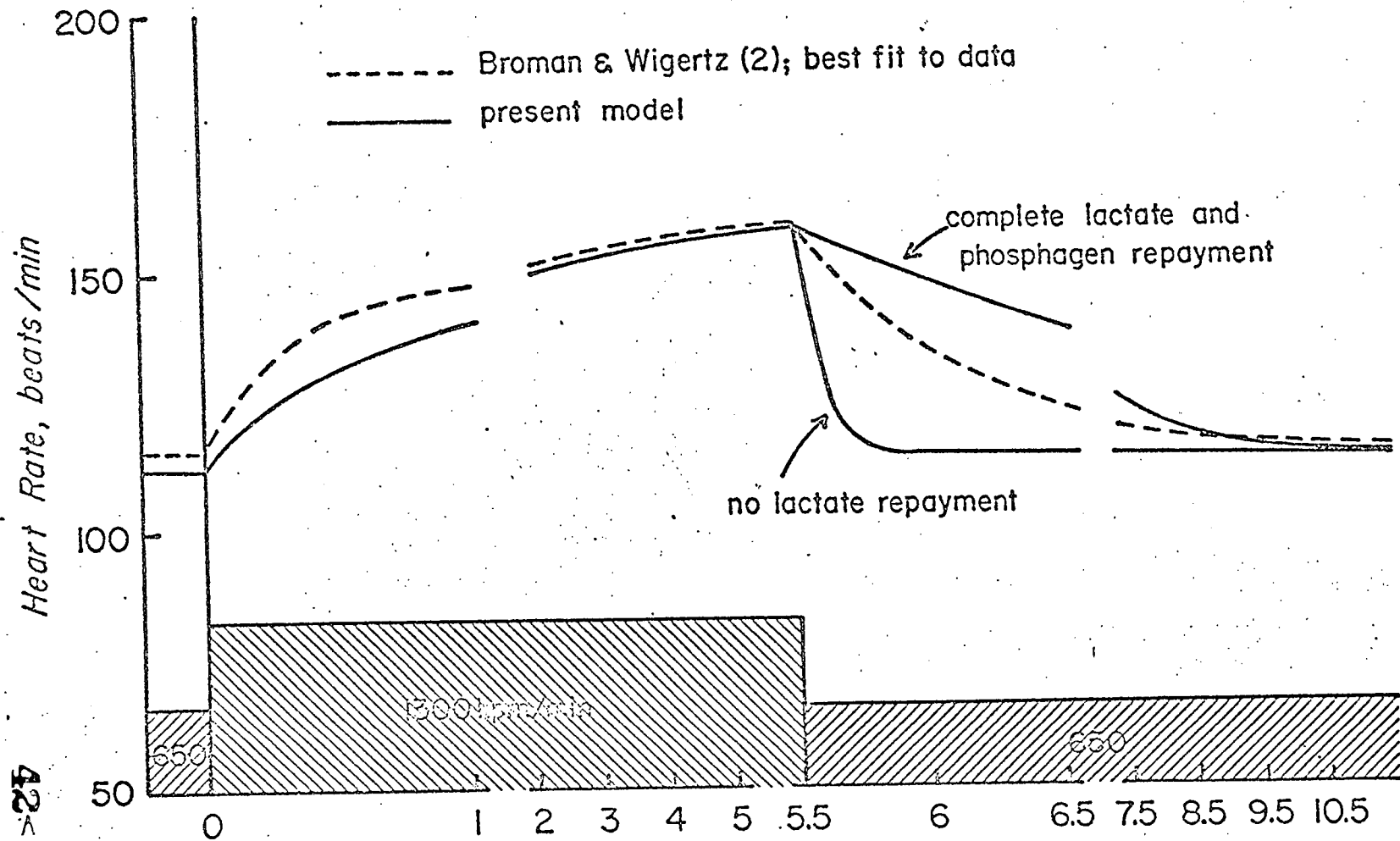
Figure 14. Observed and predicted changes in heart rate following step changes in work level.

tension is chosen as a convenient measure of the chemical state of the muscle during exercise.

The model results aid explanation of experimental observations. Previous investigators have reported that at low work levels (up to about four time resting values) metabolism is essentially aerobic (20,21). The model results show that at these low levels (less than about 1.2 L/min), the muscle P_{O_2} would be less than about 17 mm Hg (Fig. 4). From Fig. 2, it can be seen that the anaerobic contribution (the change in e_m) at these tensions is negligible. Thus, the calculations presented here illustrate that at low levels the major components of the oxygen deficit are the depletion of oxygen carried in blood and in myoglobin.

The heart rate, flow rate, and P_{O_2} curves presented in Figs. 6 through 14 are not exponential with time. The nonlinear relations between $\dot{m}_{bl}$, e_m, x_m, x, and P_{O_2} imply that the response cannot be represented by exponentials, or even in terms of known analytic functions. The idea of a time constant for an exercise transient is not really applicable.

Predicted steady state muscle blood flow rate is reached very quickly in light work, while heavier work loads require a longer period to achieve steady state (Fig. 6). Steady state is reached in about 2 minutes for 25% $\dot{V}_{O_2}$ max, 4 to 5 minutes for 50% $\dot{V}_{O_2}$ max, and 6 to 7 minutes for 75% $\dot{V}_{O_2}$ max, if thermoregulatory requirements for increased skin blood flow are ignored, as they are in this first approximation.

As shown in Figs. 8, 9, 10 and 11, the predicted heart rate increases slightly slower than the measured values. This may be due to the rapidity of increased sympathetic nervous system activity and reduced vagal tone immediately at the start of exercise. It is also possible that stroke volume does not

increase instantaneously from its resting to its exercise value as assumed in the model. The measurements of Jones et al. (9) show, in general, a constant stroke volume from the start of exercise, with any increase being moderate. However, the high resting cardiac outputs in their work (7.5 to 9 L/min) may mask any change at the start of exercise. The heart rate measurements in Figs. 8, 9, 10 and 11 show a slight decrease between 10 and 50 seconds after the start. This drop may reflect the change in stroke volume from the lower resting value to the higher exercise one.

The relative contributions of the different forms of stored energy during the transient were studied by computing blood flow response required to provide sufficient energy sources to the working muscle in different conditions. Representative results, presented in Figs. 15 and 16, were obtained by solving Eq. (3) with x_m constant, e_m constant, and e_m doubled for the respective cases shown. With no storage, the blood flow response (must be and) was found to be instantaneous to support the working muscle. The presence of myoglobin stores can significantly affect the blood flow response only at low work levels. Conversely, the capability to derive energy via production of lactate and depletion of phosphagen stores was found to affect the transient only at high work levels. If hemoglobin, myoglobin and the phosphagens were the only oxygen stores, steady state blood flow rate would be necessarily reached in about 15 seconds if work was to be sustained. Thus, the dependence upon the chemical energy stores are the major reason that the time to reach steady state is longer at the higher work levels.

The calculations for values of e_m twice those in Fig. 2 were performed to evaluate the assumption of distributing the anaerobic deficit over the entire
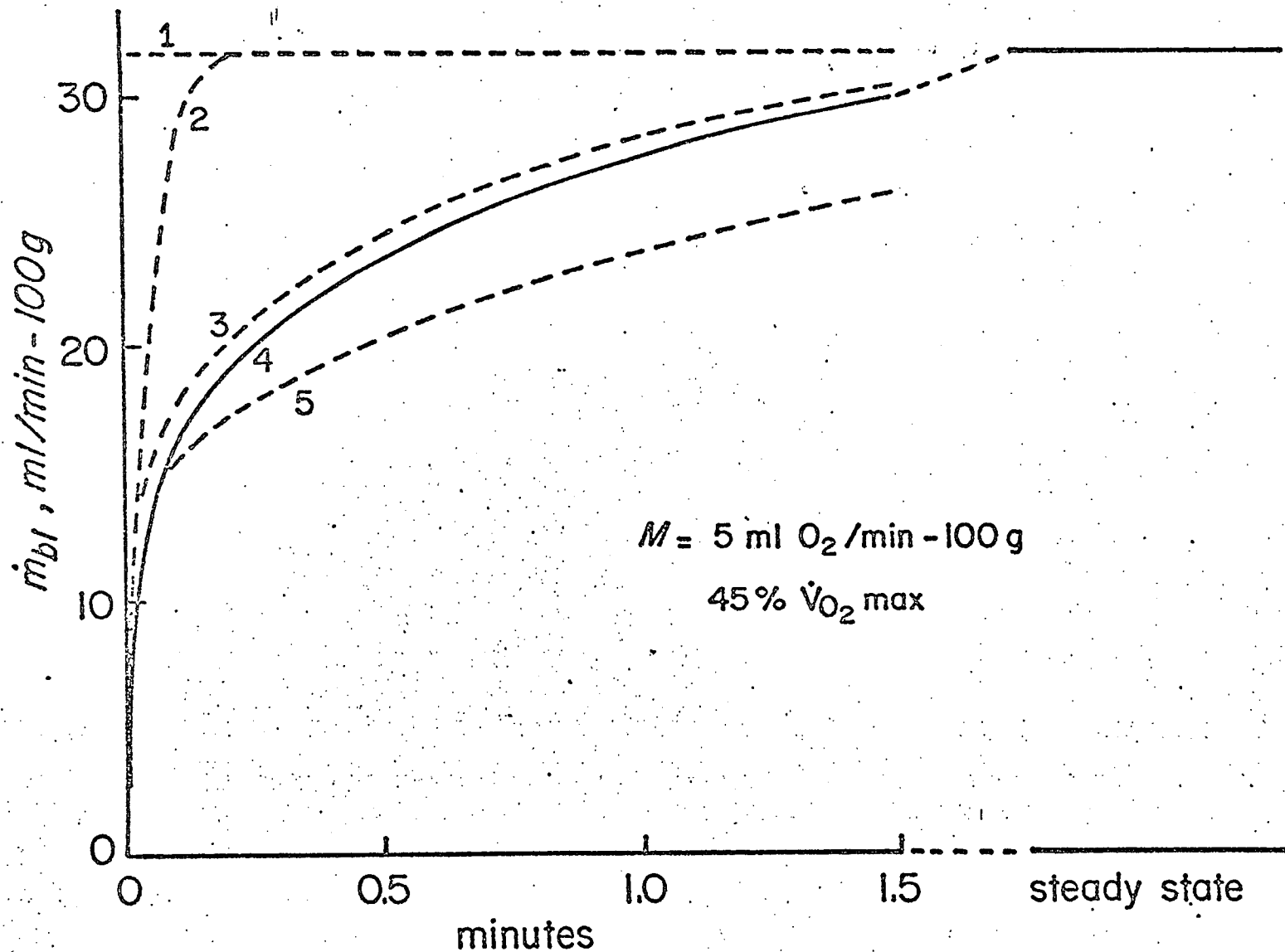
Figure 16. Predicted muscle blood flow after onset of exercise under different assumptions:
1° Instantaneous response to oxygen requirements
2° The absence of anaerobic metabolism and phosphagen stores
3° The absence of myoglobin oxygen stores
4° Normal assumptions of oxygen equivalent stores
5° Assuming double normal oxygen equivalent stores from phosphagen and anaerobic metabolism.

muscle mass. In bicycle exercise the leg muscles (16-20 kg) are primarily involved, rather than the entire musculature. At higher work loads (Fig. 16), the response with the higher e_m is considerably slower. The corresponding heart rates for this case would show a greater discrepancy between the experimental and model results (Fig. 9 - 11), but the discrepancy in cardiac output (Fig. 12) would be reduced. Further research utilizing different forms of exercise would be needed to resolve this problem.

The comparison between the predicted and calculated heart rates during the increase and decrease of work levels (Fig. 13 and 14) illustrates the difference that must exist between the mechanisms for the release and repayment of anaerobic chemical energy. The agreement during the step increase in work rate implies that the chemical energy is instantaneously available, and that thermal effects are not significant. During the step decrease, the implication is that the chemical process for repayment does not occur instantaneously, as shown by the difference between the experiment and the curve for complete repayment. However, some repayment must occur, or otherwise the heart rate would drop rapidly to the new level as shown by the lower curve. This difference between the onset and recovery is consistent with the observation that the oxygen deficit occurs in the first 2 to 4 minutes, while the debt must be determined over 20 to 30 minutes (12).

The implication of this analysis is that the initial transient for exercise is very rapid, with most of the changes that occur taking place in the first minute. Research into activities at the onset of exercise will require rapidly responding instrumentation if meaningful results are to be obtained. Models such as the present one are valuable in that they provide a ready basis for testing and interpreting previous data, and they provide a means for suggesting appropriate studies.

References

1. ASMUSSEN, E. and M. NIELSEN. Cardiac output during muscular work and its regulation. Physiol. Rev. 35: 778-800, 1955.

2. BROMAN, S., and O. WIGERTZ. Transient dynamics of ventilation and heart rate with step changes in work load from different work levels. Acta Physiol. Scand. 81: 54-74, 1971.

3. CARDUS, D. and R. K. ZIEGLER. Heart beat frequency curves. A mathematical model. Comp. Biomed. Res. 1: 508-526, 1968.

4. DI PRAMPERO, P. E., C. T. M. DAVIES, P. CERRETELLI, and R. MARGARIA. An analysis of O_2 debt contracted in submaximal exercise. J. Appl. Physiol. 29: 547-551, 1970.

5. GILBERT, R., J. H. AUCHINCLOSS, JR., and G. H. BAULE. Metabolic and circulatory adjustments to unsteady-state exercise. J. Appl. Physiol. 22: 905-912, 1967.

6. GILBERT, R., G. H. BAULE, and J. H. AUCHINCLOSS, JR. Theoretical aspects of oxygen transfer during early exercise. J. Appl. Physiol. 21: 803-809, 1966.

7. HENRY, F. M. Aerobic oxygen consumption and alactic debt in muscular work. J. Appl. Physiol. 3: 427-438, 1951.

8. JOHNSON, P. C. Physiology, Indianapolis, E. E. Selkurt, (ed.) Little Brown Co., 1966, p. 458.

9. JONES, W. B., R. N. FINCHUN, R. O. RUSSELL, JR., and T. J. REEVES. Transient cardiac output response to multiple levels of supine exercise. J. Appl. Physiol. 28: 183-189, 1970.

10. KARLSSON, J. Lactate and phosphagen concentrations in working muscles of man. Acta Physiol. Scand., Supp. 358, 1971.

11. KARLSSON, J. and B. SALTIN. Lactate, ATP and CP in working muscles during exhaustive exercise in man. _J. Appl. Physiol_. 29: 598-602, 1970.

12. KNUTTGEN, H. G. Oxygen debt after submaximal physical exercise. _J. Appl. Physiol_. 29: 651-657, 1970.

13. NADEL, E. R., J. W. MITCHELL and J. A. J. STOLWIJK. Control of local and total sweating during exercise transients. _Int. J. Biometeorol_. In press.

14. ROWELL, L. B. Circulation. _Medicine and Science in Sports_ 1: 15-22, 1969.

15. RUSHMER, R. F. _Cardiovascular Dynamics_, Philadelphia, W. B. Saunders Co., 1961, p. 437.

16. RUSHMER, R. F. _Physiology and Biophysics_, Philadelphia, T. C. Ruch and H. D. Patton (eds.), W. B. Saunders Co., 1965, p. 658.

17. SALTIN, B., G. BLOMQVIST, J. H. MITCHELL, R. L. JOHNSON, JR., K. WILDENTHAL, and C. B. CHAPMAN. Response to exercise after bed rest and after training. American Heart Association Monograph No. 23, Vol. XXXVIII, No. 5, Suppl. No. 5, November, 1968.

18. STOLWIJK, J. A. J. A mathematical model of physiological temperature regulation in man. NASA Report 9-9531, 1971.

19. THOMAS, H. D., B. BOSHELL, C. GAOS, and T. J. REEVES. Cardiac output during exercise and anaerobic metabolism in man. _J. Appl. Physiol_. 19: 839-848, 1964.

20. WASSERMAN, K., G. G. BURTON and A. L. VAN KESSEL. Excess lactate concept and oxygen debt of exercise. _J. Appl. Physiol_. 20: 1299-1306, 1965.

21. WASSERMAN, K., A. L. VAN KESSEL and G. G. BURTON. Interaction of physiological mechanisms during exercise. J. Appl. Physiol. 22: 71-85, 1967.

22. WHIPP, B. J., C. SEARD and K. WASSERMAN. Oxygen deficit – oxygen debt relationships and efficiency of anaerobic work. J. Appl. Physiol. 28: 452-456, 1970.

Part II. Physiology of the Thermoregulatory System

During the report year a number of research activities concerned with the sweating mechanism, respiratory weight loss and exercise resulted in publications and manuscripts submitted for publication. In each case the methodology, experimental results and discussion is best described in the individual papers which are included in this report.

A listing of the papers is as follows:

1. A.P. Gagge, J.A.J. Stolwijk and Y. Nishi. An effective temperature scale based on a simple model of human physiological regulatory response. ASHRAE Transactions 77, Part I, 247-262 (1970).

2. M. Cabanac, D.J. Cunningham and J.A.J. Stolwijk. Thermoregulatory set point during exercise: a behavioral approach. J. Comp. Physiol. Psychol. 76, 94-102 (1971).

3. Ethan R. Nadel, Robert W. Bullard and J.A.J. Stolwijk. Importance of skin temperature in the regulation of sweating. J. Appl. Physiol. 31, 80-87 (1971).

4. Jan A.J. Stolwijk. Thermal loads in lunar ambulation. Aerospace Med. 41, 1266-1268 (1970).

5. B. Saltin, A.P. Gagge, U. Bergh and J.A.J. Stolwijk. Body temperatures and sweating during exhaustive exercise. Submitted to J. Appl. Physiol.

6. Ethan R. Nadel, John W. Mitchell, Bengt Saltin and J.A.J. Stolwijk. Peripheral modifications to the central drive for sweating. Accepted for publication J. Appl. Physiol.

7. J.A.J. Stolwijk, E.R. Nadel, J.W. Mitchell and B. Saltin. Modification of central sweating drive at the periphery. Accepted for publication, Int. J. Biometeorology.

8. E.R. Nadel, J.W. Mitchell and J.A.J. Stolwijk. Control of local and total sweating during exercise transients. Accepted for publication, Int. J. Biometeorology.

9. John W. Mitchell, Ethan R. Nadel and Jan A.J. Stolwijk. Respiratory weight losses during exercise. Submitted to J. Appl. Physiol.

No. 2192

A. P. GAGGE
Member ASHRAE

J. A. J. STOLWIJK

Y. NISHI
Member ASHRAE

AN EFFECTIVE TEMPERATURE SCALE BASED ON A SIMPLE MODEL OF HUMAN PHYSIOLOGICAL REGULATORY RESPONSE

The purpose of the present paper is to develop an environmental temperature scale based on our current knowledge of the physiology of heat regulation as it applies to comfort, temperature sensation, and health. We will construct this scale by using first the heat exchange equations during the passive state as a rational starting basis and by introducing then the effect of known physiological regulatory controls. The principles to be described may be applied to many levels of activities, to various air movements, and to radiant heating and cooling. However, our present discussion will deal primarily with normally clothed sedentary human subjects in a uniformly heated and normally ventilated environment. As a numerical index, the new temperature scale will be defined in terms of dry bulb and normal humidity and will be comparable to temperatures of *natural* environments, which one generally experiences in temperate climates.

Physiological Bases for a Temperature Scale

The first single temperature scale, which was used to measure the thermal comfort of the environment, was developed by Houghton and Yaglou[1] for ASH&VE in 1923. By a series of carefully chosen experimental conditions they were able to predict loci of constant temperature sensation expressed in terms of dry bulb and humidity. After almost 50 years this empirical psychophysical temperature scale is still in use the world over. This scale has shown the importance of humidity and dry bulb in judging comfort and heat stress and has been used as a temperature standard for working conditions in many occupations.[2,3] In later years

A. P. Gagge is professor of environmental physiology, John B. Pierce Foundation and Yale University, New Haven, Conn. J. A. J. Stolwijk is associate professor of environmental physiology and Y. Nishi is research associate, John B. Pierce Foundation, New Haven, Conn. This paper was prepared for presentation at the ASHRAE Semiannual Meeting in Philadelphia, Pennsylvania, January 24-28, 1970.

Yaglou[4] recognized that his scale perhaps may have over-exaggerated the effect of humidity towards the lower temperatures. Minard[5] has shown the greater importance of wet bulb or dew point temperature toward the heat tolerance levels. Nevertheless there are many who still accept the older scale as a reasonable and logical estimate of environmental temperature stress for normally clothed sedentary persons.

Since the late 1930's there has been a continuing effort to rationalize sensory observations of comfort and temperature with the temperatures that occur simultaneously within the body and on the skin surface, with the various regulatory processes that result in vasoconstriction and vasodilation of the vascular system within the skin layer, and with the secretion of sweat necessary for evaporative cooling. The general nature of these processes has already been covered in the ASHRAE literature. Here the role played by this major physiological factor may be summarized briefly as follows:

Internal (or Core) Body Temperature

The experiments of Chatonnet and Cabanac[6] have shown for sedentary persons in a water bath that core temperature is a good index of thermal discomfort and that this sensation may be quite independent of skin temperature. The same observation in a calorimetry has been made by Benzinger.[7] During the exercise the same degree rise in core temperature does not cause necessarily the same discomfort as observed for sedentary conditions.

Skin Temperature

Skin temperature has long been recognized as a major factor in judging the temperature sensation caused by the environment. In cool or cold environments it also affects thermal comfort.[8,9] In both warmth and cold man's temperature sense is generally correlated with skin temperature and is

apparently independent of his activity.[10] The distribution of skin temperature over the body surface is also a major factor in the cold;[11] the greater the uniformity the greater the comfort; the less, more discomfort. In the heat the sense of discomfort or unpleasantness, in contrast, is less dependent on skin temperature and more on core temperature or those central neural processes associated with the regulatory drive.[12,10] The same skin temperature may correlate with a warmer or colder temperature sensation than normal when the body temperature is lower or higher respectively than normal.[6]

Vasoconstriction and Vasodilation

Vascular control of blood flow to and from the skin surface is a method by which the body can change the flow of heat from its central core to the environment. Although this process is not as effective as shivering would be in the cold or as sweating in the heat, it can cause significant changes in man's sensation of well-being.[8] Vasoconstriction is usually associated with a sense of cold; vasodilation, which always occurs during regulatory sweating, may cause an increased sense of warmth. Without sweating, vasodilation may cause a sense of comfort or well-being although skin temperature may be well below the temperature threshold expected for a neutral sensation.

Sweating

Man's best protection against heat is his ability to use the evaporative cooling caused by his sweating. Sweating is caused by temperature stimuli from both the skin and core; there are both local controls and central controls.[13,14] Sweating *per se* is not an uncomfortable process as long as moisture can evaporate freely from the skin surface. Discomfort occurs, when the same rate of sweating requires a larger wetted surface on the skin from which it must evaporate. Sweating at very high levels is usually accompanied by active vasodilation, which in turn may cause swelling of the extremities, skin irritation, headaches and throbbing due to increased blood flow and heart action.

The Skin Surface

The skin surface is the boundary from which the human body exchanges heat with the environment by radiation, convection and evaporation. The heat of metabolism reaches this boundary surface by conduction and by blood flow. Secretion of perspiration on the boundary surface supports the evaporative cooling. Skin temperature and skin wettedness are the two principal physical properties of this boundary surface and are the *resultant* of both the various internal regulatory processes and the external thermal stress caused by the environment.

In the following section we will describe in familiar physical terms a model that contains the many physiologically dependent and independent variables described above and combine them to predict the physiology that occurs during a thermal state of quasi-equilibrium after a fixed exposure period to any various environmental condition.

THEORETICAL BASES
The Passive System
Part 1 — Skin to the Environment

The classic heat balance equation describing the heat gained from and lost to the environment may be written

$$S = M - E + R + C - W \quad \text{energy/time} \quad , \quad (1)$$

where S = rate of heating (+) or cooling (−) by the body,
M = net rate of metabolic heat production,
E = total evaporative heat loss,
R = heat gained (+) or lost (−) by radiation,
C = heat gained (+) or lost (−) by convection,
and W = work accomplished.

In Eq (1) the conventional unit for heat exchange used by physiologists is the watt.[15] The corresponding English unit is Btuh. Since heat transfer coefficients will be involved in terms relating the heat exchange from the skin surface with the environment, the above terms will be described per unit area of the body surface as measured by DuBois.[16] For the remainder of this paper the units of choice will be W/sq m or Btu/(hr)(sq ft).

Metabolic rate M is proportional to the rate of O_2 consumption, which may be measured directly. E may be measured directly by observing the rate of body weight loss on a sensitive balance. These are well known procedures in partitional calorimetry.[17,18]

The total evaporative heat loss is divided into three parts; thus

$$E = E_{res} + E_{diff} + E_{rsw}, \quad (2)$$

where E_{res} = heat of vaporized moisture from the lungs during respiration,
E_{diff} = heat of vaporized water diffusing through the skin layer,
and E_{rsw} = heat of vaporized sweat necessary for the regulation of body temperature.

In Eq (2) above the sum, $E_{res} + E_{diff}$, is known as the "insensible" heat loss from the body; the component E_{rsw} as the "sensible" loss.

In general E_{res} is directly proportional to the vapor pressure gradient from the lungs to the ambient air and to the ventilation rate of the lungs. The latter term is proportional to the metabolic rate itself. Fanger[19] has developed the following relation for E_{res}, based on data available in physiological literature;

$$E_{res} = 0.0023 \, M \, [44 - \phi_a P_a], \quad \text{in W/sq m} \quad (3)$$

where 44 mm of Hg = the saturated vapor pressure for an average lung temperature of 35.5 C (96 F),
ϕ_a = relative humidity as a fraction,
and P_a = the saturated vapor pressure for the dry bulb or air temperature (T_a) of the environment in mm of Hg.

Eq (3) is useful for resting to moderate exercise.

The maximum evaporative heat loss E_{max} from the body surface has been shown to be[12,20]

$$E_{max} = \kappa \, h_c [P_{sk} - \phi_a P_a] \, F_{pcl} \quad \text{in W/sq m} \quad (4)$$

where κ = the Lewis relation and equals 2.2 C/mmHg (or 31 F/in Hg) at sea level,
h_c = convective heat transfer coefficient in W/(sq m)(C)

P_{sk} = the saturated vapor pressure at mean skin temperature T_{sk} in mm (or in) of Hg,

and F_{pcl} = permeation efficiency factor for water vapor evaporated from the skin surface through clothing to the ambient air.[21]

In general, E_{max} is a direct measure of the evaporative power of the environment for moisture or sweat accumulated on the body surface.

The evaporative loss E_{sk} from the skin surface may be observed experimentally by subtracting E_{res} from the value of E, found by measuring the rate of weight loss. It is also given by

$$E_{sk} = E_{diff} + E_{rsw} . \tag{5}$$

The ratio E_{sk}/E_{max} is a measure of the average wettedness (w) of the body skin surface.[22] Its minimum value occurs when the skin evaporative loss is insensible. When E_{sk} is defined as the evaporative heat loss necessary for the regulation of body temperature (i.e. $S=0$), the ratio $E_{sk}/E_{max} \times 100$ has been used by Belding and Hatch[23] as an index of environmental heat stress.

Brebner et al.[24] have shown E_{diff}, when $E_{rsw} = 0$, to be proportional to evaporative power of the environment E_{max}. Using their data

$$w_{diff} = E_{diff}/E_{max} = 0.06 \quad (N.D.) \tag{6}$$

The value of 0.06 for w_{diff} represents the normal dampness factor for human skin without sweating. This value may drop as low as 0.04 when dehydration of the skin from exposure to cold and lower humidities causes a change in its diffusive characteristics for body water.[25]

In warm environments, as the sweat glands become successively active for the regulation of body temperature and as this secretion spreads as an evaporating thin film over the body surface, the proportion of skin areas with insensible perspiration becomes less and those with wet surfaces due to sweating become greater. E_{diff} and E_{rsw} never occur on the same area of the skin at the same time. At any time w_{rsw} equals E_{rsw}/E_{max}. When the surface is fully wet, $w = w_{rsw} = 1$. When $w_{rsw} = 0$, $w = w_{diff} = 0.06$. These two limits, as well as the intermediate, conditions are described when the total loss from the skin surface E_{sk} is written as follows:

$$E_{sk} = (0.06 + 0.94 \, w_{rsw}) \, E_{max} \tag{7}$$

For the present analysis we will consider the environment to be at a uniform temperature, T_a. The dry heat exchange from the skin surface is described by

$$(R + C) = h(T_{sk} - T_a) \, F_{cl} \tag{8}$$

where h = the combined heat transfer coefficient and is the sum of the linear radiation exchange coefficient h_r and the applicable convective heat transfer coefficient h_c in W/(sq m)(C) or Btu/(hr)(sq ft)(F),

and F_{cl} = a factor that measures the efficiency for the passage of dry heat from the skin surface at T_{sk} through the clothing to the environment at T_a.

F_{cl} and F_{pcl} are analogous factors for heat transfer by convection and for mass transfer by water vapor respectively and, as concepts, were originally proposed by Burton and Edholm.[26] Both are a function of the clothing insulation I_{cl} in clo units as well as the coefficients h and h_c respectively[21].

In a uniform environment we may now write the complete heat balance equation for a subject not doing external work (W)

$$S = M \, [1 - 0.0023(44 - \phi_a P_a)]$$
$$- 2.2 \, h_c \, (0.06 + 0.94 \, w_{rsw}) \, [P_{sk} - \phi_a P_a] \, F_{pcl}$$
$$- (h_r + h_c) \, (T_{sk} - T_a) \, F_{cl} \tag{9}$$

Alternatively Eq (9) may be rewritten

$$S = M \, [1 - \mu - 0.0023 \, (44 - P_{dew})] - 2.2w \, h_c \, (P_{sk} - P_{dew})$$
$$F_{pcl} - h(T_{sk} - T_a) \, F_{cl} \tag{10}$$

where the mechanical efficiency μ is equal to W/M and P_{dew} is the saturated vapor pressure at dew point, T_{dew}.

The reader will recognize the first term on the right of Eqs (9) or (10) as the net heat produced by the body which is lost through the skin surface; the second is the total evaporative heat loss from the skin surface; and the third is the dry heat exchange. The units in use may be either W/sq m, W/(sq m)(C), mm Hg; or Btu/(hr)(sq ft), Btu/(hr)(sq ft)(F), or in of Hg.

PART 2 – FOR CORE AND SKIN

The core and skin of the human body will be treated analytically as two concentric shells (Fig. 1); the skin is represented by a thin shell with mass m_{sk}; the body interior by
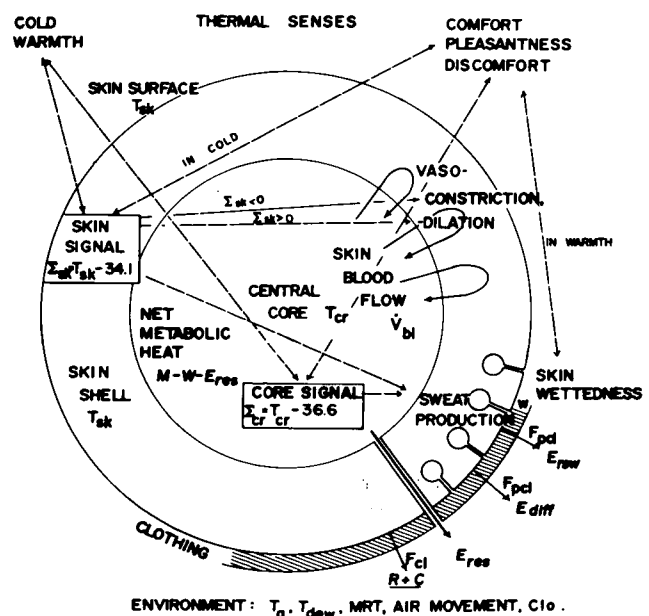

Fig. 1 A concentric shell model of man and his environment

a central core with mass m_{cr}; the total body mass m in kg will be $m_{sk} + m_{cr}$; the body surface area will be A, which will be equal in magnitude to the DuBois area (2.0 sq m) of a standard man (81.7 kg-weight and 1.77 m height). The core mass is considered as 78.3 kg and the skin shell as 3.4 kg. The SI-system[15] of units will be used in the following analysis.

The net heat flow to and from the skin shell is given by the relation

$$S_{sk} = K_{min} (T_{cr} - T_{sk}) + c_{bl} \dot{V}_{bl} (T_{cr} - T_{sk}) - E_{sk} - (R + C) \quad (11)$$

where S_{sk} = rate of heat storage in W/sq m,
c_{bl} = specific heat of blood in W(hr)/(kg)(C),
$\dot{V}_{bl}$ = rate of skin blood flow in liter/(hr)(sq m),
and K_{min} = minimum heat conductance of skin tissue W/(sq m)(C).

The net heat flow to and from the core is given by

$$S_{cr} = (M - E_{res} - W) - K_{min}(T_{cr} - T_{sk}) - c_{bl}\dot{V}_{bl}(T_{cr} - T_{sk}). \quad (12)$$

It is clear to the reader that the total body heat storage S is given by

$$S = S_{sk} + S_{cr}, \qquad \text{W/sq m} \quad (13)$$

as may be seen from Eqs (9) or (10) above.

The total thermal capacity of the skin shell and core are

$$c'_{sk} = 0.97 \, m_{sk}, \qquad \text{W(hr)/C} \quad (14)$$

and

$$c'_{cr} = 0.97 \, m_{cr}, \qquad \text{W(hr)/C} \quad (15)$$

where the primes refer to the entire shell or core and 0.97 is the specific heat of the body in W(hr)/(kg)(C).

The rate of change in skin (shell) temperature T_{sk} and central core temperature T_{cr} are given by

$$\dot{T}_{sk} = S_{sk} A / c'_{sk}, \qquad \text{C/hr} \quad (16)$$

and

$$\dot{T}_{cr} = S_{cr} A / c'_{cr}. \qquad \text{C/hr} \quad (17)$$

In Eqs (16) and (17) the cooling and warming is considered as Newtonian for the core and shell and assume these are uniformly at temperature T_{sk} and T_{cr} respectively. We also assume that the same body surface area A applies to the skin surface and core surface. If the skin and body are at 34.1 C and 36.6 C respectively on initial exposure to an environment described by T_a and ϕ_a, then the values of T_{sk} and T_{cr} at any time are given by

$$T_{sk} = 34.1 + \int_0^t \dot{T}_{sk} dt, \quad (16a)$$

and

$$T_{cr} = 36.6 + \int_0^t \dot{T}_{cr} dt. \quad (17a)$$

The Controlling System

As implied in our physiological considerations above, we will assume that the temperature signals Σ from the skin shell and the central core are given by the two relations:

$$\Sigma_{sk} = T_{sk} - 34.1 \quad (18)$$

$$\Sigma_{cr} = T_{cr} - 36.6 \quad (19)$$

The values 34.1, and 36.6 have been observed[27] as the average temperature of the skin and core, when there is minimal regulatory effort in maintaining body temperature either by any vascular effort or by sweating. When these temperatures occur simultaneously during rest, the body is in a state of "physiological thermal neutrality."

When Σ_{sk} is negative, the skin senses "cold." When Σ_{sk} is positive, the skin senses "warmth." Likewise, when Σ_{cr} is negative, the core senses "cold" and positive the core senses "warmth." A "cold" signal from the skin primarily governs "vasoconstriction" in the vascular bed of the skin and thus reduces the blood flow from core to skin. A warm signal from the skin, as will be seen later, plays a more important role in body temperature regulation by governing sweating than by governing vasodilation. A warm signal from the core, will cause dilation in the vascular bed and evoke sweating. The corresponding cold signal from the core will cause vasoconstriction but not as rapidly or effectively as one from the skin. In a multicompartment model Stolwijk and Hardy[27] have estimated for each deg C drop for a cold Σ_{sk}, skin blood flow will encounter a proportional increase in resistance. For the hands and feet alone this resistance factor may be twice as great with each degree drop; for the trunk vasoconstriction may be negligible. For the core, each deg C rise will cause an increase in skin blood flow of 75 liter/(hr)(sq m) above a *normal* skin blood flow of 6.3 liter/(hr)(sq m), a value which occurs at rest during thermal neutrality. The above statements may be described by the following equation, which gives the skin blood flow $\dot{V}_{bl}$ at any time as:

$$\dot{V}_{bl} = (6.3 + 75 \, \Sigma_{cr})/(1 - 0.5 \, \Sigma_{sk}) \text{ in } 1/(hr)(sq m) \quad (20)$$

In Eq (20) when Σ_{cr} represents a cold signal (i.e. $T_{cr} < 36.6$) and/or when Σ_{sk} represents a warm signal (i.e. $T_{sk} < 34.1$), the numerical value of Σ in either case is considered as zero.

The glands that produce the regulatory sweating $\dot{m}_{rsw}$ in g/(hr)(sq m) at the skin surface, necessary for temperature regulation by evaporation, are activated both by the core signal Σ_{cr} and by the product $(\Sigma_{sk})(\Sigma_{cr})$. The rate of sweat production may be written as:

$$\dot{m}_{rsw} = 250 \, \Sigma_{cr} + 100 \, (\Sigma_{cr}) (\Sigma_{sk}). \quad (21)$$

The first term of Eq (21) has significance primarily during exercise; each degree change in core temperature (e.g. rectal temperature) above 36.6 C has been observed by Saltin *et al.*[28] to cause an average increase in sweat secretion of 250 g/(hr)(sq m) (C). The second term has been shown by Hardy and Stolwijk[14,27] to describe the sweat drive during rest; the factor 100 g/(hr)(sq m) (C²), represents the dual effect of a gain controller with an output described by the product $(\Sigma_{cr}) (\Sigma_{sk})$. The double – Σ terms have less significance during exercise as T_{sk} falls below 34.1 C. Whenever Σ is negative (i.e. a cold signal), its value in Eq (21) is zero.

Bullard *et al.*[13] have recently shown that skin temperature can modify locally the production of sweat. This non-dimensional control factor may be represented by a power function described by $2^{(T_{sk} - 34.1)/3}$. Thus each 3 deg rise in T_{sk} above 34.1 C doubles the ease of sweat production; this may occur during exposure to radiant heat for example. A 3 deg drop in T_{sk} from 34.1 C will reduce the local sweat production to a half; this drop occurs during exercise and

TABLE 1

STANDARD ENVIRONMENTAL PARAMETERS

Term	Units	Type of Activity					
		A	B	C	D	E	F
M	mets * (W/sq m)	1 (58.2)	2 (130.1)	1 (58.2)	4 (233)	4	5 (291)
h_r	W/(sq m)(C)	5.23	5.23	5.23	5.23	5.23	5.23
h_c	W/(sq m)(C)	2.91	4.30	2.91	6.8	10.00	5.37
h		8.14	9.53	8.14	12.03	15.23	10.60
I_{cl}	clo*	0.6**	0.6**	0	0.1#	0.1	0.1
μ		0	0	0	0	0	0.2

*The met unit[31] is equal to 50 kcal/(hr)(sq m); 58.2 W/sq m or 18.5 Btu/(hr)(sq ft); for an average sized man this unit corresponds approximately to 90 kcal/hr, 100 watts, or 400 Btuh. The clo[31] unit is 0.18 (C)(sq m)(hr)/(kcal); 0.88 (F)(sq ft)(hr)/(Btu); or 0.155 (C)(sq m)/W.

**Equivalent to KSU Standard Clothing.[29]

#Indoor athletic clothing.

modifies the powerful control drive of sweat secretion caused by Σ_{cr}. The heat loss from regulatory sweating may now be written as;

$$E_{rsw} = 0.7 \, \dot{m}_{rsw} \, [2^{(T_{sk} -34.1)/3}] \, , \qquad (22)$$

where 0.7 is the latent heat of sweat in W(hr)/g.

The equations of state describing the energy exchanges and temperatures of the shell-core model at any time are given by Eqs (9), (10), (12), (16) and (17). From the initial conditions that describe thermal neutrality it is possible to integrate the changes in skin and core temperature and the sweating caused by the controlling system (Eqs 18, 19, 20 and 22) and to predict all the physiological energy and temperature factors after any period exposure to any new environment described by T_a and humidity.

In a complex multiloop regulatory system, such as we use here, the coefficients used in Eqs (20) and (21) are not as significant in changing predictions of body temperatures as are the choice of the set points for T_{sk} and T_{cr} in Eqs (18) and (19). In contrast, the prediction of any environmental temperature for any energy exchange relationship or any value skin wettedness is not significantly changed by a small displacement 0.5 C (or 1 F) in any set point. The heat transfer coefficients introduced by the environment and the metabolic level primarily govern the pattern of loci of constant wettedness, of constant skin temperature or of constant core temperature in terms of dry bulb and humidity.

An annotated Fortran program incorporating the above equations, is given in the Appendix and is generally applicable to moderate levels of activity, to normal fabric clothing and to environmental conditions described by dry bulb temperature in range 5-45 C and by humidities down to 10%. The effect of radiant heating has not been included. There are seven independent variables in our model; namely (1) the metabolic rate; (2) the work accomplished; (3) the combined and (4) convective heat transfer coefficients, which both include the effect of air movement caused by both room ventilation and body activity; (5) the insulation of normal clothing used; (6) the dry bulb temperature; (7) the humidity, as measured by either ϕ_a, T_{wet}, or T_{dew}.

For these seven variables the principal physiological factors, predictable by our model, are average skin temperature T_{sk}, the core temperature T_{cr}, the regulatory sweating E_{rsw} and the corresponding skin wettedness w_{rsw}. Engineers interested in using this model can find a variety of sources in literature for evaluating variables (1) − (5).

Standard Conditions

Four types of general activity will be considered in the present paper:

A − Clothed and sedentary
B − Moderate activity
C − Unclothed and sedentary
D − 3 mph on treadmill
E − 3 mph free walking
F − Bicycle Ergometer (50 rpm)

The environmental parameters to be used for each are given in Table 1.

In Table 1, activity A occurs most probably for 90% of our current living conditions in the home and office. Activity C is the same sedentary level but unclothed. Activity B may be classed as a light physical effort and occurs most frequently in industries such as bakery, brewery, small machine work and also in house cleaning, domestic work, and washing by hand. It also corresponds to the moderate activity report by McNall et al.[29] Activities C and the following are of interest in heat tolerance and health studies. Activity D, E and F represent moderate exercise. For activity F the work-efficiency is 20% and is at approximately 40% of man's average maximal oxygen capacity. The values for h_c in Table 1 were measured directly by naphthalene sublimation.[30]

It is now possible to use our model to predict at the end of 1 hour exposure the equilibrium status for any combination of T_a or humidity. Fig. 2 for example indicates the predicted partitional calorimetry of a subject sitting and resting when clothed (Activity A). Five factors have been plotted for the temperature ranges 10-40 C or 50-105 F; T_{sk}, T_{cr}, $\Delta T_B/\Delta t$ (i.e. change in mean body temperature/hr), E_{rsw}, E_{ins},(i.e. $E_{res} + E_{diff}$), w_{rsw} and $\dot{V}_{bl}$ (skin blood
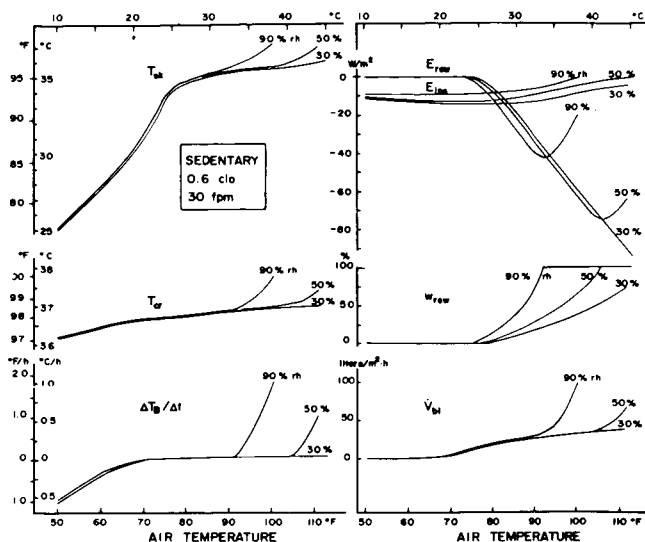
Fig. 2 Changes in various body temperatures, skin blood flow, evaporative cooling for a normal clothed man, predicted by our model and Fortran program in Appendix.

Fig. 3 Variations in E_{max} with increasing ambient temperature. The corresponding skin wettedness predicted by model has been shown for activities and humidity levels indicated.

flow). Regulatory sweat for both high and low humidity begins at 24 C (75.2 F) and increases steadily with increasing temperature. The rate of rise is slightly higher with increasing humidity. The skin temperature is also slightly higher with humidity, as air temperature increases. When skin wettedness reaches 100%, skin temperature, core temperature and skin blood flow all rise sharply and this leads to the sudden collapse of the subject. Above this maximum (w=1), the effective E_{rsw} steadily drops. The relationships in Fig. 2 are idealized in the sense that the assumption has been made that all evaporation takes place on the skin surface. As the predicted wettedness limit passes through 75-100% range, the true maximum evaporative loss may occur at lower temperatures because of evaporation of sweating in the clothing itself. The general pattern and principles outlined above still apply and represent realistic prediction for normal lightweight porous clothing.

During exercise and with increased body motion the ability to regulate body heat by evaporation is increased about 2 times over the sitting-resting levels. This fact is illustrated in Fig. 3 where the values of E_{max} for the resting and bicycling unclothed subjects at various dry bulbs and humidities are compared. Skin wettedness for various points along the E_{max} curves has been indicated. The magnitude of the net metabolic levels concerned are shown.

In studies of heat tolerance the physical factors that set the maximum wettedness limits are the net metabolism, clothing, air movement, and the dry and dew point temperatures. Fig. 4 demonstrates some of these inter-relationships on a T_{dew}-T_a plot. In the upper section of Fig. 4 for a constant metabolic rate of 1 met, the effect of various clothing insulation on the 100% wettedness limit is demonstrated. In all cases, the ambient air movement is held constant as conditions A and C in Table 1. Over the range 0-0.9 clo clothing has a small effect on the regulatory limit. In the lower section of Fig. 4 the net metabolic rate has been adjusted to a 4 met level. The limits for three types of exercise — free walking, treadmill walk, and bicycle ergometry — are indicated. When the environmental variables are held constant, the maximum wettedness limit lowers
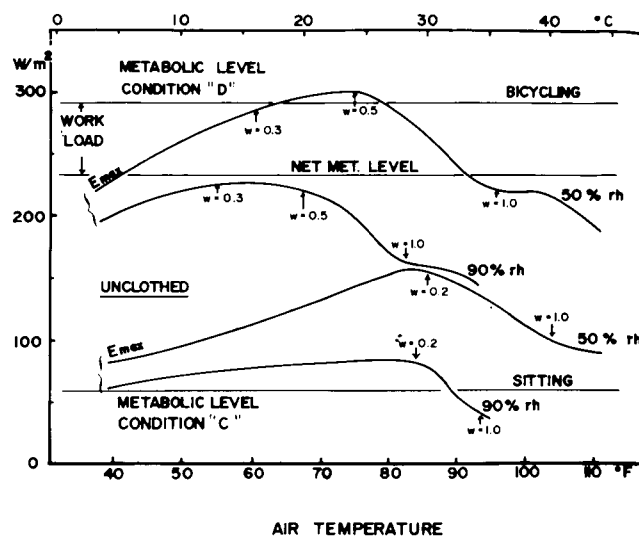
with increasing activity. Clothing increases the relative importance of the dew point temperature (and of wet bulb) in relation to the ambient temperature. Increasing air movement also increases the significance of dew point temperature over dry bulb.

Humid Operative Temperature (T_{oh})

Let us define humid operative temperature as the imaginary temperature at which the body will lose the same heat as he would by radiation, convection and evaporation in the
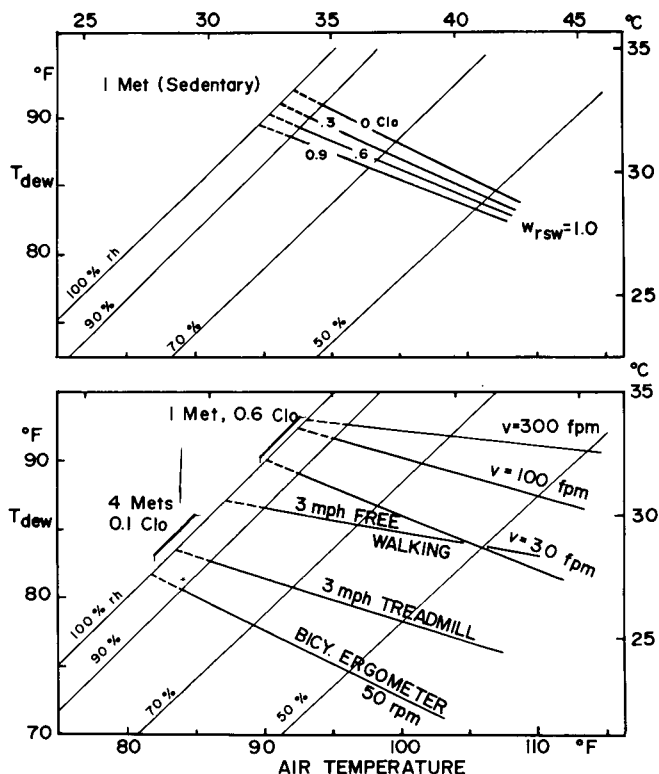

Fig. 4 The effect of clothing insulation, air movement, and exercise on the loci for maximum wettedness ($w = w_{rsw} = 1$)
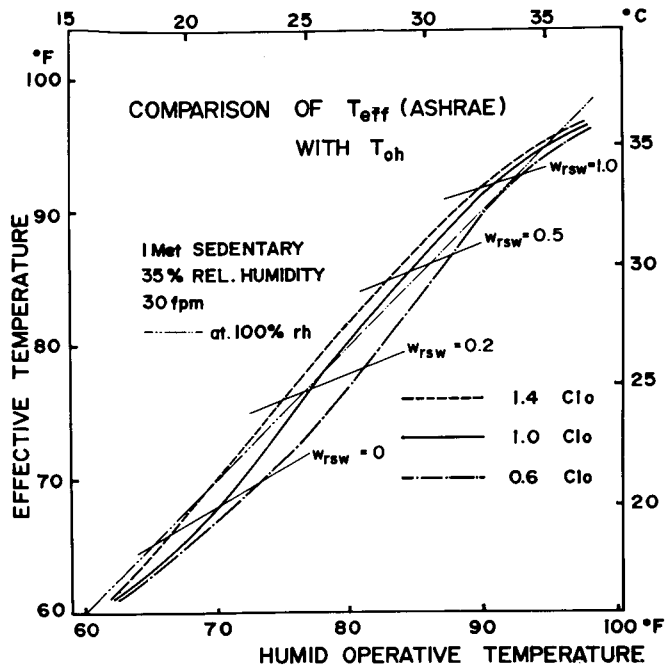
252

Fig. 5 A comparison of the predicted humid operative temperature with the current ASHRAE effective temperature scale for various clothing insulations
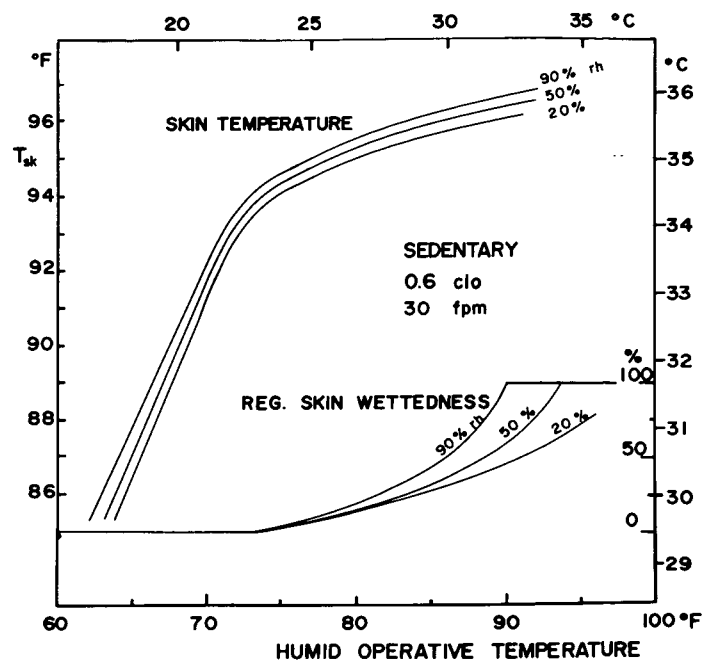

Fig. 6 The relationship between skin temperature, regulatory skin wettedness, and humid operative temperature as predicted by our model

actual environment. From Eq (10) the total heat loss from the skin surface H_{sk} may be written as

$$H_{sk} = h(T_{sk}-T_a) F_{cl} + 2.2 \ w \ h_c \ (P_{sk}-P_{dew})F_{pcl} \ . \qquad (23)$$

Over the range 10 C-40 C (50 F-105 F) for our physiological model, the following relation is true,

$$P_{sk}-P_{dew} = 1.4 \ (T_{sk}-T_{dew}) \ . \qquad (24)$$

By combining Eqs (23) and (24), the equations defining T_{oh} in a uniform environment are

$$H_{sk} = A(T_{sk}-T_a) + B(T_{sk}-T_{dew}) \ ; \qquad (25)$$

where $\quad A = h \ F_{cl} \ , \qquad (25a)$

$\qquad B = 3.08 \ w \ h_c \ F_{pcl} \ ; \qquad (25b)$

and $\quad H_{sk} = (A+B) \ [T_{sk}-(A \ T_a + B \ T_{dew})/(A+B)] \ , \ (26)$

where by definition

$$T_{oh} = (A \ T_a + B \ T_{dew})/(A+B) \ . \qquad (27)$$

T_{oh} may be also defined as the average of T_a and T_{dew} weighted by the transfer coefficients and wettedness concerned. These definitions of T_{oh} parallel the classic definitions for operative temperature and mean radiant temperature. The use of Eq (24) will cause a variation in T_{oh} of ±2%.

In Fig. 5 for a sedentary subject the older ASHRAE effective temperature has been compared with humid operative temperature for three levels of clothing insulation (1.4, 1.0, and 0.6 clo). At 100% rh the reader will quickly recognize from Eq (27) that $T_a = T_{oh} = T_{eff}$. As the hu-

midity is dropped to 35%, the best fit between the T_{eff} (old) and T_{oh} occurs for 1 clo, which clothing insulation corresponds to garments worn by Houghton and Yaglou's subjects in 1923. The greatest discrepancy for the 1 clo curve is approximately 1 C (2 F) and occurs towards the cooler temperatures. Over the range of regulation $w_{rsw} = 0.2$ to 1.0 the agreement between T_{oh} and T_{eff} is within 1 F. In general for 1 clo, T_{eff} tends to over-exaggerate slightly the effect of humidity in the cold and underestimate its effect in warmth.

From Fig. 5 it is possible to conclude that the older effective temperature scale of ASHRAE may have rational basis. Although it was constructed originally by empirical experimentation, it can now be derived on a physical and physiological basis. The present model makes it possible to revise the older ASHRAE T_{eff} in terms of 0.6 clo instead of the original 1 clo. The value 0.6 clo is more representative of modern everyday clothing than the heavier types used in 1923 and has been proposed by Kansas State University (KSU) as a standard for comfort analysis.

In Fig. 6 average skin temperature and wettedness predicted by our model have been plotted against humid operative temperature. Over the range of evaporative regulation ($T_{oh}>75$ F or 24 C) a change of humidity range from 20% to 90% causes approximately a rise of T_{sk} of 0.6 F or 0.3 C. In general the T_{sk} curves for all humidity levels parallel each other over the range of T_{oh} ranges shown. T_{sk} is thus highly correlated with T_{oh}. Since temperature sensation, which is the basis of the ASHRAE comfort scale, has been shown experimentally (Refs 8, 9, 10) to have a close correlation with skin temperature, a similar close relationship should be expected with T_{oh}. Above T_{oh} levels of 29 C or 85 F wettedness is greatly affected by the humidity level. From Eq (26) if the net heat loss from the skin surface is constant and if the mean skin temperature had remained constant, then theoretically there will be a unique one-to-
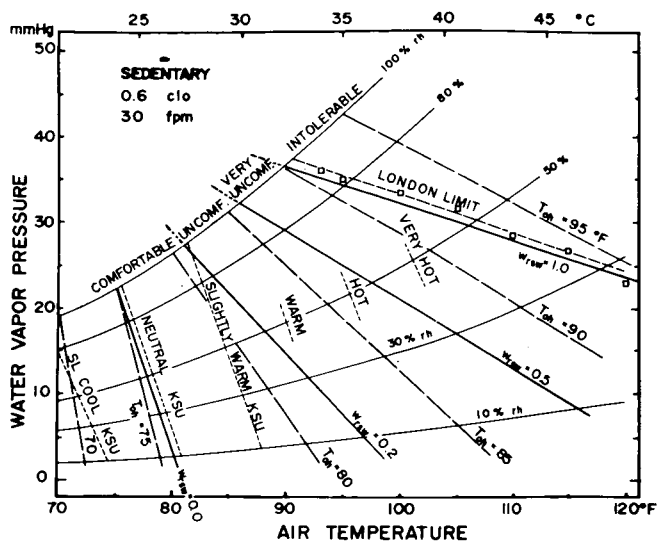
Fig. 7 *Loci of constant humid operative temperature and of constant wettedness are compared with KSU measurement of temperature sensation, the Pierce Laboratory observations of warm discomfort, and the "London" limit for heat tolerance*

one relation between w and T_{oh}, and the variation shown in the lower part of Fig. 6 will disappear. The important observation from this figure is that T_{oh} may be used as an index of T_{sk} within ± 0.5 F (0.2 C) and perhaps temperature sensation, but it is not an index of the upper limit of evaporative regulation, which is set when $w = w_{rsw} = 1$.

Comparison of Loci of Constant T_{oh} and wettedness

In Fig. 7 on a psychometric type chart with vapor pressure on the ordinate and dry bulb temperature on the abscissa lines of constant T_{oh} and constant w_{rsw} are drawn. Up to values of $w_{rsw} = 0.4$ the trends of these two types of loci are essentially parallel to each other, as may also be seen in Fig. 6. Also plotted are the latest recommended curves for "slightly cool," "comfortable" (i.e. neutral), and "slightly warm" for 1 hour exposure.[32] The T_{oh} lines may be considered somewhat similar to the old ASHRAE effective temperature scale, corrected for 0.6 clo. The KSU "comfort" line appears to fall very close to the $w_{rsw} = 0$ line or where physiological thermal neutrality occurs. Below $w_{rsw} = 0$, the remaining slope reflects the psychrometric effect of insensible evaporation from the skin and lungs. Above the $w_{rsw} = 0.5$ line the flatter slopes for the wettedness lines reflect the increasing importance of humidity.

Although a neutral temperature sensation has proved a reliable index of comfort for sedentary individuals, this same sensation may not be satisfactory to describe "comfort" during exercise or to predict "discomfort" in the zone of evaporative regulation.[10,12] In the cold temperature sensation, skin temperature, and ambient air temperature are all closely related with cold discomfort and all are associated with increased vasoconstriction before the initiation of shivering or of some type of behavioral regulation like exercising or using more clothing.

At the warm extreme the "London" limit, recommended by Ellis[2] and McArdle[33] as the upper working limit for "fit" young healthy men, has been plotted. This curve falls very near the 100% wettedness line for the new scale, al-

though their clothing and activity levels may be different from ours. As we saw in Fig. 4 the 100% limit for an unclothed subject is very close to the upper limit for the clothed subject. Low levels of exercise will not change this limit significantly since higher metabolic levels and increased sweating rates are matched by greater values for E_{max}, as a result of the higher evaporative heat transfer coefficient due to increased air movement with exercise.

For resting subjects, when unclothed, we observed[8] that a change of 5 C (9 F) in T_a would cause approximately one category in temperature sensation vote in the cold. With exercise this was raised to 7 C (13 F). When "discomfort" is used in the cold as a category basis, approximately 10 C (18 F) would be necessary to change a vote on a discomfort scale. On a "discomfort" scale both "slightly warm" or "slightly cool" are acceptable as comfortable. The body's temperature sense is twice as sensitive in the cold to changes in skin temperature as to air temperature. Fanger[34] in an extensive study of temperature sensation, for Danish and American clothed subjects, reports an average change of 3 C (5 F) in T_a for each category vote of temperature sensation, and 5 C (9 F) for exercise. In general we can say, that, when comfort is defined as a "neutral" temperature sensation, as is the case for the majority of the ASHRAE studies to date, the resulting comfort zone is governed by the ambient and skin temperatures and less by factors caused by body temperature regulation. From Pierce data[8] in the zone of body temperature regulation the sensations of comfort and discomfort and even pleasantness and unpleasantness appear to be more associated with energy exchange and temperature regulatory processes than with temperatures of the skin or air, specially.

Construction of a new "Effective" Temperature Scale

In Fig. 8, we have traced over an ASHRAE psychrometric chart #1 scales for dry bulb and wet bulb temperatures, the mmHg at saturation, and the relative humidity curves. An extra heavy line has been drawn for the 50% rh curve. Based on data computed by the Fortran program listed in the Appendix, loci of constant wettedness caused by regulatory sweating have been drawn at 5 F (dry bulb) intervals on this heavier 50% rh curve. Where normal temperature regulation is no longer possible, these scale lines are drawn parallel to the w = 1 line toward the extreme heat and to the $w_{rsw} = 0$ line toward the cold. The w = 1 line occurs on the right at the boundary between the lightly shaded area (for uncomfortable warm) and the heavily shaded area (for intolerable). The $w_{rsw} = 0$ locus for physiological thermal neutrality passes through 77 F (25 C) on the 50% rh curve. The double shading on each side of this line represents "comfortable comfort" and closely coincides with the "neutral-comfort" recommended by the KSU group.[32] The extended lighter shading covers a broader comfort zone that includes sensations of "slightly cool" and "slightly warm." The lightly shaded area to the left of the 56 F (13 C) locus represents the beginning of an "uncomfortably cold" zone. These various loci as drawn represent the proposed new "effective temperature" scales.

In the past it has been customary to name the numerical value of an "effective temperature" scale where the loci for constant sensation or heat stress intersect the 100% rh line, where $T_a = T_{wet} = T_{dew}$. We could have done the same for the new scale. Instead we have chosen tempera-
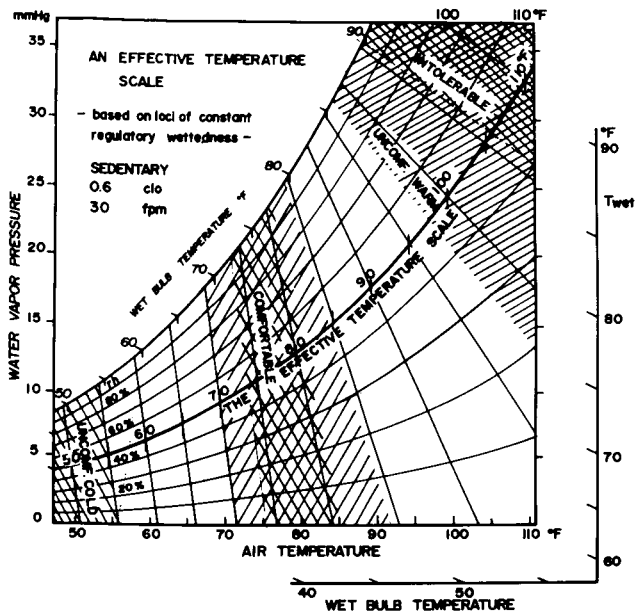
Fig. 8 A new effective temperature scale based on loci of constant wettedness caused by regulatory sweating. The lightly shaded zone on the left represents "uncomfortably cold." The lightly shaded zone on the right is "uncomfortably warm." The heavily shaded area is tolerable only for limited perioas of exposure. This chart is drawn over the corresponding section of ASHRAE Psychrometric chart #1

tures on the 50% rh line for our numerical index as these values are more easily associated with environments, which we experience in our everyday living. The probable average humidity may be considered as 50%. If one had chosen the 100% rh line, the complete range of thermoregulation would cover approximately 75 F-90 F or 24 C-32 C, a numerical range currently used in the present ASHRAE effective temperature scale. On the 50% curve the same range will extend approximately 77 F-106 F or 25 C-41 C. Above and below the outer limits of regulation the change in new "effective temperature" on our new scale will follow more closely the dry bulb towards the cold and the wet bulb temperature for the extreme heat.

By using the intersection of the loci of constant skin wettedness with the dry bulb − 50% rh curve we have essentially doubled the numerical effective temperature scale over the zone of temperature regulation by sweating. However, above and below this range ($w_{rsw} = 0 - 1.0$) the new scale parallels dry bulb. In the cold the scale temperature is approximately 1 F (0.5 C) below the dry bulb. In the hot intolerable range it is consistently 16 F (9 C) above dry bulb.

Discussion

In Fig. 9 there is tabulated the general relationship between the new T_{eff} scale for sedentary clothed subjects, the thermal sensation, the physiological responses and the health factors concerned. This general chart was first presented early in 1970 to the Society by Prof. James D. Hardy, Pierce Laboratory, at the 2nd Human Factors Symposium at the ASHRAE 1970 Semiannual Meeting in San Francisco.[35] On the right side of Fig. 9 the older T_{eff} scale, corrected by T_{oh} for 0.6 clo, has been shown for comparison.

Although the present new T_{eff} does not include the effect of radiant heating, a theoretical basis has been laid to include this additional factor. Within the range of regulatory sweating and toward the cold, the Operative Temperature and the dry bulb temperature of a uniform environment are interchangeable in predicting the physiological responses and partitional calorimetry at normal humidities. There is no useable data in the literature on the combined effects of radiant heat and high humidity in the warm-uncomfortable range.

Finally, the present two-node model is not the most sophisticated available in the current literature and its usefulness is limited for exposure times shorter than an hour. However, it does include, as we know them today, all the important parameters, coefficients and controls for man and his environment necessary to predict the quasi-equilibrium status for the *whole* body and the probable values of the three principal parameters related to the judgement of comfort and thermal sensation − skin and core temperature and skin wettedness. There are many other factors involved in the judgement of comfort that have not been considered: individual skin temperatures of the hands, feet, arms, legs and trunk as well as heart rate, blood pressure, and cardiac output. These factors are important in states of severe discomfort or during heavy exercise and all must ultimately be taken into account by a more elaborate model and by more extensive experimentation, than there is available this date in the literature.

Summary

Heat balance equations for the passive state have been developed for a human subject which include the following parameters: metabolic rate, clothing insulation, dry bulb or uniform ambient temperature, humidity, and air movement. These equations are based on our latest knowledge of man's heat exchange by radiation, convection and evaporation with his environment.

A two-node model of body temperature regulation has been developed in which skin temperature and core temperature are the controllers. The threshold temperatures
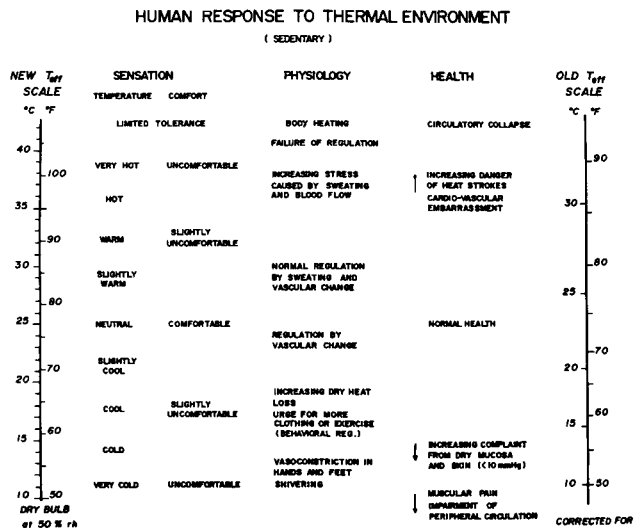

Fig. 9 An outline of response of sedentary persons to the thermal environment. (Ref Hardy, 1970)

255

and the control coefficients used are based on the most recent data available for *whole* man. This simple model incorporates the effector processes of vasoconstriction, vasodilation and sweat secretion.

The passive and control systems have been combined to predict the skin and core temperatures, the skin wettedness due to regulatory sweating, and the humid operative temperature for various activities and environmental stress.

Humid operative temperature (T_{oh}) is defined as the temperature of an imaginary environment to which the body will lose the same heat by radiation, convection and evaporation as in the actual environment. T_{oh}, which is derived rationally from the partitional calorimetry involved, is equal for practical purposes to the old ASHRAE effective temperature, when corrected to 0.6 clo instead of the original 1.0 clo. It is thus possible to derive the older ASHRAE T_{eff} scale rationally rather than empirically.

A new "effective temperature" scale, for a sedentary normally clothed (0.6 clo) subject, has been constructed based on loci of constant wettedness caused by regulatory sweating. The new T_{eff} scale is "named" numerically by the dry bulb temperatures at the intersection of its loci with the 50% rh curve, found on an ASHRAE psychrometric chart, rather than by the saturated temperature curve used before.

Algebraic		Unit	Fortran
S	Total body heat storage	W/sq m	STORE
S_{sk}	Heat storage of skin shell	W/sq m	HFSK
S_{cr}	Heat storage of body core	W/sq m	HFCR
M	Metabolic heat production	W/sq m	RM
E	Total evaporative heat loss	W/sq m	EV
E_{res}	Respired vapor loss	W/sq m	ERES
E_{sk}	Total skin evaporative loss	W/sq m	ESK
E_{diff}	Skin vapor loss by diffusion	W/sq m	EDIF
E_{rsw}	Skin evaporative loss by regulatory sweating	W/sq m	ERSW
E_{max}	Maximum evaporative loss from skin surface	W/sq m	EMAX
H_{sk}	Total heat loss from skin surface	W/sq m	
w	Total skin wettedness	N.D.	PWET
w_{rsw}	Wettedness due to regulatory sweat	N.D.	PRSW
w_{diff}	Skin wettedness due to diffusion	N.D.	PDIF
W	Rate of work accomplished	W/sq m	W
μ	Mechanical efficiency (W/M)	N.D.	WE
$R+C$	Total dry heat loss	W/sq m	DRY
T_a	Dry bulb or air temperature	C	TA
T_{dew}	Dew point temperature	C	TDEW
T_{wet}	Wet bulb temperature	C	TWET
ϕ_a	Relative humidity as a fraction	N.D.	RH
T_{sk}	Temperature of skin shell	C	TSK

Algebraic		Unit	Fortran
T_{cr}	Temperature of body core	C	TCR
T_{eff}	ASHRAE effective temperature	F	
P_a	Saturated vapor pressure at T_a	mmHg	PTTBL(TA)
P_{sk}	Saturated vapor pressure at T_{sk}	mmHg	PTTBL(TSK)
P_{dew}	Saturated vapor pressure at T_{dew}	mmHg	PTTBL(TDEW)
$\phi_a P_a$	Ambient vapor pressure	mmHg	PPHG
h	Combined heat transfer coefficient	W/(sq m)(C)	CTC
h_r	Linear radiation exchange coefficient	W/(sq m)(C)	CHR
h_c	Convective heat transfer coefficient	W/(sq m)(C)	CHC
I_{cl}	Insulation of clothing	clo	CLO
F_{cl}	Clothing thermal efficiency factor	N.D.	FCL
F_{pcl}	Permeation efficiency factor for clothing	N.D.	FPCL
m	Total body mass	kg	81.7
m_{sk}	Mass of skin shell	kg	3.4
m_{cr}	Mass of body core	kg	78.3
A	DuBois surface area	sq m	2.0
$\dot{V}_{bl}$	Rate of skin blood flow	1/(hr)(sq m)	SKBF
$\dot{V}_{bl\text{-}min}$	Min. skin blood flow	1/(hr)(sq m)	6.3
c_{bl}	Specific heat of blood	(W)(hr)/(kg)(C)	1.163
k_{min}	Min. skin heat conductance	W/(sq m)(C)	KMIN
c'_{sk}	Thermal capacity of skin shell	(W)(hr)/(C)	TCSK
c'_{cr}	Thermal capacity of body core	(W)(hr)/(C)	TCCR
t	Time	hr	TIME
Δt	Increment of time	hr	DTIM
$\dot{T}_{sk}$	Rate of change of T_{sk} ($\Delta T_{sk}/\Delta t$)	C/hr	
$\dot{T}_{cr}$	Rate of change of T_{cr} ($\Delta T_{cr}/\Delta t$)	C/hr	
ΔT_{sk}	Incremental change in T_{sk}	C	DTSK
ΔT_{cr}	Incremental change in T_{cr}	C	DTCR
Σ_{sk}	Skin signal	C	SKSIG
	When (+) warm signal		WARMS
	When (−) cold signal		COLDS
Σ_{cr}	Core signal	C	CRSIG
	When (+) warm signal		WARMC
	When (−) cold signal		COLDC
$\Delta \overline{T}_B/\Delta t$	Change in mean body temperature (rate)	C/hr	STORC
$\dot{m}_{rsw}$	Rate of sweat secretion	g/(hr)(sq m)	REGSW

USEFUL CONVERSION FACTORS

W/sq m	to	Btu/(hr)(sq ft)	multiply by 0.317
W/(sq m)(C)	to	Btu/(hr)(sq ft)(F)	multiply by 0.176
W (hr)	to	Btu	multiply by 3.413
W (hr/g)	to	Btu/lb	multiply by 1548
W (hr)/(kg)(C)	to B	Btu/(lb)(F)	multiply by 0.860

Fortran symbols not listed above are def⁻⁻
itself in terms of the ᵃᵇ⁻

REFERENCES

1. F. C. Houghton and C. P. Yaglou, Determining Lines of Equal Comfort. ASH&VE TRANSACTIONS Vol. 28, pp 163-176 and pp 361-384, 1923.
2. F. P. Ellis, Tolerable and Desirable Levels of Warmth in Warm Climates, with Special Reference to Requirements of Men in the Royal Navy, Ann. of the Roy. Coll. of Surg. of Eng., 13:369-391, 1953.
3. A. R. Lind, Effect of Individual Variation on Upper Limit of Prescriptive Zone of Climates, J. Appl. Physiol., 28:57-62, 1970.
4. C. P. Yaglou, A Method for Improving the Effective Temperature Index, ASH&VE TRANSACTIONS, Vol. 53, pp 307-326, 1947.
5. D. Minard, Prevention of Heat Casualties in Marine Corps Recruits, Military Medicine, 126:261-272, 1961.
6. J. Chatonnet and M. Cabanac, The Perception of Thermal Comfort, Int. J. of Bioclimatol. and Biometeorol., 9:183-193, 1965.
7. T. H. Benzinger, On Physical Heat Regulation and the Sense of Temperature in Man, Proc. Nat'l. Acad. Sci., 45:645-659, 1959.
8. A. P. Gagge, J. A. J. Stolwijk and J. D. Hardy, Comfort and Thermal Sensations and Associated Physiological Responses at Various Ambient Temperatures, Environmental Research, 1:1-20, 1967.
9. C—E. A. Winslow, L. P. Herrington and A. P. Gagge, Relations Between Atmospheric Conditions, Physiological Reactions and Sensations of Pleasantness, Am. J. Hygiene, 26:103-115, 1937.
   also,
   Physiological Reactions and Sensation of Pleasantness Under Varying Atmospheric Conditions, ASH&VE TRANSACTIONS, Vol. 44, pp 179-194, 1939.
10. A. P. Gagge, J. A. J. Stolwijk and B. Saltin, Comfort and Thermal Sensations and Associated Physiological Responses During Exercise at Various Ambient Temperatures, Environmental Research, 2:209-229, 1969.
11. M. Nielsen, Studies on the Relation Between Sensation of Comfort, Degree of Heating and Physiological Reactions, Contribution #3, Zoophysiological Lab., Univ. of Copenhagen, 1947.
12. A. P. Gagge, J. A. J. Stolwijk and Y. Nishi, The Prediction of Thermal Comfort When Thermal Equilibrium Is Maintained by Sweating, ASHRAE TRANSACTIONS, Vol. 75, Part II, pp 108-125, 1969.
13. R. W. Bullard, M. R. Banerjee, F. Chen, R. Elizondo and B. A. MacIntyre, Skin Temperature and Thermoregulatory Sweating: A Control System Approach, Chapter 40 in *Physiological and Behavioral Temperature Regulation*, edited by J. D. Hardy *et al*, Charles C. Thomas, Springfield, Ill., 1970.
14. J. D. Hardy and J. A. J. Stolwijk, Partitional Calorimetry Studies of Man During Exposure to Thermal Transients, J. Appl. Physiol., 21:1799-1806, 1966.
15. American Physiological Society, Proposed Standard System of Symbols for Thermal Physiology, J. Appl. Physiol., 27:439-446, 1969.
16. E. F. DuBois, Height-Weight-Surface Area, in *Basal Metabolism in Health and Disease*, see p. 183, Lea and Feibiger, Philadelphia, Pa. 1927.
17. J. A. J. Stolwijk and J. D. Hardy, Partitional Calorimetric Studies of Responses of Man to Thermal Transients, J. Appl. Physiol., 21:967-977, 1966.

18. C—E. A. Winslow, L. P. Herrington and A. P Gagge, A New Method of Partitional Calorimetry, Am. J. of Physiol., 116:641-655, 1936.
19. P. O. Fanger, Calculation of Thermal Comfort: Introduction of a Basic Comfort Equation, ASHRAE TRANSACTIONS, Vol. 73, Part II, pp 1-20, 1967.
20. K. Ibamato and Y. Nishi, Thermal Sensation Analysis and Its Application to Air-Conditioning, Bulletin #46, Faculty of Eng. Hokkaido Univ., Japan, 1968.
21. Y. Nishi and A. P. Gagge, Moisture Permeation of Clothing — A Factor Governing Thermal Equilibrium and Comfort, ASHRAE TRANSACTIONS, Vol 76, Part I, 1970.
22. A. P. Gagge, A New Physiological Variable Associated with Sensible and Insensible Perspiration, Am. J. Physiol., 120:277-287, 1937.
23. H. S. Belding and T. F. Hatch, Index for Evaluating Heat Stress in Terms of Resulting Physiological Strain, ASHRAE TRANSACTIONS, Vol. 62, pp 213-236, 1956.
24. D. F. Brebner, D. McK. Kerslake and J. L. Waddell, The Diffusion of Water Vapour Through Human Skin, J. Physiol., 132:225-231, 1956.
25. A. B. Goodman and A. V. Wolf, Insensible Water Loss From the Human Skin as a Function of Ambient Vapor Concentration, J. Appl. Physiol., 26:203-207, 1969.
26. A. E. Burton and O. G. Edholm, *Man in a Cold Environment*, Edward Arnold Publishers, London, 1955.
27. J. A. J. Stolwijk and J. D. Hardy, Temperature Regulation in Man — A Theoretical Study, Pflügers Archiv, 291:129-162, 1966.
28. B. Saltin, A. P. Gagge, and J. A. J. Stolwijk, Body Temperatures and Sweating During Thermal Transients Caused by Exercise, J. Appl. Physiol., 28:318-327, 1970.
29. P. E. McNall, P. W. Ryan, F. H. Rohles, R. G. Nevins and W. Springer, Metabolic Rates at Four Activity Levels and Their Relationship to Thermal Comfort, ASHRAE TRANSACTIONS, Vol. 74, IV. 3. 1-17, 1968.
30. Y. Nishi and A. P. Gagge, Direct Evaluation of Convective Heat Transfer Coefficient by Naphthalene Sublimation, 29: 830-838, 1970, J. Appl. Physiol.,1971.
31. A. P. Gagge, 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.
32. F. Rohles and R. Nevins, The Nature of Thermal Comfort for Sedentary Man, ASHRAE 1971 Semiannual Meeting, Philadelphia, Pa., 1971.
33. B. McArdle, W. Dunham, H. E. Holling, W. S. S. Ladell, J. W. Scott, M. L. Thomson and J. W. Weiner, The Prediction of the Physiological Effects of Warm and Hot Environments, Med. Res. Council (Eng.), report #47—391, 1947.
34. P. O. Fanger, *Thermal Comfort, Analysis and Applications in Environmental Engineering.* Danish Technical Press, Copenhagen, 1970.
35. J. D. Hardy, Thermal Comfort and Health, 2nd Human Factors Symposium, ASHRAE 1970 Semiannual Meeting in San Francisco, to be published as an ASHRAE Bulletin, 1970.

61<

APPENDIX

AN ANNOTATED FORTRAN PROGRAM FOR A CORE-SHELL
MODEL OF HUMAN TEMPERATURE REGULATION

```
C              THE ENVIRONMENTAL FACTORS AND THE TYPE OF ACTIVITY
C          ARE DEFINED IN AN INITIAL 'READ' STATEMENT BY
C                  RM,WE,CTC,CLO,TA,RH
C          A 'DO' STATEMENT MAY BE USED FOR TA OR RH OR BOTH

C STANDARD MAN IS 81.7KG 1.77M HT AND 2.0 SQ.M DUBOIS AREA
C IN PROGRAM 1.163=CONV. FACTOR KCAL/HR TO WATTS
C            1.163=SPECIFIC HEAT OF BLOOD IN W*HR/(L*C)
C            0.7=LATENT HEAT IN WATT*HR/G
C            0.97=SPECIFIC HEAT OF BODY IN W*HR/(KG*C)
C            2.2=LEWIS RELATION

C INITIAL CONDITIONS - BODY IN PHYSIOL. THERMAL EQUILIBRIUM
        REAL KMIN
        TSK=34.1
        TCR=36.6
        ERES=0.0023*RM*(44.-RH*PTTBL(TA))
C  PTTBL(TA) IS FUNCTION FOR VAPOR PRESSURE(MMHG) AT TEMP. TA DEG. C
        EDIF=5.0
        EV=ERES+EDIF
        ERSW=0.0
        EDRIP=0.
C.  EDRIP IS UNEVAPORATED SWEAT FROM SKIN SURFACE
        WRSW=0.0
        CHR=5.23
        CHC=CTC-CHR
        WK=WE*RM
C    FOR NEXT TWO CARDS SEE REF.21
        FCL=1./(1.+0.155*CTC*CLO)
        FPCL=1./(1.+0.143*(CTC-CHR)*CLO)
C KMIN = MIN. CONDUCTANCE IN W/(SQ.M*C)
        KMIN=5.28
C SKBFN = NORMAL SKIN BLOOD FLOW L/(SQ.M*HR)
        SKBFN=6.3
        SKBF=SKBFN
        TIME=0.0

  600 CONTINUE

C HEAT BALANCE EQUATIONS  FOR PASSIVE SYSTEM

C HEAT FLOW FROM CORE TO SKIN TO AIR IN W/SQ.M
        HFCR=RM-(TCR-TSK)*(KMIN+1.163*SKBF)-ERES-WK,
        HFSK=(TCR-TSK)*(KMIN+1.163*SKBF)-CTC*(TSK-TA)*FCL-(EV-ERES)

C THERMAL CAPACITY OF SKIN SHELL FOR AV. MAN IN W.HR/C
        TCSK=0.97*3.4
C THERMAL CAPACITY OF CORE FOR AV. MAN IN W.HR/C
        TCCR=0.97*78.3
C CHANGE IN SKIN SHELL AND CORE IN DEG. C PER HOUR
        DTSK=(HFSK*2.0)/TCSK
        DTCR=(HFCR*2.0)/TCCR
C NOTE UNIT OF TIME IS ONE HOUR
        DTIM= 1./60.
```

62

```
C TO ADJUST INTEGRATION OVER SMALL STEPS IN DTSK AND DTCR
      U=ABS(DTSK)
      IF(U*DTIM-0.1) 873,873,874
  874 DTIM=0.1/U
  873 CONTINUE
      U=ABS(DTCR)
      IF(U*DTIM-0.1) 973,973,974
  974 DTIM=0.1/U
  973 CONTINUE
      TIME=TIME+DTIM
      TSK=TSK+DTSK*DTIM
      TCR=TCR+DTCR*DTIM

C CONTROL SYSTEM
C DEFINING SIG. FOR CONTROLS FOR VASO-CONSTRICT.-DILATION
C FROM SKIN
      SKSIG=(TSK-34.1)
      IF(SKSIG)900,900,901
  900 COLDS=-SKSIG
      WARMS=0.0
      GO TO 902
  901 COLDS=0.0
      WARMS=SKSIG
C FROM CORE
  902 CRSIG=(TCR-36.6)
      IF(CRSIG)800,800,801
  800 COLDC=-CRSIG
      WARMC=0.0
      GO TO 802
  801 WARMC=CRSIG
      COLDC=0.0
C FACTORS 0.5(COLD) AND 75.(WARM) GOVERN STRIC AND DILAT SEE NEXT 2 CARDS
  802 STRIC=0.5*COLDS
      DILAT= 75.*WARMC
C  CONTROL OF SKIN BLOOD FLOW
      SKBF=(SKBFN+DILAT)/(1.+STRIC)
C CONTROL OF REG. SWEATING
C REGSW IN G/(SQ.M*HR)
C DURING REST
      IF(RM-60.)401,401,402
  401 REGSW=100.*WARMC*WARMS
      GO TO 403
C DURING EXERCISE
  402 REGSW=250.*WARMC+100.*WARMC*WARMS
C BULLARD VAN BEAUMONT EFFECT, MODIFIED BY STOLWIJK
  403 ERSW=0.7*REGSW*2.**((TSK-34.1)/3.)
C TO AVOID IMPOSSIBLE SOLUTIONS MAX. REGSW IS 16 G/MIN
      IF(ERSW -500.)404,404,100

C  WRSW IS REG. SWEAT IN 100CC UNITS PER MAN FOR TIME EXP.
  404 WRSW= WRSW+ (ERSW*2.0/(0.7* 100.))*DTIM
      EMAX=2.2*CHC*(PTTBL(TSK)-RH*PTTBL(TA))*FPCL
      PRSW=ERSW/EMAX
      PWET=(0.06+0.94*PRSW)
```

63

```fortran
C NOTE TOTAL EVAPORATIVE LOSS FROM SKIN IS PWET*EMAX
      EDIF=PWET*EMAX-ERSW
      EV=ERES+ERSW+EDIF
      IF(ERSW-EMAX)220,220,201
  201 EDRIP=ERSW-EMAX
      EV=ERES+EMAX
      ERSW=EMAX
      EDIF=0.0
      PRSW=1.0
      PWET=1.0
  220 CONTINUE

C TO CALCULATE QUASI-EQUILIBRIUM AFTER ONE HOUR EXPOSURE
      IF(TIME-1.00) 600,601,601
C THE EXPOSURE TIME (1.00) CAN BE CHANGED UP OR DOWN TO 0.25
  601 CONTINUE
      DRY=CTC*(TSK-TA)*FCL
C STORE IN WATT PER SQ.METER   STORC IN DEG.C PER HOUR
C STORE IS ALSO EQUAL TO HFSK PLUS HFCR
      STORE=RM-EV-DRY-WK
      STORC=STORE*2.0/(81.7*0.97)

C CALCULATION TWET FROM TA AND RH
      TWET=TA
 1204 E=RH-(PTTBL(TWET)-0.00066*760.*(TA-TWET)*
     X  (1.0+0.00115*TWET))/PTTBL(TA)
      IF(E)1203,1202,1202
 1203 TWET=TWET-0.10
      GO TO 1204
 1202 CONTINUE

C CALCULATE TDEW
      TDEW=-10.
 1702 X= PTTBL(TWET)-(TA-TWET)/2.
      DDEW=(PTTBL(TDEW)-X)
      IF(DDEW)1700,1701,1701
 1700 TDEW=TDEW+0.10
      GO TO 1702
 1701 CONTINUE

C   THE NISHI ENVIRONMENTAL EQUATIONS
      XA=FCL*CTC
      XB=2.2*1.4*CHC*FPCL
      TOH=(XA*TA+PWET*XB*TDEW)/(XA+PWET*XB)

C ADD CARDS FOR PRINTOUT OF HEAT EXCHANGE DATA
C ADD CARDS FOR PRINTOUT OF PHYSIOLOGICAL DATA

  100 CONTINUE
      END
```

6*

DISCUSSION

P.E. McNALL, JR., (Kansas State University, Manhattan, Kan.): I think this is a very good piece of work, Dr. Gagge, and I wonder now whether this scale shouldn't have a name. In other words, call it something different from "effective temperature" since this does have a meaning to many of us in the Society and if we could think up a good new term for it, it might increase its use and solve the problem of confusion in the future.

I would suggest that if we have to borrow a term, that we borrow one from some other area that is not well known to ASHRAE. Perhaps the physiological literature has terms which we could use.

Also, as many of you know, we are rewriting the chapter on comfort in ASHRAE HANDBOOK OF FUNDAMEN- TALS. This new scale certainly should be covered there. Let's see if we can't do something with that terminology so that we don't add to the confusion in the future.

J.D. HARDY, (John B. Pierce Foundation, New Haven, Conn.): Dr. McNall has raised the important point of what to call Dr. Gagge's proposed "new effective temperature scale." At the 1970 Semiannual Meeting, using many of the considerations presented in Dr. Gagge's paper, I suggested the term "Comfort-Health-Index" or "CHI" to replace the usual THI or "Temperature-Humidity-Index." Since the proposed scale is not truly a temperature scale perhaps the term index might be more appropriate.

DR. GAGGE: We can answer rather simply both Dr. McNall's and Dr. Hardy's question, whether or not to call our proposed scale a "temperature" or an "index." "Tem- perature" should refer to a rationally derived or measurable quantity, and one with a defined physical meaning. "Oper- ative" temperature, as described by us in the past had such a meaning, can be used in a heat balance equation describ- ing man's heat exchange with his environment. "Effective Temperature," as used by ASHRAE, should have been calssified as an "Index" but not as a "Temperature," since it never has had any meaning in a heat balance equation. Our present proposal, illustrated in Fig. 8, must be classi- fied as an "index" as the indicated temperature has no meaning in a heat balance equation.

The use of the words "Comfort-Health," as descriptive names for an index, has its virtues. The proposed new effec- tive temperature scale has a psychological, physiological and health basis as seen in Fig. 9 and, by using an equivalent 50% RH value for all scale values, the index will coincide more with man's human every day experience. We accept Dr. Hardy's suggestion that an appropriate name for the scale illustrated in Fig. 8 is a "Comfort-Health-Index" (CHI).

The concept of "Humid Operative Temperature" (T_{oh}), as an imaginary temperature at saturation, is a rationally derived temperature and does have a meaning in the heat balance equation. This temperature has biometeorological significance and by definition relates the exchange of heat from average skin temperature $\overline{T}_{sk}$ to the environment by radiation, convection and evaporation.

F.H. FULLER (E.I. duPont de Nemours, Wilmington, Del.): I would agree that you should change the name of effective temperature to something else, except that it has taken 15 to 20 years to get my management to think in terms of

effective temperature. Effective temperature is an accepted term that would be hard to change.

Some years ago, Mosher of Kodak put together a chart based on some old work of ASHRAE. He plotted maximum effective temperature versus metabolic rate. This chart has been quite useful to the ventilation and air-conditioning engineers. Do you intend to develop such a plot or to ex- tend your research so that you can develop such a plot?

DR. GAGGE: I am unfamiliar with Dr. Mosher's work at Kodak and evidently he did not publish his ideas in our TRANSACTIONS. In general the present proposed Effec- tive Temperature Index (ETI) and as illustrated in our Fig. 8 has significance only for sedentary subjects. Its re- lationship to the corresponding psychological, physiological and health factors is shown in Fig. 9. As was the case for the old ET, it is possible to average rationally the combined thermal effects caused by various combinations of clothing, air movement and radiant heating and to express as a single temperature on the abscissa of Fig. 8, by using a concept we suggested several years ago "Standard Operative Tem- perature" (see Am. J. Physiol. 131, 93-103, 1940 and J. Appl. Physiol. 23, 248-258, 1967).

For each new level of activity (increasing metabolic rate) there will be both a new and lower ambient temperature for comfort as well as a lower skin temperature for a "neutral" Temperature Sensation, as was shown by Fanger (1967) and McNall et al. (1967). In an earlier paper (see Ref 12) we suggested a skin wettedness of 20% could serve as a common index for an upper limit for comfort over quite a range of exercise activities.

The use of a physiological basis for an "Effective Tem- perature" scale is not new. Yaglou, in paper #1319 ASH&VE TRANSACTIONS 1947, proposed lines of con- stant skin temperature be used as an index. The difficulty of using skin temperature as a basis of thermal comfort has been shown by Chatonet and Cabanac (Ref 6) who demon- strated that temperature and comfort sensation are affected also by changing internal body temperature with activity.

Our recommendation for a proposed CHI is limited in the present paper to sedentary conditions for which there is a sound and extensive experimental basis thanks to the ef- forts of the KSU-ASHRAE studies under the direction of Dr. Ralph Nevins and Dr. Preston McNall. Analytically, we are also proposing a method, that is rational physically, to extend our predictions to other activity levels for which there is still a need for a great deal more experimental data.

P.O. FANGER, (Technical University of Denmark, Copen- hagen, Denmark): The authors should be complimented on their important and most interesting research work.

Their two-node model is derived in an elegant way by combining equations describing in detail the passive and the controlling system of man's thermo-regulation. This model, including the corresponding computer program, has here been used to develop a new effective temperature scale which will undoubtedly be a most useful tool in the future, also for other applications.

In my opinion, the authors present a solid foundation for the new effective temperature diagram, applicable to sedentary subjects at low velocity, clothed in 0.6 clo, and mean radiant temperature equal to air temperature. On the basis of the same model, analogous diagrams can be drawn

65

up showing the significance of humidity at other combinations of the parameters.

I am not, however, in agreement with the authors' comparison with the old ASHRAE effective temperature scale, which in my opinion accords far too great an influence to humidity in the comfort zone.

The authors conclude tha the old ASHRAE effective temperature scale may have a rational basis. Admittedly, the old scale gives a significantly greater humidity dependence than the present study, but according to the authors, this is due to the clothing at the time being 1 clo, and it is maintained that the difference is slight when converted to 0.6 clo.

It should be remembered, however, that Yaglou et al. drew up not only a "normal" effective temperature scale (Ref a, 1925) for subjects wearing customary indoor clothing ($\sim$ 1 clo), but also a "basic" effective temperature scale (Ref 1), which was drawn up already in 1923 for semi-nude subjects. The two scales show a moderate difference, but contrary to the clothing correction in the present paper, Yaglou found greater humidity dependence for the semi-nude persons than for the clothed.

From the curves of the new effective temperature lines in the comfort zone (and colder) it will be found that a change from RH=0% to RH=100% corresponds to a temperature change of $\sim$ 5F (Fig. 8) compared with 12-16 F from Yaglou's diagrams.

I therefore take the results of the present excellent investigation as a new confirmation of the far too great humidity dependence in the old effective temperature scale at low and moderate temperatures. The old scale has for many years been the cause of numerous misunderstandings among engineers. It should be replaced as soon as possible by a scale such as the present one which shows the humidity influence on a rational basis.

One final question: In the paper the authors derive expressions both for wettedness and for humid operative temperature. I should like to know why the authors finally chose to base the new scale on loci of constant wettedness and not on loci of constant operative temperature.

REFERENCES
a. C.P. Yaglou and W. Edw. Miller, Effective Temperature with Clothing. ASH&VE Vol 31, pp 89-99, 1925.

DR. GAGGE: We agree with everything you say about the older ET scale. A careful comparison of our humid operative temperature scale (T_{oh}) for 1 clo with the old ET value does correct the older scale in the directions you indicate are so necessary. A more exact equation (see Eq 27) relating the vapor pressure gradient to the difference between T_{sk} and T_{dew} perhaps would improve the accuracy of this correction.

We pointed out in our text that, if skin temperature had remained constant over the whole range of skin wettedness from 0 to 100%, then lines of constant wettedness would have coincided with lines of constant humid operative temperature (T_{oh}). However, such a model ($\overline{T}_{sk} \sim$ const) would require an infinitely thin skin shell and a uniformly heated core. This does not exist in nature. Since skin blood flow is so essential for body temperature regulation, and since the skin shell has a finite mass, $\overline{T}_{sk}$ must vary with the environmental temperature and thus in a "living" model lines of constant wettedness never coincide with lines of constant T_{oh} except when there is no skin sweating. As said in text, loci of constant wettedness are associated more closely with "Discomfort." Loci of constant skin temperature follow loci of constant T_{oh}, and hence with temperature sensation, which is the classical ASHRAE definition of comfort. Since health and heat tolerance considerations are governed by skin wettedness and since in the comfort zone, while sedentary, there is little significant difference between the two concepts, we chose loci of constant wettedness as the physiological basis for the new CHI.

THERMOREGULATORY SET POINT DURING EXERCISE:

A BEHAVIORAL APPROACH

M. CABANAC, D. J. CUNNINGHAM, AND J. A. J. STOLWIJK

John B. Pierce Foundation Laboratory, Yale University School of Medicine

THERMOREGULATORY SET POINT DURING EXERCISE:

A BEHAVIORAL APPROACH[1]

M. CABANAC,[2] D. J. CUNNINGHAM,[3] AND J. A. J. STOLWIJK

John B. Pierce Foundation Laboratory, Yale University School of Medicine

Response to a peripheral thermal stimulus has been shown to be an indicator of thermal status with respect to the thermoregulatory set point. The subjects were provided with a glove perfused with water, adjustable in temperature between 15° C. and 45° C. The subjects were asked to maintain the glove temperature at the level they considered most pleasant. In response to environmental temperatures ranging 15–45° C. and to exercise at levels of 500 and 1,000 kgm/min, the selected glove temperature ranged 20°–40° C. The preferred glove temperature depended strongly on internal body temperature; it was affected to a lesser extent by mean skin temperature and not at all by exercise alone. The results suggest there is no change in thermoregulatory set point during exercise.

M. Neilsen (1938) was one of the first to observe that the rectal temperature of working subjects stabilized at values which were related to the level of exercise and largely independent of the ambient temperature. This observation was subsequently confirmed in later studies conducted at the same laboratory and by several other groups of investigators (Nielsen, 1966; Nielsen & Nielsen, 1962; Robinson, 1963; Stolwijk, Saltin, & Gagge, 1968). These findings led to the conclusion that the internal temperature of exercising subjects is regulated at a higher than normal level in a manner similar to that observed in fever. This sustained elevation in body temperature during exercise was viewed as being beneficial for the enzymatic reactions occurring within the working muscle, resulting in an increase in the efficiency of the work (Asmussen & Bøje, 1945).

This concept of a reset of the thermostat to a higher temperature during exercise has been challenged, however, on the basis of both experimental and theoretical arguments. The recording of evaporative heat loss in the dog or man has shown that an increase in panting or sweating occurs during exercise at a given internal temperature (Hammel, 1968; Jackson & Hammel, 1963; Kuno, 1956; Minard, 1963; Minard & Copman, 1963; van Beaumont & Bullard, 1963, 1966). Therefore, an opposing theory has also been proposed which states that exercise may lower the thermoregulatory set point. This lowering of the set point would reduce the risk of hyperthermia at a time of considerable heat production. In the context of this paper the thermoregulatory set point is defined as that internal temperature at which neither heat loss mechanisms nor heat conservation mechanisms are activated. Internal temperatures above the set point then produce sweating and vasodilatation in man, and below the set point vasoconstriction and shivering are activated.

The purpose of the present work was to explore the set point for temperature regulation during exercise using a behavioral indicator. Previous studies have shown that a thermal sensation is pleasant to a subject whenever the thermal stimulus offered tends to improve or to restore homeothermia. When cold stimuli are pleasant and warm stimuli unpleasant, the internal temperature is found to be above the set point; when warm stimuli are perceived as pleasant and cold stimuli as unpleasant, the internal temperature is below the set point (Cabanac, 1969). We have utilized this behavioral phenomenon in an attempt to dem-

onstrate the presence of a change in the set point level with exercise. We have, in addition, studied the effect of changes in the internal temperature and/or the mean skin temperature upon the pleasantness or unpleasantness of a peripheral thermal sensation as an index of the set point during periods of rest and exercise.

METHOD

The experiments were conducted in a climatic chamber with controlled dry-bulb and wet-bulb settings. The ambient temperature of the chamber could be rapidly changed over a range of dry-bulb temperatures 10°–48° C., with the relative humidity maintained at about 35%.

Six men and three women served as subjects in 15 experiments. The men wore cotton trunks and the women wore cotton shorts and a sleeveless shirt.

Skin temperatures were measured at 10 locations on the body by thermocouples supported on tygon-coated metal rings and held in place with elasticized straps. Skin thermocouples were placed on the forehead (1), upper chest (2), the hypogastric (3), dorsal lumbar (4), and scapular areas (5), on the upper arm (6), forearm (7), palm (8), thigh (9), and calf (10). At Locations 2, 5, 7, 8, and 9 calibrated copper-tellurium heat-flow discs were placed adjacent to the skin thermocouples for measurements of the rate of local heat transfer. Each heat-flow disc was soldered to a copper tube which functioned as a constant temperature heat sink. The five copper tubes were connected in series by rubber tubing and perfused with water at a rate of about 300 ml/min. The water in the perfusion system was cycled through a constant temperature water bath which was set to maintain the water temperature at 26° C. Conductance values are expressed in mcal.cm.$^{-2}$ sec.$^{-2}$ °C.$^{-1}$, on the basis of the ΔT between the water and the internal temperature. A tympanic thermocouple recorded core temperature and a thermocouple-tipped catheter was introduced into the esophagus down to the right atrial level in most of the experiments for an additional measurement of internal temperature.

The subjects wore on the right hand a gauntlet-length neoprene glove which was held in place by a strap that passed diagonally across the chest and back and was supported on the left shoulder. The entire hand, wrist, and adjacent areas of the forearm were immersed in water within the glove. Water flowed into the glove via connections at the tip of each of the five fingers and drained from a single outlet on the cuff. The glove was continuously perfused at a flow rate of 300–400 ml/min by means of a centrifugal pump placed between the water supply and the glove. The water was pumped from and returned to one of two 5-gal. constant temperature water baths. One bath contained a thermostatically controlled heating element and a stirrer which maintained the bath at 50° C. The other bath was held at 10° C. by a stainless-steel cooling coil connected to a thermostatically controlled compressor cooling unit. A small metal box with an on-off switch, which controlled two electrically driven solenoid valves, was suspended in front of the subject. The position of the switch determined whether the glove was connected to the 50° C. or the 10° C. water bath and thus allowed the subject to regulate the temperature of the water within the glove. The temperature of the two baths permitted water temperatures in the glove over a range of 15°–44° C. Prior to each run the subject was instructed to maintain throughout the course of the experiment a water temperature within the glove which he found to be "most pleasant." The glove temperature was measured by a thermocouple located close to the outflow connection on the cuff.

The subjects were seated on a bicycle ergometer with the right arm and the glove extended at their side and exercised for periods of 15-20 min. during the experiment. The work level for the men was roughly 1,000 kgm/min and for the women, 500 kgm/min., at a pedaling rate of 60 rpm.

The 10 skin temperatures and 5 heat flow rate measurements, as well as the tympanic temperature, esophageal temperature, temperature of the water in the glove, dry-bulb, and wet-bulb temperature, were recorded once each minute.

RESULTS

Resting Subjects

The glove temperatures which a male subject found to be most pleasant while both his mean skin and internal temperatures were falling over a period of 80 min. is shown in Figure 1. During the time that the chamber temperature was falling from 45° to 17° C. with a corresponding fall in mean skin and core temperature, the glove temperature selected by the resting subject increased progressively from 29° to 37° C. The results of this experiment demonstrate an inverse relationship between the direction of change in the glove temperature which a subject selects as being most pleasant and the direction of change in his internal and mean skin temperature.

Figure 2 presents the results of an experiment in which the ambient temperature was rapidly cycled in order to significantly alter the mean skin temperature while the internal temperature remained relatively constant. An inverse relationship between the
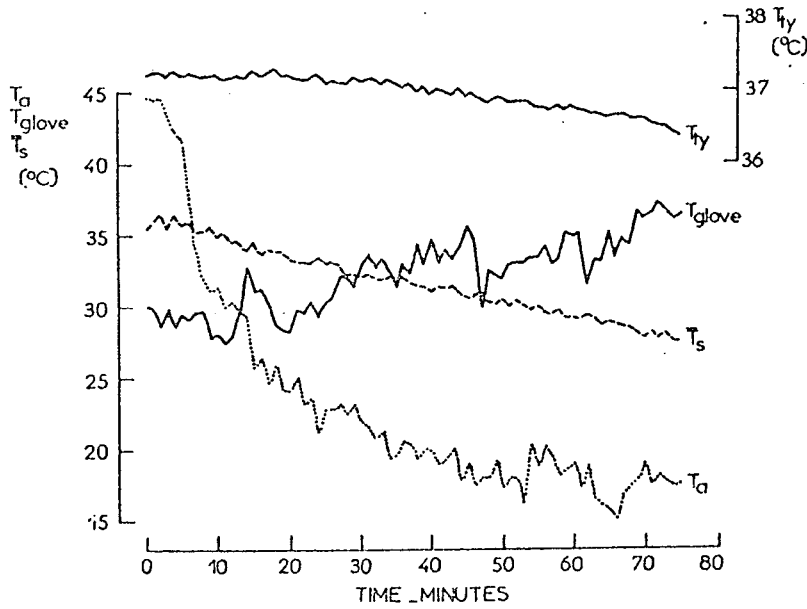
Fig. 1. Glove temperatures preferred by a resting subject while his mean skin and internal temperatures are falling, during a lowering of the ambient temperature from 45° to 17° C.

selected glove temperature and the mean skin temperature was repeatedly demonstrated during the time that the core temperature remained essentially unchanged. In this figure at 30 and 80 min. we see the subject select a low glove temperature with an internal temperature below 37° C., presumably only in response to the rising skin temperature. Note, also, that at 60 min. the largest part of the reduction in the selected glove temperature occurred even before there was a recordable rise in the mean skin temperature but during a period when the superficial tissues would be undergoing a high rate of temperature change.

Exercising Subjects

The glove temperature which a subject found to be most pleasant during a 20-min. period of exercise with a resulting increase in both internal and mean skin temperature is shown in Figure 3. A marked reduction in the preferred water temperature is associated with the rise in both internal and mean skin temperatures. Note that prior to and following the period of exercise the subject selected progressively higher glove temperatures during the time that his internal and mean skin temperatures were falling. A

similar relationship was demonstrated in Figure 1.

In Figure 4 are shown the responses of a subject during rest and exercise while exposed to an ambient temperature of 12° C. During the first half of the exercise period the internal and mean skin temperature remained constant and there was no marked change in the glove temperature. About 10 min. after the onset of exercise, the internal temperature began to rise, while the mean skin temperature remained stable. At this time the subject selected a markedly lower glove temperature although his mean skin temperature remained at about 27° C. Note also that the time at which the subject began to return the glove temperature to 42° C. corresponds with a fall in the internal temperature with the mean skin temperature essentially unchanged. In Figure 4 are also plotted the palmar conductance values. The palm remains highly vasoconstricted throughout the 25-min. exercise period, even during the time that there is a marked reduction in the selected glove temperature. These results indicate that a subject will change from 40° C. to 20° C. water as being most pleasant in response to a rise in the internal temperature even though the
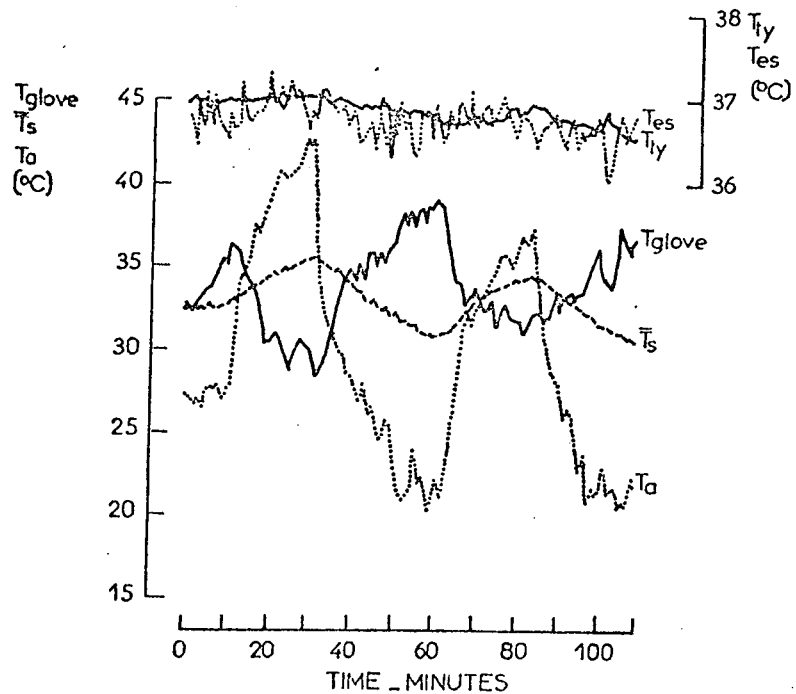
FIG. 2. Selected glove temperatures associated with rapid cycling of the ambient temperature, resulting in significant changes in the mean skin temperature while the internal temperature remains relatively unchanged.

hand is highly vasoconstricted. Figures 5 and 6 illustrate the effect of a combination of a rising core temperature and a falling mean skin temperature on the choice of water temperature. This condition was observed during the period of exercise in two experiments. In both experiments the subjects continued to select a lower glove temperature during a rise in the internal temperature, even though the mean skin temperature was falling at this time.

Table 1 lists six relationships between internal, mean skin, and glove temperatures which we observed along with the number of times that each combination was recorded experimentally.

The importance of the manner in which a subject is prepared for these experiments was demonstrated in the results obtained on two subjects, one female and one male. The glove temperatures selected by the female subject during changes in mean skin and internal temperatures are shown in Figure 7. While the subject did show a slight trend toward an increase in the glove temperature

with a fall in mean skin temperature, and a slight reduction in the glove temperature with a rise in core temperature, the range of temperatures which she selected was considerably narrower than was characteristic of other subjects and was no greater than she would allow as short-term fluctuations. This subject, however, never experienced the full temperature range of the glove available to her, even when encouraged to do so during the early part of the experiment. The limited range of glove temperatures which she selected throughout the 2 hr. of the experiment was within that which she would allow during the initial familiarization period and may be related to her restriction in the range of water temperatures during a marked fall in the mean skin temperature and a rise in internal temperature. Furthermore, the one male subject who maintained his glove temperature within a narrow range during significant changes in internal and mean skin temperatures also did not experience the full range of glove temperatures available to him prior to the experi-
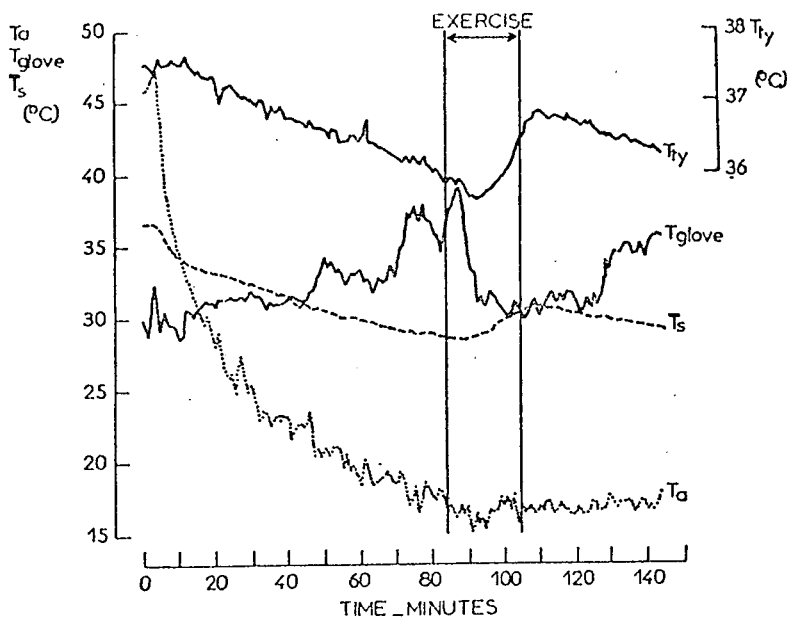
FIG. 3. Water temperatures chosen by a subject as being most pleasant during a period of rest and exercise while the ambient temperature is lowered from 47° to 17° C.

ment or during the early part of the run. In view of the nature of the responses of these two subjects, we feel that it is important that all subjects experience the upper and lower limits in the glove temperature available to them prior to the experiment so that they are familiar with the full range of water temperatures which they may select.

DISCUSSION

The results of these studies indicate that a change in internal temperature will result
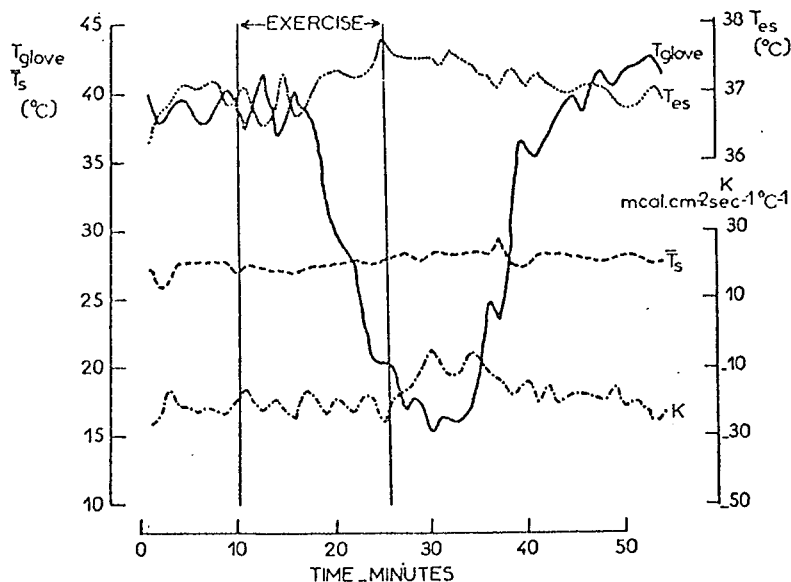
FIG. 4. Glove temperatures and palmar conductance values during and following exercise, in association with changes in the internal temperature and a constant mean skin temperature.
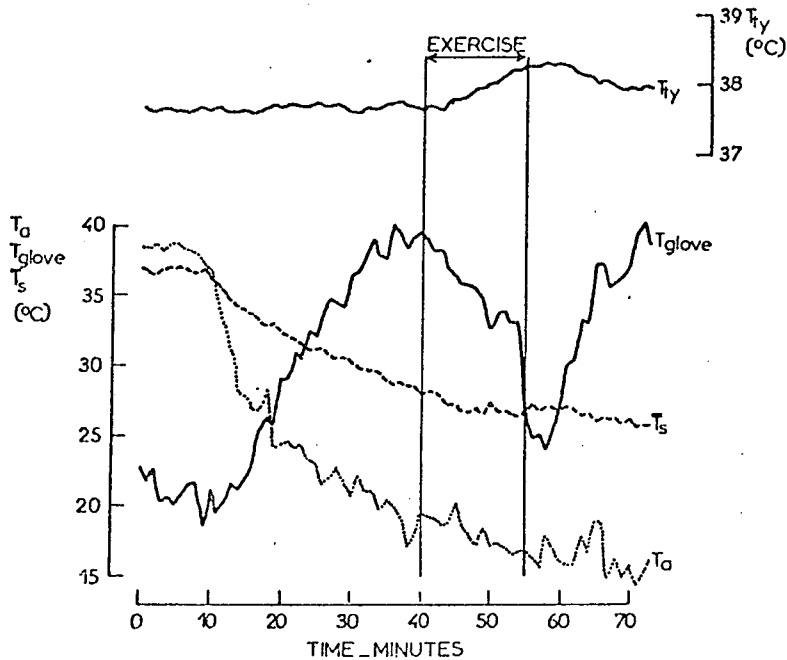
Fig. 5. Selected glove temperatures during and following exercise with resulting changes in the internal temperature and a continuing decline in the mean skin temperature.

in a compensating change in the selected glove temperature, i.e., with a rise in internal temperature the subject selects a lower water temperature as being most pleasant, while with a fall in internal temperature a higher water temperature is chosen. This relationship has been found to exist when a change in mean skin temperature parallels the change in core temperature, without a change in mean skin temperature, or with a continuing fall in mean skin temperature in association with a rise in internal temperature. The present results as well as earlier reports (Chatonnet & Cabanac, 1965; Gagge, Stolwijk, & Hardy, 1967) support the concept that the appreciation of "thermal comfort" is determined by the combination of level and rate of change of peripheral thermal input and the status of the internal temperature with respect to its set point. Thus, if there is a displacement of the internal temperature above its set point then peripheral thermal stimuli which are recognized as "warm" are also perceived as unpleasant or uncomfortable. The results reported here allow extension of this finding to include the effect of mean skin tempera-

ture and of exercise. In the absence of a deviation of the core temperature from its set point the mean skin temperature appears to be able to determine the preferred stimulus temperature as shown in Figure 2. However, the effect of a deviation of central temperature on the choice of stimulus temperature is able to override any such effect of the mean skin temperature as can be seen in Figure 4 where the skin temperature remains low throughout the experiment. The inverse relationship which we have observed between the direction of change in the mean skin temperature and the preferred glove temperature in the absence of any change in the internal temperature can be interpreted as resulting from a shift in the set point temperature, i.e., with a rise in the mean skin temperature there is a lowering of the set point while with a fall in the mean skin temperature there is an elevation in the set point. These shifts in set point would encourage corrective behavioral responses and thus tend to prevent the mean skin temperature from rising or falling into extreme temperature ranges.

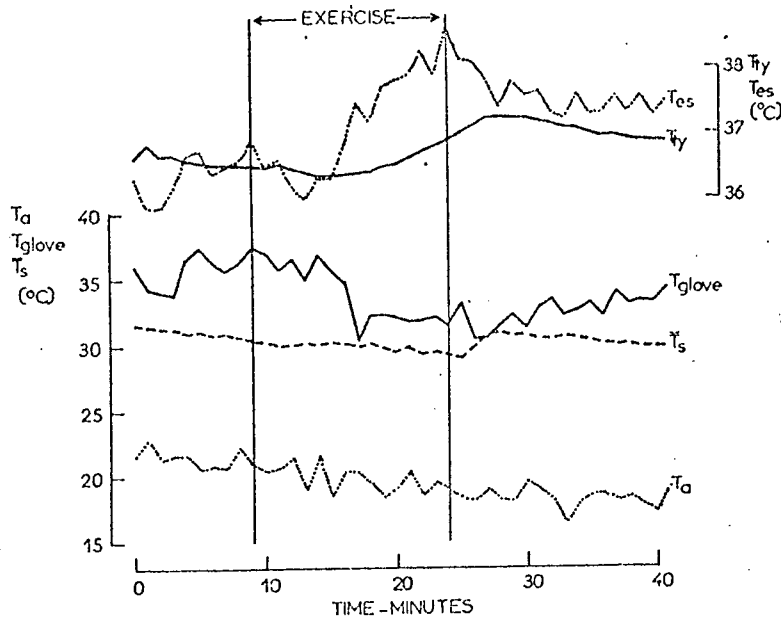It has been proposed that a change in

FIG. 6. Glove temperatures selected before, during, and following exercise in a cool environment with slowly falling mean skin temperature.

central set point temperature occurs with the onset of exercise. In view of this, we have looked for a significant change in the glove temperature associated with the beginning and the end of the exercise period. In all experiments (Figures 3–6) it is apparent that no significant change in the preferred glove temperature was observed concurrent with the beginning and/or the end of the exercise period. Those changes in the preferred glove temperature which were observed during exercise did not occur within the first few minutes of the exercise period, but rather appear to be related to the rise in internal temperature which was observed about 10 min. after the onset of exercise

TABLE 1

RELATIONSHIPS BETWEEN INTERNAL, MEAN SKIN, AND GLOVE TEMPERATURES

Internal temperature	M skin temperature	Glove temperature	Number of times observed
↓	↓	↑	9
↑	↓	↓	2
↓	→	↑	6
↑	→	↓	6
→	↑	↓	7
→	↓	↑	5

Note.—Abbreviations: rise, ↑; fall, ↓; no change, →.

and to the fall in internal temperature which occurred about 10 min. after the end of the exercise period. If we assume that the selected glove temperature reflects the difference between actual internal temperature and set point temperature, then there is no substantiation for the concept of a shift in set point temperature associated with exercise in the results of these experiments. Furthermore, if we assume that selected glove temperatures are correlated with total load error in thermoregulation, then the present data would also indicate that there is no evidence for a nonthermal input during exercise which would effect the thermoregulatory set point. A mathematical model of thermoregulation in man, described by Stolwijk (1971), can be used to simulate the effects of several levels of exercise at various ambient temperatures. Without having to assume a nonthermal input and with a constant set point, this model will equilibrate at internal temperatures related to the level of exercise and independent of the ambient temperature, yielding a response similar to that observed in Nielsen's studies (Nielsen, 1966; Nielsen & Nielsen, 1962; Nielsen, 1938). Thus, the relationship between equilibrated core temperature and the level of exercise appears to be asso-
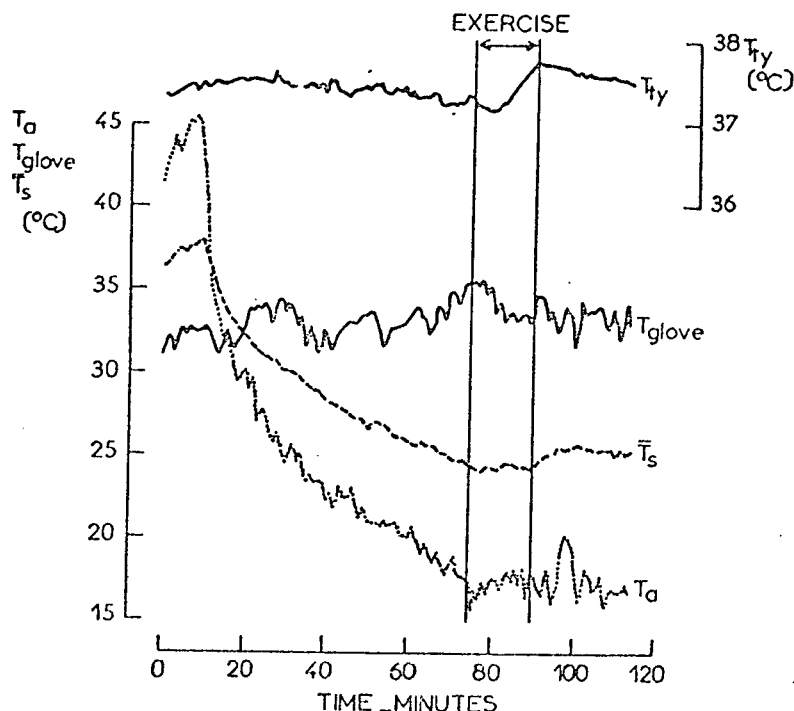
FIG. 7. Narrow range of glove temperatures selected by a subject during induced changes in internal and mean skin temperatures. (Between the time interval of 50–70 min. the subject was instructed to experience the full range of water temperatures available to her.)

ciated with the characteristics of the passive system more than with the active thermoregulatory component.

The conclusion that the set point is not shifted during exercise is supported by several other observations. It has been shown in dogs immersed in a water bath with both hypothalamic and skin temperature controlled that the threshold for panting is unchanged by the transition from rest to vigorous swimming (Chatonnet, Cabanac, & Jeddi, 1965). If the elevated internal temperature established during exercise results from a resetting of the hypothalamic set point, one would expect salicylates to show some effect. However, no effect of salicylates on exercise hyperthermia can be demonstrated (Bass & Jacobson, 1963; Downey & Darling, 1962). Also, the fact that shivering persists during exercise in a cold environment until the internal temperature is returned to normal (Stolwijk et al., 1968) tends to indicate that the set point is not raised during exercise. Furthermore, the increase in peripheral vasoconstriction recorded at the onset of exercise (Robinson,

Meyer, Newton, Ts'ao, & Holgersen, 1965) is not consistent with a lowering of the hypothalamic set point during exercise.

There is sound experimental evidence to support the view that the internal set point may be altered by thermal input from the skin (Cabanac, Chatonnet, & Philipot, 1965; Chatonnet, 1967; Chatonnet, Cabanac, & Mottaz, 1964; Hammel, Jackson, Stolwijk, Hardy, & Strømme, 1963). Webb and Annis (1968) have shown that in the steady state the core temperature for threshold sweating during exercise can be raised by more than 1° C. when the mean skin temperature is lowered by 5°–6° C. Under these conditions it is possible to explain the elevated internal temperature during exercise by the thermal input coming from the cool skin. This effect of peripheral thermal input upon the central set point may explain the apparently contradictory data obtained in man and the dog. A falling skin temperature resulting from either the exposure to a low environmental temperature or from the onset of sweating will tend to raise the internal set point. Thus, during

exercise at a neutral ambient the skin temperature in man is affected by the thermoregulatory vasomotor and sweating responses, and this may give rise to positive or negative feedback from the periphery. Bradbury, Fox, Goldsmith, and Hampton (1964) have proposed that, in the dog, exercise will raise the mean skin temperature since there is no sweating, and in turn this will result in a lowering of the set point. Therefore, it is possible that a resetting of the set point does occur as a result of change in peripheral thermal inputs alone. Experimental evidence for the substantiation of this hypothesis can be found in the results of the present study.

REFERENCES

ASMUSSEN, E., & BØJE, O. Body temperature and capacity for work. *Acta Physiologica Scandinavica*, 1945, 10, 1–22.

BASS, D. E., & JACOBSON, E. D. Effect of salicylate on work in the heat. *Federation Proceedings*, 1963, 22, 176.

BRADBURY, P. A., FOX, R. H., GOLDSMITH, R., & HAMPTON, J. F. G. The effect of exercise on temperature regulation. *Journal of Physiology* (*London*), 1964, 171, 384–396.

CABANAC, M. Plaisir ou deplaisir de la sensation thermique et homeothermie. *Physiology and Behavior*, 1969, 4, 359–364.

CABANAC, M., CHATONNET, J., & PHILIPOT, R. Les conditions de température cérébrale et cutanée moyennes pour l'apparition du frisson thermique chez le chien. *Comptes Rendus Hebdomadaires des Séances de l'Academie des Sciences*, 1965, 260, 680–683.

CHATONNET, J. Sur les modalités de mise en jeu des commandes nerveuses de la régulation thermique. *Les concepts de Claude Bernard sur le milieu intérieur.* Paris: Masson, 1967.

CHATONNET, J., & CABANAC, M. The perception of thermal comfort. *International Journal of Biometeorology*, 1965, 9, 183–193.

CHATONNET, J., CABANAC, M., & JEDDI, E. Le niveau de reglage de la temperature interne est il modifiée par le travail musculaire? *Comptes Rendus de La Société de Biologie*, 1965, 159, 1576–1578.

CHATONNET, J., CABANAC, M., & MOTTAZ, M. Les conditions de température cérébrale et cutanée moyenne pour l'apparition de la polypnée thermique chez le chien. *Comptes Rendus Hebdomadaires des Séances de l'Académie des Sciences*, 1964, 260, 1354–1356.

DOWNEY, J. A., & DARLING, R. C. Effect of salicylates on elevation of body temperature during exercise. *Journal of Applied Physiology*, 1962, 17, 323–325.

GAGGE, A. P., STOLWIJK, J. A. J., & HARDY, J. D.
Comfort and thermal sensations and associated physiological responses at various ambient temperatures. *Environmental Research*, 1967, 1, 1–20.

HAMMEL, H. T. Regulation of internal body temperature. *Annual Review of Physiology*, 1968, 30, 641–710.

HAMMEL, H. T., JACKSON, D. C., STOLWIJK, J. A. J., HARDY, H. D., & STRØMME, S. B. Temperature regulation by hypothalamic proportional control with an adjustable set point. *Journal of Applied Physiology*, 1963, 18, 1146–1157.

JACKSON, D. C., & HAMMEL, H. T. Hypothalamic "set" temperature decreased in exercising dog. *Life Sciences*, 1963, 8, 554–563.

KUNO, Y. *Human perspiration.* Springfield, Ill.: Charles C Thomas, 1956.

MINARD, D. Sweat rate during work and rest at elevated internal temperatures. *Federation Proceedings*, 1963, 22, 177.

MINARD, D., & COPMAN, L. Elevation of body temperature in health. In J. D. Hardy (Ed.), *Temperature—Its measurement and control in science and industry.* New York: Reinholt, 1963.

NIELSEN, B. Regulation of body temperature and heat dissipation at different levels of energy and heat production in man. *Acta Physiologica Scandinavica*, 1966, 68, 215–227.

NIELSEN, B., & NIELSEN, M. Body temperature during work at different environmental temperatures. *Acta Physiologica Scandinavica*, 1962, 56, 120–129.

NIELSEN, M. Die Regulation der Körpertemperatur bei Muskelarbeit. *Skandinavische Archiv für Physiologie*, 1938, 79, 193–230.

ROBINSON, S. Temperature regulation in exercise. *Pediatrics*, 1963, 32, 691–702.

ROBINSON, S., MEYER, F. R., NEWTON, J. L., TS'AO, C. H., & HOLGERSEN, L. O. Relation between sweating cutaneous blood flow and body temperature in work. *Journal of Applied Physiology*, 1965, 20, 575–582.

STOLWIJK, J. A. J. Integration of central and peripheral signals and thermoregulation. In J. D. Hardy, A. P. Gagge, & J. A. J. Stolwijk (Eds.), *Physiological and behavioral temperature regulation.* Springfield, Ill.: Charles C Thomas, in press.

STOLWIJK, J. A. J., SALTIN, B., & GAGGE, A. P. Physiological factors associated with sweating during exercise. *Aerospace Medicine*, 1968, 39, 1101–1105.

VAN BEAUMONT, W., & BULLARD, R. W. Sweating: Its rapid response to muscular work. *Science*, 1963, 141, 643–646.

VAN BEAUMONT, W., & BULLARD, R. W. Sweating exercise stimulation during circulatory arrest. *Science*, 1966, 152, 1521–1523.

WEBB, P., & ANNIS, J. F. Cooling required to suppress sweating during work. *Journal of Applied Physiology*, 1968, 25, 489–493.

Importance of skin temperature in the regulation of sweating

ETHAN R. NADEL, ROBERT W. BULLARD, AND J. A. J. STOLWIJK
John B. Pierce Foundation Laboratory and Department of Epidemiology,
Yale University School of Medicine, New Haven, Connecticut 06519

NADEL, ETHAN R., ROBERT W. BULLARD, AND J. A. J. STOL-WIJK. *Importance of skin temperature in the regulation of sweating.* J. Appl. Physiol. 31(1): 80–87. 1971.—The major thermal inputs to the thermoregulatory center, internal and skin temperatures, were independently varied to evaluate their interaction and relative contributions toward the regulation of sweating in resting man. Average skin temperature ($\bar{T}_s$) was modified by rapid application and removal of radiant heat. Sweating rate from the thigh, measured by resistance hygrometry, was found to be directly related to the $\bar{T}_s$ during skin heating; during skin cooling, however, local sweating rate was a function of the rate of change of $\bar{T}_s$ as well. Increased internal temperatures were achieved by interposing a brief bout of heavy exercise between heating intervals. The relationship between internal and mean skin temperatures in the control of sweating rate was described by a summation model, where sweating rate was linearly related to internal (esophageal) temperature and the level of $\bar{T}_s$ shifted this relationship in the appropriate direction. Clamping local skin temperature at different levels during skin heating revealed the local skin temperature as exerting a modifying effect on the output from the central controller, acting as a multiplier in the determination of local sweating rate. The following model described the major influences on local sweating rate:

local sweating rate

$$= [\alpha(T_{es} - T_{es0}) + \beta(\bar{T}_s - \bar{T}_{s0} + \gamma[d\bar{T}_s/dt - r_0])]e^{(T_{s\,l} - \bar{T}_{s0})/\delta}$$

local sweating rate; temperature regulation; sweating rate; internal temperature

VARIOUS MODELS describing the regulation of sweat secretion have been proposed (9, 14–16, 29, 30, 33), with most of these predicting the regulatory output from an interaction between internal (hypothalamic) and skin temperatures. Recently, the input from the skin has been investigated more thoroughly (7, 22, 23, 30, 33); however, varied and sometimes conflicting interpretations of experimental data have appeared.

The nature of the interaction between internal temperature and the average temperature of the skin in the determination of sweating rate in resting man has been difficult to assess because of the difficulty of forcing internal temperature beyond a very narrow range. Consequently, both additive (14, 29, 31) and multiplicative (16, 33) models have been derived to define the interaction between these primary thermal inputs.

Bullard et al. (9, 10, 21) have established the importance of the local skin temperature in the determination of local sweating rate and have suggested that its effect was related to the internal temperature drive via some multiplicative mechanism. They theorized that the local effect occurred outside the central integrator, probably in the vicinity of the sweat glands themselves as a result of a temperature dependence of release of transmitter substance per neural impulse at the neuroglandular junction (10). If the local skin temperature effect is mediated peripherally, its influence may modify the output from the thermoregulatory center under certain circumstances. This local influence, heretofore unrecognized, may offer a partial explanation for the variability of the models describing the regulation of the heat dissipation response.

Several recent studies (2, 32) have established a basis for the inclusion of a rate of change of skin temperature component in the model, suggesting a connection with the results obtained by Hensel and co-workers (17, 18) with peripheral nerve fibers which were responsive to both the level and the rate of change of local skin temperature.

To evaluate the relative contributions of these factors which have been implicated and their interaction in the regulation of sweating, it became necessary to cause independent variations of average skin temperature, internal temperature, local skin temperature, and rate of change of skin temperature. To avoid the complications inherent in evaporative rate measurements during transients (5), rate of sweat secretion was measured as the variable under physiologic control within the regulatory mechanism. The synchronous cyclical discharge of sweat over the body (1) strongly suggests a single controlling center, with different skin areas requiring different levels of threshold stimulation from this controlling center (19). Thus, by measuring local sweating rate as the response to the partitioning of these various thermal inputs, the efferent outflow from the thermoregulatory center could be effectively characterized and a comprehensive model constructed.

METHODS

Six male subjects, described in Table 1, were exposed to fixed ambients between 25 and 35° C for 1.5–2 hr. Relative humidity was constant at 35% and air movement was minimal. Each subject, minimally clothed, lay supine on a 5-cm fish netting suspended from a rectangular aluminum frame (2.1 x 0.6 m) which was supported on a Potter beam

balance. A continuous record of body weight provided total evaporative loss per minute by sensing averaged 1-min weights on an IBM 1131 on-line computer. The balance had a 1-g sensitivity.

Rapid alterations in skin temperature were superimposed on the relatively steady state by manipulating a polished aluminum shutter which shielded two Chromalox heating lamps situated 1.6 m above the subject. The energy output from these lamps could be readily controlled by a voltage transformer. Irradiance from the lamps was determined by wide-angle radiometer, which had been calibrated by a Bureau of Standards radiation lamp. Gagge and Hardy (13) had previously reported the irradiated body area in a similar design to be about 32 %. Total irradiance most often used in the present study was 750 W/m^2 of projected area, with irradiance to the subject (effective radiant flux) amounting to 215 W/m^2 of skin surface area. Irradiance was applied stepwise or exponentially and removed in stepwise, exponential, or linear manners. Varying the mode of application and removal provided wide variations in rate of change of skin temperature during skin warming and cooling. Irradiance was continually measured during each exposure by means of a wide-angle radiometer positioned at the level of the subject.

Temperatures from 10 skin surface locations were obtained once per minute from thermocouple recordings and average skin temperature (T_s) was calculated from the following modification of the Hardy-DuBois equation:

$$T_s = .07 \, T_1 + .07 \, T_2 + .09 \, T_3 + .09 \, T_4 + .09 \, T_5 \\ + .09 \, T_6 + .07 \, T_7 + .11 \, T_8 + .16 \, T_9 + .16 \, T_{10}$$

where

T_1 = forehead temperature
T_2 = dorsal bicep temperature
T_3 = right scapular temperature
T_4 = left scapular temperature
T_5 = lateral lumbar temperature
T_6 = chest temperature
T_7 = lateral forearm temperature
T_8 = palm temperature
T_9 = ventral thigh temperature
T_{10} = dorsal calf temperature

Care was taken to measure T_1, T_6, and T_9 from skin surfaces exposed to irradiation and all other skin temperatures from nonirradiated surfaces. The percentage of irradiated body surface (13) was equal to the sum of the weighting factors for T_1, T_6, and T_9, thereby providing a continuously accurate calculation of mean skin temperatures during application and removal of radiant heat. Observations of irradiated skin temperature by thermocouple were in close agreement with those from a radiometer, after correcting for reflectance of the infrared radiation by the skin.

Tympanic membrane temperature (T_{ty}) was also recorded each minute. The tympanic probe consisted of tightly coiled leads which were held firmly in place by an ear plug incorporated onto the wire; this design permitted unrestricted movement on the part of the subject with consistent placement on the tympanum and minimal discomfort. Esophageal temperature (T_{es}) was continuously recorded from a thermocouple positioned at the level of

TABLE 1. *Physical characteristics of subjects*

Subj	Age, yr	Ht, cm	Wt, kg	DuBois Body Surface Area, m^2
DS	23	176	70.6	1.87
RL	21	188	77.1	2.05
CJ	26	170	56.7	1.67
DB	24	181	74.1	1.93
RP	26	188	95.1	2.19
PO	24	178	77.0	1.95

the heart in many, but not all, experiments. T_{es} was used to represent internal temperature when available, since its response during the transient of exercise was observed to be faster than that of T_{ty}, suggesting a closer approximation to blood temperature; otherwise, T_{ty} was used as the representation of internal temperature.

Local sweating rate was recorded from a 12-cm^2 skin area on each ventral thigh surface by parallel resistance hygrometry circuits (7, 10). Air of known humidity was drawn at 1.5 L/min through a sweat collection capsule on the thigh and then past a hygrosensitive element housed in a constant-temperature chamber (49° C) 1 m downstream from the capsule. Any water appearing on the skin surface underneath the capsule would be immediately evaporated into the airstream; thus, a change in the humidity of the airstream was resultant from sweating underneath the capsule and was detected as a resistance change in the hygrosensitive element. Each element was calibrated directly by pumping distilled water at known rates into the sealed capsules from a Harvard constant-infusion pump with a synchronous motor and variable speed control. Calibrations were checked periodically. Time lag of the system due to transport delay was 3 s.

At the beginning of each experiment, the subject was placed on the netting and, after all instrumentation was attached, initial base lines were established for all variables. After this period of lying quietly in the near neutral ambient, the subject was exposed to a preconditioning period of intense irradiation that lasted 4–10 min until obvious sweating activity was established. Gagge and Hardy (13) had reported that under the chosen experimental conditions a preconditioning, or "priming," period was necessary to elevate internal body temperature and stimulate sweat gland activity. Bickford (4) and Bullard (7) have attributed the lag in sweat secretion in an initial electrical stimulation of a population of sweat glands to the time required for filling the sweat ducts and hydrating the epidermis. After the preconditioning period, the subject was exposed to intense irradiation for 2–4 min, followed by 3- to 6-min recovery intervals between irradiations.

The relationships between T_s, internal T, and local sweating rate were studied in five of the subjects in a number of experiments. After a minimum of six resting irradiation intervals, the subject mounted a bicycle ergometer adjacent to the bed and worked for 10–15 min at 60–80 % $\dot{V}_{O_2 \, max}$ to elevate his internal temperature. He then immediately resumed the supine position and was again exposed to intermittent irradiation. With this technique, rapid alterations in skin temperature could be achieved in

the presence of a wide range of internal temperatures in resting man.

The influence of local skin temperature (T_{sl}) on local sweating rate at different levels of $\bar{T}_s$ was evaluated in three of the subjects. A two-chambered copper sweat collection capsule was placed on one thigh surface and the skin temperature underneath that capsule, continuously monitored by thermocouple, was clamped at different levels by perfusing the copper chamber of the copper capsule with warm or cold water. These subjects were then exposed to on-off irradiation as previously described and local sweating rate from the thermally clamped thigh recorded. Local sweating rate from the contralateral thigh, shielded from the radiant energy, was simultaneously obtained as a representation of control activity.

RESULTS

Sudden exposure to intense radiant heat caused an immediate increase in the temperature of irradiated skin. Nonirradiated skin areas did not show any consistent temperature alterations during the same brief intervals. T_{sl} under the capsule also remained relatively constant ($\pm 0.1^\circ$ C). The typical pattern of average skin temperature change during three consecutive heating-cooling cycles appears in Fig. 1. The rate of change of skin temperature, always greatest at the onset of heating or cooling, approached rates of 12° C/min for ventral surface temperature and 4° C/min for average skin temperature within the initial 10 s, and with time during both heating and cooling. Chest skin temperatures as high as 41.2° C were recorded, but $\bar{T}_s$ increases rarely exceeded 1.0° C during a 2-min irradiation and 1.4° C during 4 min of irradiation.

Also represented in Fig. 1 are the typical local sweating responses to rapid alterations in $\bar{T}_s$. In the "primed" subject, the onset of increased local sweating (and increased whole-body evaporative losses) occurred in conjunction with elevated $\bar{T}_s$, and rapid decreases in local sweating rates were associated with the cooling cycle. Energy dissipation calculated from both local evaporative losses and whole-body evaporation approximated the radiant energy added during the brief intervals. During rapid cycling of
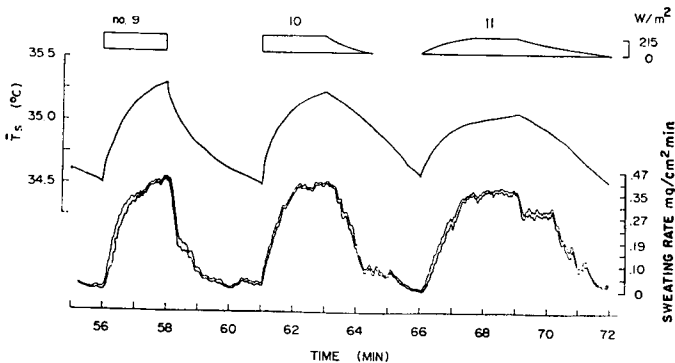

FIG. 1. Mean skin temperature and local sweating rate responses from 12 cm^2 of both ventral thigh surfaces during three consecutive exposures to 215 W/m^2 irradiation. Rate of cooling in the three exposures was progressively decreased by varying the mode of removal of irradiation. $T_a = 27^\circ$ C, $T_{es} = 36.95^\circ$ C.
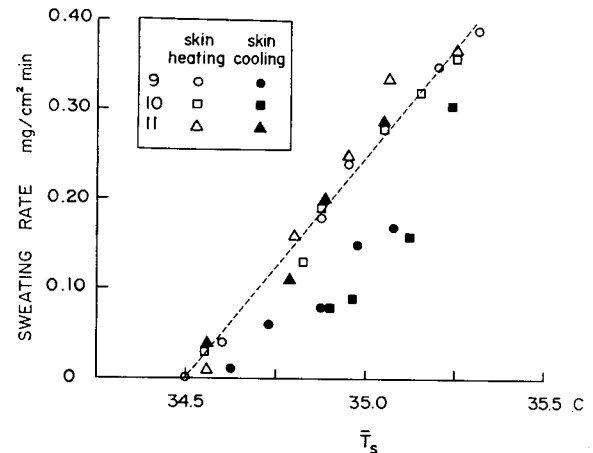

FIG. 2. Linear relationship between local sweating rate and mean skin cooling at low rate (filled triangles). Other filled symbols illustrate depression of sweating during increased rates of skin cooling. Data points taken at 20-s intervals from the three curves in Fig. 1.

$\bar{T}_s$, internal temperatures (T_{ty} and T_{es}) remained essentially constant.

The initial intervals during which local sweating rate did not follow increased $\bar{T}_s$ at the onset of irradiation were not utilized in the analysis, since these most likely represented conditions where the internal temperature-$\bar{T}_s$ relationship at the onset of skin heating was sufficiently below the combined threshold for sweating and/or the sweat ducts or epidermal layers were depleted of fluid and a finite time lag was necessary for their hydration. Once the onset of sweating coincided with the initiation of skin heating, this coincident relationship was maintained in subsequent intervals.

During skin heating, local sweating rate was directly related to the instantaneous value of $\bar{T}_s$ over wide ranges of $\bar{T}_s$ and sweating rate (Fig. 2), and independent of the rate of increase of skin temperature. During skin cooling, however, the proportional decline in sweating rate was greater than the decline in T_s in nearly all cases, as is also illustrated in Fig. 2. There was a good relation between the rate of decrease of $\bar{T}_s$ and the reduction in sweating rate from the steady-state $\bar{T}_s$-sweating rate regression line at lower and moderate levels of sweating, but this relation tended to fail during skin cooling in the presence of elevated internal temperatures where the sweating rate decrease became more linearly related to or even lagged the fall in $\bar{T}_s$. Elevated internal temperatures ($T_{ty} > 37.5^\circ$ C) during rest were usually the result of repeated heating intervals in ambients above thermoneutrality ($28-30^\circ$ C), where dissipation of heat by convection was reduced and $\bar{T}_s$ remained higher. In these circumstances, the sweating drive from internal temperature signals became increasingly important and, therefore, the drive from the skin relatively less important; hence, the drive from $d\bar{T}_s/dt$ information was likewise diminished in importance.

Deviations from the linear sweating rate-$\bar{T}_s$ graphs for each subject were tabulated at 15- to 30-s intervals from at least 2 separate days and plotted against $d\bar{T}_s/dt$. Figure 3 illustrates the absence of any effect of $+d\bar{T}_s/dt$ on local sweating rate and the inhibitory effect of $-d\bar{T}_s/dt$. The

instantaneous sweating rate-$\bar{T}_s$ points in Fig. 3 were gathered at 15-s intervals on 3 separate experimental days from a number of heating-cooling cycles. Ambients on these days were 25°, 28°, and 32° C. Without exception, when $-d\bar{T}_s/dt$ was greater than 0.2° C/min, local sweating rate was reduced in comparison to sweating during the steady state, when $d\bar{T}_s/dt = 0$. During $+d\bar{T}_s/dt$, local sweating rate was not affected by the rate component over a wide range in any subject. The slope of this relationship for subject DS (Fig. 3) was found to be -0.40 mg/cm²·min per $-1.0°$ C per min of $\bar{T}_s$ change, when the rate of change exceeded $-0.1°$ C/min. The similar relationship for the other subjects appears in Table 2.

After exercise, subjects maintained elevated levels of internal temperature and local sweating rate. In the presence of elevated T_{es}, local sweating rate was again linearly related to $\bar{T}_s$ during skin heating, but the slope of the sweating rate-$\bar{T}_s$ relationship was reduced. Replotting these data along T_{es} isotherms revealed that local sweating rate
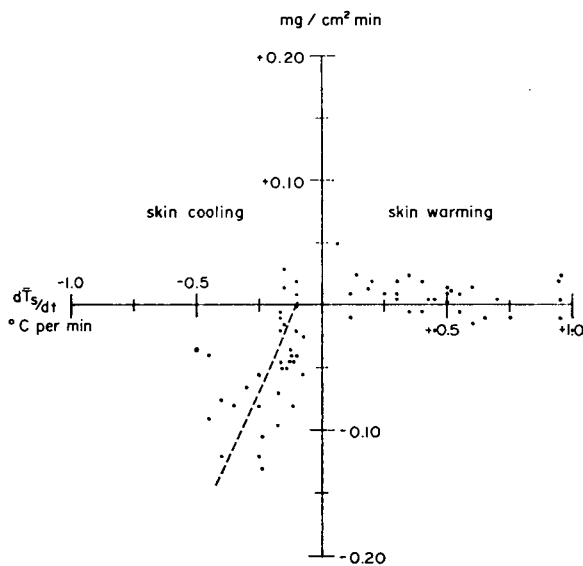

mg / cm² min

FIG. 3. Illustration of sensitivity of rate of sweating to rate of change of $\bar{T}_s(d\bar{T}_s/dt)$ in *subject DS*. Points represent the deviation from the linear sweating rate-$\bar{T}_s$ relationships at 15- to 30-s intervals from a number of irradiation cycles in ambients of 25°, 28°, and 32° C. Characteristics of rate sensitivity for all subjects appears in Table 2.

TABLE 2. *Average deviation from linear sweating response of six resting* $(T_{ty} < 37.5°\ C)$ *subjects during skin cooling*

Subj	Rate Control Constant (γ), mg/cm²·min per °C per min	Rate Threshold (r_o), °C per min	Resting $\bar{T}_s$ Below Which No Sweating Occurs ($\bar{T}_{s_0}$), °C
DS	−0.40	−0.10	35.5
RL	−0.50	−0.10	34.0
CJ	−0.32	−0.19	34.3
DB	−0.22	−0.18	33.7
RP	−0.18	−0.35	34.3
PO	−0.16	−0.22	34.5

Instantaneous $\bar{T}_s$-sweating rate points obtained at 15- to 30-s intervals over 2-3 min from no fewer than 10 cooling cycles in at least two different ambients.
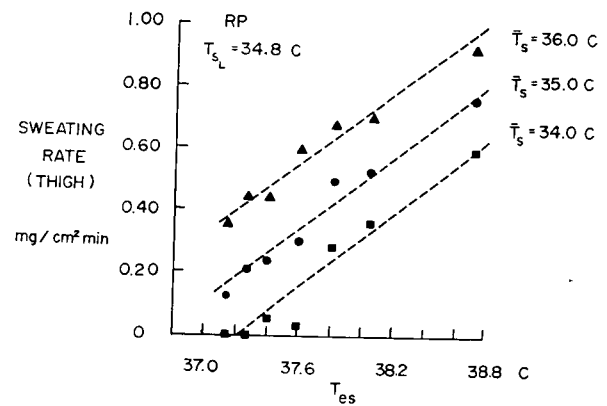

FIG. 4. Effect of T_{es} and $\bar{T}_s$ on local sweating rate with local (under sweat collection capsule) skin temperature = 34.8° C.

was directly related to T_{es}, with the effect of $\bar{T}_s$ shifting this relationship in the appropriate direction (Fig. 4).

Clamping T_{s_l} at different levels during skin heating altered the slope of the linear sweating rate-$\bar{T}_s$ relationship, but not the T_{s_l} threshold at constant internal temperatures (Fig. 5). This verified that the primary drive affecting the rate of local sweat secretion during skin heating in the presence of constant $T_{internal}$ was provided by $\bar{T}_s$, with T_{s_l} modifying this drive. Replotting these data revealed a nonlinear relationship between local sweating rate and T_{s_l} at any level of $\bar{T}_s$, verifying previous observations of Bullard et al. (9, 10). There was interindividual consistency in the local sweating rate-T_{s_l} relationship, since the data from all subjects could be described by a single exponential function.

DISCUSSION

Once the sweating mechanism had been sufficiently primed in the presence of a constant internal thermal drive, sweating rate was related either directly to the average temperature of the skin or to a derivative thereof. By effectively separating the peripheral and central thermal inputs to the thermoregulatory center, the modifications in rate of sweat secretion caused by irradiation were clearly mediated entirely through peripheral thermoreception. The relationship between sweating rate and $\bar{T}_s$ confirmed Gagge's (12) report of increased evaporative losses in subjects exposed to high radiant heat, and supported Gagge and Hardy's (13) observation that under conditions where heat storage was negligible, the change in evaporative loss caused by thermoregulatory sweating was quantitatively related to the effective radiant flux. These observations also verified Kerslake's (20) report of a 1.6-s physiological delay between cyclical irradiation and sweat production. The present data are added evidence in contradiction of Benzinger's (3) conclusions regarding the noninvolvement of peripheral thermoreceptors at skin temperatures greater than 33° C in the control of sweating.

By independently varying $\bar{T}_s$ and T_{es}, local sweating rate was shown to be linearly related to $(\bar{T}_s - \bar{T}_{s_0})$ when T_{es} and T_{s_l} were constant (only during skin heating) and linearly related to $(T_{es} - T_{es_0})$ when $\bar{T}_s$ and T_{s_l} were constant (Fig. 4). The interaction between T_{es} and $\bar{T}_s$ represented a
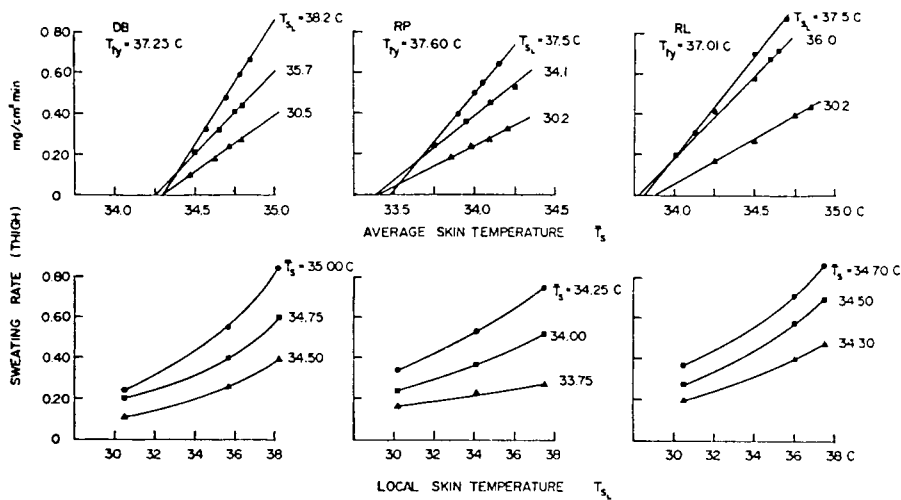
FIG. 5. Influence of local skin temperature (T_{s_l}) on linear sweating rate-T_s relationships during skin heating in three subjects (*upper graphs*). Replotting these data illustrates exponential influence of T_{s_l} on local sweating rate at any level of T_s.

summation of temperature signals in the determination of efferent activity. A summation model for evaporative heat loss has been proposed by Stolwijk and Hardy (29) for rest and by Stolwijk et al. (31) for exercise. However, they noted that other relationships such as multiplicative ones have been suggested and that internal and average skin temperatures may not be the only factors influencing evaporative losses in man. This summative relationship between T_{es} and $\bar{T}_s$ in the determination of sweating rate when T_{s_l} variations were clamped at a constant level was observed in all five subjects studied, despite individual differences in the proportional control constants α and β. The mathematical expression of this interrelation between these temperatures was as follows:

$$\text{local sweating rate} = (\alpha[T_{es} - T_{es_0}] + \beta[\bar{T}_s - \bar{T}_{s_0}]) \quad (1)$$

where T_{es_0} and $\bar{T}_{s_0}$ represent threshold temperatures for sweating in the steady state of rest.

The effect of varying the average skin temperature by skin heating in the presence of different, fixed local skin temperatures (with internal temperature constant) is shown in the upper graphs of Fig. 5. Rather large modifications in local sweating rate were observed in the presence of a constant sweating drive from internal and average skin temperature signals in response to moderate alterations in local skin temperature. This observation offered a partial explanation for the diversity in the models describing the regulation of sweating, because it was apparent that the model contained both additive and multiplicative elements, as follows:

$$\text{local sweating rate} = (\alpha[T_{es} - T_{es_0}] + \beta[\bar{T}_s - \bar{T}_{s_0}])L \quad (2)$$

where L represents the local skin temperature effect. The inclusion of both additive and multiplicative elements in this model tends to unify the viewpoints of Hammel (14), Stolwijk and Hardy (29), and Stolwijk et al. (31), whose descriptions of the heat loss response can be readily represented as a summation between the primary thermal inputs, with the observations of Bullard et al. (9, 10), Hardy and Stolwijk (16), and Wyndham and Atkins (33) that there is a multiplicative effect of these thermal inputs. Stitt et al. (28) have described the control of sweating in the squirrel monkey as a summation between thermal inputs from the

hypothalamus and the skin, and Nadel and Stitt (24) have reported that the sweating rate from the foot of squirrel monkeys was related to local temperature in the presence of constant internal and average skin temperatures; thus, the elements controlling sweating rate in man have been reported as participating in a like manner in another species as well.

The nature of the local skin temperature effect on local sweating rate has been described by Bullard et al. (9) as a nonlinear relationship, with increasing increments in local sweating rate per unit of local temperature increase. The present data describing the T_{s_l}-sweating rate relation (Fig. 5, lower graphs) were relatively consistent with Bullard's data, although the Q_{10} of sweat gland activity in the present study was slightly lower. The average sweating rate Q_{10} when internal temperature was constant for the three subjects in the present study was 3.0 (subject range 2.6–3.5), with each subject showing consistency over a wide range of $\bar{T}_s$. Calculations from Bullard's curves (one subject) show an average Q_{10} between 4 and 5 for different levels of $\bar{T}_s$. This discrepancy may be attributed to *1*) the differential placement of the sweat collection capsule, encompassing a population of sweat glands innervated by a considerably different concentration of nerve branchlets, or *2*) the fact that the calculation of the Q_{10} from the derivative of local sweating rate with respect to local skin temperature is very sensitive to minor alterations in average skin temperature. By deriving the local sweating rate-T_{s_l} functions from the linear sweating rate-$\bar{T}_s$ relations at given levels of T_{s_l}, the small variations in $\bar{T}_s$ which would occur over the time course of an experiment and the effects of these variations have been minimized. Calculations from the whole-body data of Wyndham and Atkins (33) using our model revealed a Q_{10} effect of 2.7, a surprisingly close fit with the calculated Q_{10} from this study considering the differences between local sweating and total evaporative measurements.

The local temperature effect (L in *eq 2*) was determined to be an exponential, with the local temperature error signal in the numerator of the exponent and the Q_{10} constant, δ, in the denominator. The calculation from the three subjects revealed $\delta = 9.1$ when $L = e^{(T_{s_l} - \bar{T}_{s_0})/\delta}$. The consistency in the sweating rate-T_{s_l} data between subjects

permitted derivation of δ from the pooled data. The similarity between subjects supported the concept that local temperature has its effect on sweating rate at the neuroglandular junction, where physicochemical laws tend to govern the reaction rate, rather than within the central controller, which shows significant interindividual variability. The mode of action at the neuroglandular junction has yet to be determined, as Ogawa (27) has suggested that high local skin temperature may act on specific receptor mechanisms of glandular cells to increase their sensitivity to specific stimuli, rather than by simply accelerating cellular metabolism.

The observation of a rate of change of $\bar{T}_s$ effect on local sweating rate during skin cooling supported earlier reports of such an effect (2, 32). There was no effect of rate of change of $\bar{T}_s$ on rate of sweat secretion during skin warming. This observation indicated that the thermoregulatory center processed peripheral thermal information somewhat differently during skin heating and skin cooling. The lack of correlation between alterations in skin temperature and weight loss observed by Colin and Houdas (11) during a heating transient in five of eight subjects could be attributed to the time lag associated with the priming phenomenon rather than noninvolvement of skin thermoreceptors.

Although the firing rate of warm and cold sensitive afferents have been shown to be markedly elevated during rate of change of skin temperature (17, 18), Banerjee et al. (2) noted that the temporal characteristics of the neural patterns during the transient have not been shown to correspond with the time course of the inhibition of sweating. Local T_s transients induced by Banerjee et al. (2) were 30 s–2 min, whole $\bar{T}_s$ transients utilized by Wurster and McCook (32) lasted 10–15 min. $\bar{T}_s$ transients in the present study were 2–4 min. Alterations in firing rate of cutaneous thermosensitive afferent fibers during thermal transients were only reported for 2- to 4-s intervals by Hensel and co-workers (17, 18). Firing rate during extended thermal transients, such as used in the above studies, has not been reported. It is likely that the various thermosensitive fibers in any area of skin display a wide diversity in their activity during transient thermal situations. The similarity between firing rate of cold sensors as reported by Hensel and co-workers (17, 18) and pattern of sweating rate during cooling transients may only indicate that there are dynamic overshoots in physiological systems at several levels. The absence of similarity between these parameters on the warm side suggested that the control over rate of local sweat secretion was not exclusively dependent on firing rate of peripheral thermoreceptors during transients. Rather, a combination of input from the thermosensitive afferent fibers, integration in the thermoregulatory center, and mechanical limitations between the motor activity and actual liberation of sweat were responsible for the sweating response during transients and the differences in responsiveness between heating and cooling transients. Further insight into the control of sweating during thermal transients might be attained with a nerve-gland preparation.

If the $d\bar{T}_s/dt$ effect on local sweating rate were mediated centrally, the rapidly changing skin temperature could either be acting to a) reduce the gain of the controlling system, thereby assigning the error signal reduced im-portance, b) stimulate an upward shift in hypothalamic threshold temperature for sweating, thereby reducing the error signal which drives sweating activity, or c) provide a signal to the integrator in conjunction with the $\bar{T}_s$ error signal. Since the entire sweating variability in the present set of conditions at constant local skin temperature was directly accounted for by the change in $\bar{T}_s$ and its negative rate derivative, the latter would be the most likely explanation. The observation that the rate effect on sweating rate was diminished when the internal temperature was elevated above $37.5°$ C would support this interpretation, since the total input from the skin was relatively less important in the determination of sweating rate in the presence of an elevated internal temperature.

Other evidence that the $d\bar{T}_s/dt$ effect on sweating rate was of central rather than local origin was that skin temperature under the shielded sweat capsule was relatively constant during the alterations in $\bar{T}_s$. If the rate effect were of local origin, it could not have been observed under such circumstances. Brown and Brengelmann (6) have demonstrated an overshoot in metabolic response during rapid skin cooling, further indicating that the rate effect was through centrally mediated rather than local activities.

Thus, the total influence of the average skin temperature error signal within the central integrator was expanded to include the rate of change information, as follows:

$$\beta(T_s - T_{s_0} + \gamma[dT_s/dt - r_0]) \qquad (3)$$

where the average values of γ (the rate constant), $\bar{T}_{s_0}$, and r_0 for each subject appear in Table 2. The rate component has its effect only when $d\bar{T}_s/dt$ exceeds its threshold value, r_0, during skin cooling. The inclusion of a rate of change component in the model describes a control system that is highly responsive to sudden decreases in ambient and, therefore, average skin temperatures.

A curious phenomenon occasionally observed was a rebound or sudden increase in sweating following the marked depression during rapid skin cooling. As $d\bar{T}_s/dt$ was reduced during the 2nd or 3rd min of cooling, a burst of activity from the sweat glands elevated sweating rate to levels that would have been predicted directly from $\bar{T}_s$. These rebounds were transient in nature and did not persist for intervals greater than 20–30 s. Rebound sweating probably represented the overriding of the rate component influence on sweating rate by the steady state or error component influence. Because of the transience and irregularity of rebound sweating, quantification of this phenomenon was not attempted.

A synthesis of the information described in *equations 1–3* reveals the major determinants of local sweating rate to be interrelated as follows:

$$\text{local sweating rate} = [\alpha(T_{es} - T_{es_0}) + \beta(\bar{T}_s - \bar{T}_{s_0} + \gamma[d\bar{T}_s/dt - r_0])]e^{(T_{s_l} - \bar{T}_{s_0})/\delta} \qquad (4)$$

Total evaporative heat loss data obtained in this laboratory during steady-state resting (ref 16, Fig. 11, 13; ref 29, Fig. 17; ref 31, Fig. 2) exposures in a wide range of ambients were compared to the model represented by *equation 4*. These data were readily fit to the skeleton equation by utilizing the following constants:

E in W/m^2 = (197(T$_{in}$ — 36.7)

$\qquad$ + 23($\bar{\text{T}}_s$ — 34.0))$e^{(\Sigma\ \text{weighted}\ \text{T}_{sl}\ -\ 34.0)/10}$ $\qquad$ (5)

Figure 6 illustrates the dependence of these steady-state evaporative heat loss data on internal and skin temperatures, with the solid lines drawn from *equation 5*. It is apparent that the different T$_s$ isotherms have different slopes as well as intercepts. The following conclusions from the local sweating information can then be extended to include the control of total body sweating as estimated from weight loss in a steady state during rest and exercise: *1*) at constant skin temperature, sweating is proportional to internal temperature; *2*) at constant internal temperature, sweating is proportional to mean skin temperature; and *3*) at a given combination of internal and mean skin temperatures, local sweating is dependent on local skin temperature with a Q$_{10}$ of slightly less than 3.

It is clear that Fig. 6 has included the local skin temperature multiplier effect from the nonparallel splay of the T$_s$ isotherms. If the relationship between internal and skin temperatures were entirely multiplicative, the T$_{es}$ sweating threshold for all levels of T$_s$ would be constant; obviously, this is not the case.

The output from the central controller toward the determination of whole-body sweat loss can be found by eliminating the peripheral multiplier influence from *equation 5*, and this relation appears in Fig. 7. This interaction has only been validated for the steady state; the relation during transients such as skin cooling, as illustrated previously, or the onset of exercise introduce new inputs into the control mechanism which must be considered.

In conclusion, the partitioning of the major inputs to the

FIG. 7. Output from central controller toward determination of total evaporative heat loss calculated by eliminating local effect. Solid lines calculated from E = 197(T$_{in}$ — 36.7) + 23($\bar{\text{T}}_s$ — 34.0).

thermoregulatory center coupled with continuous estimates of the efferent outflow from this center (measurement of rate of local sweat secretion) has permitted certain insights into the controlling mechanism. Additive as well as multiplicative aspects of the integration of central and peripheral signals in the determination of the sweating response have been identified. The relationship between internal and mean skin temperatures in the control of local sweating rate was found to be a summation while the local skin temperature contribution was identified as a modifying effect upon the output from the central controller, acting as a multiplier in the determination of local sweating rate. The rate of change of skin temperature was also shown to provide a significant signal to the integrator during skin cooling. Moreover, total evaporative data taken from steady-state experiments of this laboratory (16, 29, 31) and others (26, 33) could readily be described by the equation derived from the interrelations between internal and skin temperatures and local sweating rate. There no doubt are other thermal and/or nonthermal inputs that participate in the regulation of sweating, but these may be difficult to partition and may operate only during exceptional circumstances. Considerably more data are required for a complete description of the regulation of sweating, particularly in the area of regulation during transients.
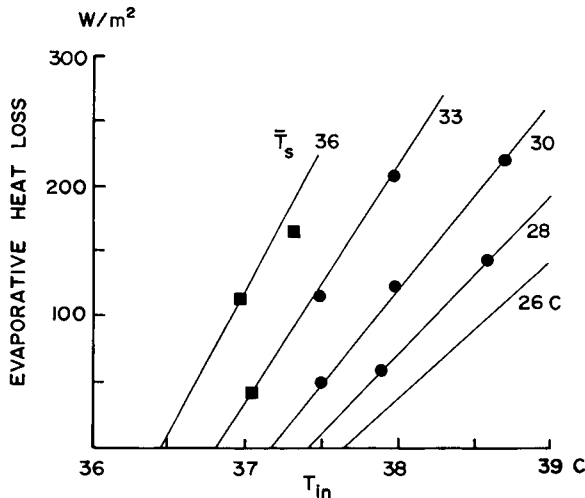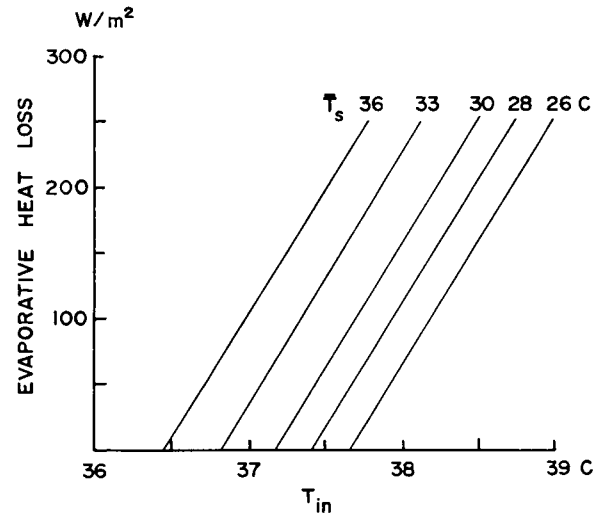
FIG. 6. Dependence of total evaporative heat loss on internal and skin temperatures. Data points obtained from steady-state resting (filled squares) and exercising (filled circles) exposures in a wide range of ambients (16, 29, 31). Solid lines calculated from E = (197 (T$_{in}$ — 36.7) + 23(T$_s$ — 34.0))$e^{(\Sigma\ \text{weighted}\ \text{T}_{sl}\ -34.0)/10}$.

REFERENCES

1. ALBERT, R. E., AND E. D. PALMES. Evaporation rate patterns from small skin areas as measured by an infrared gas analyzer. *J. Appl. Physiol.* 4: 208–214, 1951.
2. BANERJEE, M. R., R. ELIZONDO, AND R. W. BULLARD. Reflex responses of human sweat glands to different rates of skin cooling. *J. Appl. Physiol.* 26: 787–792, 1969.
3. BENZINGER, T. H. On physical heat regulation and the sense of temperature in man. *Proc. Natl. Acad. Sci., US* 45: 645–659, 1959.

4. Bickford, R. G. The mechanism of local sweating in response to Faradism. *Clin. Sci.* 3: 337–341, 1938.

5. Brebner, D. F., and D. McK. Kerslake. The relation between sweat rate and weight loss when sweat is dripping off the body. *J. Physiol., London* 202: 719–735, 1969.

6. Brown, A. C., and G. L. Brengelmann. The interaction of peripheral and central inputs in the temperature regulation system. In: *Physiological and Behavioral Temperature Regulation,* edited by J. D. Hardy. Springfield, Ill., Thomas, 1970, chapt. 47.

7. Bullard, R. W. Continuous recording of sweating rate by resistance hygrometry. *J. Appl. Physiol.* 17: 735–737, 1962.

8. Bullard, R. W. Studies on human sweat gland duct filling and skin hydration. *J. Physiol., Paris.* In press.

9. Bullard, R. W., M. R. Banerjee, F. Chen, R. Elizondo, and B. A. MacIntyre. Skin temperature and thermoregulatory sweating: a control systems approach. In: *Physiological and Behavioral Temperature Regulation,* edited by J. D. Hardy. Springfield, Ill., Thomas, 1970, chapt. 40.

10. Bullard, R. W., M. R. Banerjee, and B. A. MacIntyre. The role of the skin in negative feedback regulation of eccrine sweating. *Intern. J. Biometeorol.* 11: 93–104, 1967.

11. Colin, J., and Y. Houdas. Initiation in sweating in man after abrupt rise in environmental temperature. *J. Appl. Physiol.* 20: 984–990, 1965.

12. Gagge, A. P. Body perspiration on exposure to thermal radiation (Abstract). *Federation Proc.* 23: 473, 1964.

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

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

15. Hardy, J. D. Physiology of temperature regulation. *Physiol. Rev.* 41: 521–606, 1961.

16. Hardy, J. D., and J. A. J. Stolwijk. Partitional calorimetric studies of man during exposures to thermal transients. *J. Appl. Physiol.* 21: 1799–1806, 1966.

17. Hensel, H. Electrophysiology of thermosensitive nerve endings. In: *Temperature, Its Measurement and Control in Science and Industry,* edited by J. D. Hardy. New York: Reinhold, 1963, vol. 3, part 3, p. 191–198.

18. Hensel, H., A. Iggo, and J. Witt. A quantitative study of sensitive cutaneous thermoreceptors with C afferent fibers. *J. Physiol., London* 153: 113–126, 1960.

19. Hertzman, A. B., W. C. Randall, C. N. Peiss, and R. Seckendorf. Regional rates of evaporation from the skin at various environmental temperatures. *J. Appl. Physiol.* 5: 153–161, 1953.

20. Kerslake, D. McK. Thermal radiation in the investigation of cutaneous vasomotor and sudomotor control. In: *Thermal Problems in Aerospace Medicine,* edited by J. D. Hardy. Maidenhead, England: Technivision Services, 1968, p. 57–70.

21. MacIntyre, B. A., R. W. Bullard, M. Banerjee, and R. Elizondo. Mechanism of enhancement of eccrine sweating by localized heating. *J. Appl. Physiol.* 25: 255–260, 1968.

22. Nadel, E. R., and S. M. Horvath. Peripheral involvement in the thermoregulatory response to an imposed heat debt in man. *J. Appl. Physiol.* 27: 484–488, 1969.

23. Nadel, E. R., S. M. Horvath, C. A. Dawson, and A. Tucker. Sensitivity to central and peripheral thermal stimulation in man. *J. Appl. Physiol.* 29: 603–609, 1970.

24. Nadel, E. R., and J. T. Stitt. Control of sweating in the squirrel monkey (Abstract). *Physiologist* 13: 267, 1970.

25. Nadel, E. R., and J. A. J. Stolwijk. Physiologic control of rate of local sweat secretion in man. *J. Physiol., Paris.* In press.

26. Nielsen, B., and M. Nielsen. Influence of passive and active heating on the temperature regulation of man. *Acta Physiol. Scand.* 64: 323–331, 1965.

27. Ogawa, T. Local effect of skin temperature on threshold concentration of sudorific agents. *J. Appl. Physiol.* 28: 18–22, 1970.

28. Stitt, J. T., E. R. Adair, E. R. Nadel, and J. A. J. Stolwijk. The relation between behavior and physiology in the thermoregulatory response of the squirrel monkey. *J. Physiol., Paris.* In press.

29. Stolwijk, J. A. J., and J. D. Hardy. Partitional calorimetric studies of responses of man to thermal transients. *J. Appl. Physiol.* 21: 967–977, 1966.

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

31. Stolwijk, J. A. J., B. Saltin, and A. P. Gagge. Physiological factors associated with sweating during exercise. *Aerospace Med.* 39: 1101–1105, 1968.

32. Wurster, R. D., and R. D. McCook. Influence of rate of change in skin temperature on sweating. *J. Appl. Physiol.* 27: 237–240, 1969.

33. Wyndham, C. H., and A. R. Atkins. A physiological scheme and mathematical model of temperature regulation in man. *Arch. Ges. Physiol.* 303: 14–30, 1968.

Thermal Loads in Lunar Ambulation

Jan A. J. Stolwijk

John B. Pierce Foundation, Yale University, New Haven, Connecticut 06519

Stolwijk, J. A. J. *Thermal loads in lunar ambulation.* Aerospace Med. 41(11):1266-1268. 1970.

Extravehicular activity on the lunar surface takes place in a severe thermal environment which can range from very high to very low operative temperatures. The Apollo EVA suit system incorporates extremely effective insulation. The major thermal stress is thus due to internally produced metabolic heat. Average metabolic heat production during the Apollo XI extravehicular activity was estimated at about 400 watts, based on indirect measurements. The use of conductive skin cooling with inhibition of sweat production causes some limitation in the ability of the liquid-cooled garment to eliminate metabolically produced heat under conditions of thermal comfort. In contrast to undersea operations where the major thermal hazard is hypothermia, the lunar environment with the use of current technology represents a hazard of hyperthermia.

MAN IS EXPOSED to variations in environmental temperatures and changes in external heat transfer characteristics which change the effective thermal environment. In the face of such variations, as well as wide variations in rates of internal heat production, man's internal body temperature is kept within a very narrow range by the combined action of two separate defense mechanisms. The first of these, and the most powerful, is the behavioral response. In addition to this voluntary response, there are involuntary physiological responses by mechanisms generally recognized as making up the system responsible for body temperature regulation. These physiological responses consist of changes in skin blood flow, sweating or shivering. The behavioral responses are quite varied and can consist of avoidance or escape, limiting the time of exposure, use of insulating clothing or the use of power to create a micro-environment which is more acceptable.

Man experiences as comfortable a thermal environment in which his involuntary thermophysiological responses are minimal. In general, any extreme vasomotor response, or sweating or shivering is experienced as uncomfortable and will provide motivation for behavioral adjustments, although conditions which evoke such responses could often be sustained for long periods.

Most complex and non-uniform thermal environments can be described in terms of operative temperature as developed by Gagge.[3] The operative temperature of a complex thermal environment is the temperature of a uniform environment with still air at one atmosphere with equal air temperature and mean radiant temperature which transfers heat to the body at a rate equal to that of the complex environment. Figure 1 illustrates the range of operative environmental temperatures found on the lunar surface and the operative temperature extremes on the earth surface compared with the zone within which long-term physiological thermoregulation is possible and with the range of thermal comfort.

In Figure 1 the zone of physiological thermoregulation is differentiated into zones where shivering or sweat evaporation are required, and a central zone where vasomotor adjustment alone can maintain normal body temperatures. If sweat evaporation is limited or impossible, the acceptable zone for physiological regulation is considerably reduced. Conditions outside the physiologically acceptable range require behavioral adjustments, and conditions outside the comfort zone will result in some impairment in performance of complex tasks, and, generally, will provide motivation for behavioral adjustments.

In the space environment, it is necessary for reasons other than man's thermal environment to provide him with a full pressure suit. The suit can then be designed to provide considerable attenuation of the thermal extremes encountered in deep space or on the lunar surface.

The design specifications for the Apollo EVA suit system called for a maximum heat gain from the environment of 70 watts and a maximum heat loss to the environment of 96 watts. Although this very efficient insulation reduces the effective environmental thermal stress, it also interferes with the effectiveness of physi-
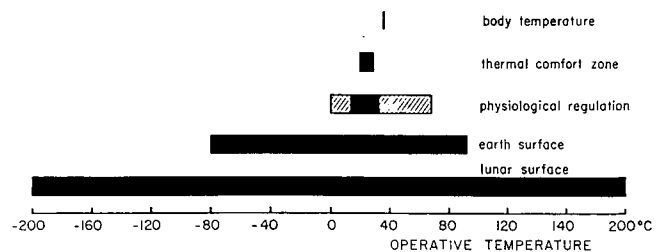

Fig. 1. Schematic representation of the operative environmental temperature range on the lunar and earth surface. Also shown are zones for long-term physiological thermoregulation and thermal comfort at rest.

85

ological regulation since sweat evaporation is limited, and the effects of vasomotor adjustments will be negligible. The mean skin temperature range which can be obtained over the long term as a result of vasomotor adjustments extends perhaps from 20°C to 37°C, and this will not materially change the thermal gradient $(\bar{T}_s - T_{amb})$ if T_{amb} is either $+ 200°C$ or $-200°C$.

The metabolic heat production during the first lunar EVA mission was estimated from simulations to average about 450 watts for the commander and 425 watts for the lunar module pilot, with peak heat production rates of 800 watts (Figure 2). Although no direct measurements of actual heat production were made during the Apollo XI lunar surface activities, estimates based on indirect measurements were given by Berry.[1] Measurements were made of heart rate, oxygen supply pressure and of inlet temperature and difference between inlet and outlet temperature of the liquid-cooled garment, and the resulting observations have been entered in Figure 3. It will be noted that both metabolic rates were somewhat lower than expected with the commander reaching only 70% of the expected average metabolic rate.

It will be clear from the schematic diagram in Figure 2 that the major thermal load during lunar surface activity in the Apollo XI mission arose from the internally produced heat of exercise. The environmental load was minor in comparison, since at the time and place of landing the sun was only 7° above the horizon which would tend to bring the operative environmental temperature close to a thermally neutral value.

The metabolic heat produced during exercise in man is eliminated via the skin. Two physiological mechanisms aid in this elimination: increased blood flow from the interior and possibly from the working muscle to the skin, and evaporation of secreted sweat. The rate of skin blood flow determines the thermal conductance from the body core to the skin, and it is increased by a rise of internal temperature and decreased by lowered skin temperature. Sweat secretion follows the same pattern and, in fact, the two responses are very closely coupled, as shown in Figure 4.

This close correlation of sweating and skin blood flow has important consequences. Under normal conditions, the thermal conductance is increased as the heat flow increases. As a result, the skin temperature during exercise is relatively independent of the level of exercise and closely dependent on the effective environmental temperature as shown by Stolwijk et al.[5]

Another consequence applies to the liquid-cooled garment and its use in the Apollo EVA suit system. In this suit system, sweating should be kept at 100 grams per hour or less in the interest of comfort and effectiveness. Referring to Figure 4, it can then be seen that the core to skin thermal conductance is limited to about 40-50 watts per °C if the skin temperature is reduced to limit sweating to less than 100 grams per hour. As reported by Waligora and Michel,[6] and by Chalmers,[2] as well as

Fig. 3. Expected and observed metabolic cost of lunar surface extra vehicular activities during Apollo XI. After Berry.[1]
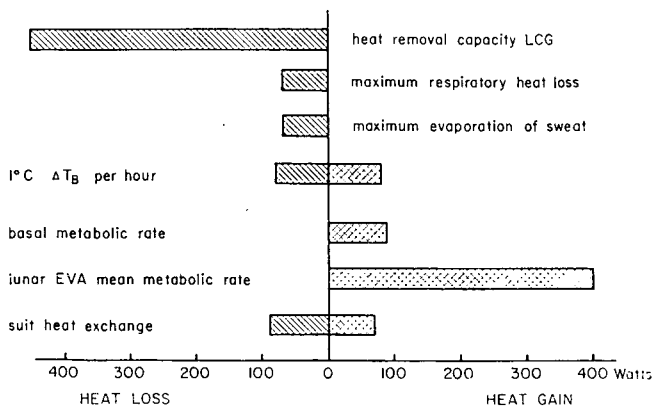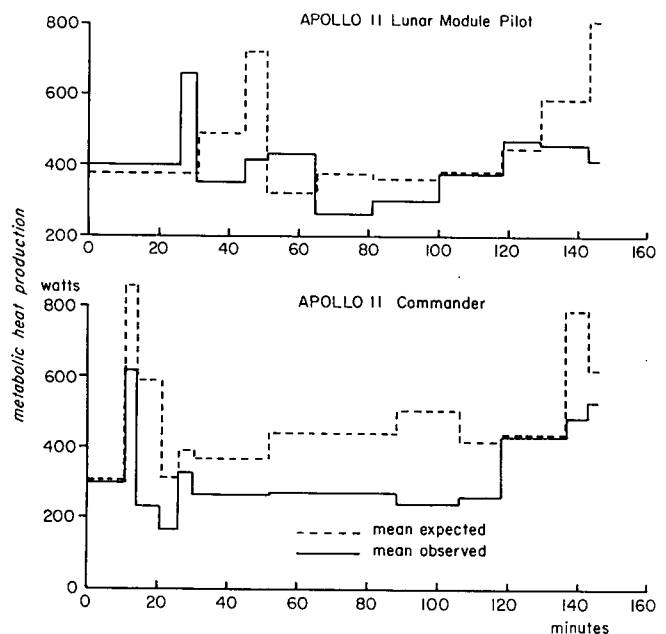
Fig. 2. Schematic presentation of thermal factors operating during lunar ambulation. In order to account for the protection afforded by allowable heat storage, the heat gain or loss rates causing a change in average body temperature of 1°C per hour are given in the center.
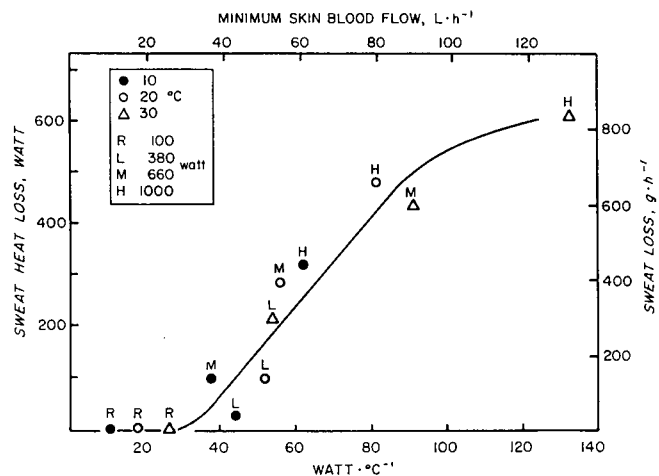
Fig. 4. Relationship between sweat secretion and core to skin thermal conductance as influenced by variations in work rate (380, 660 and 1000 watts) and in environmental temperature (10°, 20° and 30°C). Redrawn from Stolwijk et al.[5]

by Gagge et al.[4] at high metabolic rates the required amount of skin cooling may lead to skin temperatures which are incompatible with thermal comfort. It is likely that the extent of skin surface area cooled and the distribution of cooling power over the skin surface will have a material effect on the maximum work rate which can be comfortably dealt with in the Apollo EVA suit system.

The space environment contains extremes of operative temperature, but the combination of exclusively radiant heat transfer and extreme vacuum makes it possible to provide almost perfect thermal insulation from the thermal environment. This same insulation requires that metabolically produced heat be eliminated with special equipment. The major thermal peril during extravehicular activity on the lunar surface is that of hyperthermia.

The peril is thus almost diametrically opposite to that of undersea operations where the operative temperature tends to be below thermal neutrality, heat transfer is almost purely convective and where, due to high environmental pressures, it is difficult to provide effective insulation.

REFERENCES

1. BERRY, C. A.: Summary of medical experience in the Apollo 7 through 11 manned space flights. *Aerospace Med.* 41: 550-519, 1970.
2. CHALMERS, A. B.: Controlling thermal comfort in the EVA space suit. *ASHRAE J.* 12:33-38, 1970.
3. GAGGE, A. P.: Standard operative temperature, a generalized temperature scale, applicable to direct and partitional calorimetry. *Am. J. Physiol.* 131:93-103, 1940.
4. GAGGE, A. P., J. A. J. STOLWIJK and B. SALTIN: Comfort and thermal sensations and associated physiological responses during exercise at various ambient temperatures. *Environmental Res.* 2:209-229, 1969.
5. STOLWIJK, J. A. J., B. SALTIN and A. P. GAGGE: Physiological factors associated with sweating during exercise. *Aerospace Med.* 39:1101-1105, 1968.
6. WALIGORA, J. M., and E. L. MICHEL: Application of conductive cooling for working men in a thermally isolated environment. *Aerospace Med.* 39:485-487, 1968.

87<

Body Temperatures and Sweating during

Exhaustive Exercise.

B. Saltin*, A. P. Gagge, U. Bergh*,

J. A. J. Stolwijk

John B. Pierce Foundation; Yale University
New Haven, Connecticut 06519 USA

Running head: Body temperature and sweating during exhaustive work

Send correspondence to: A. Pharo Gagge
                         John B. Pierce Foundation
                         290 Congress Avenue
                         New Haven, Connecticut 06519

*Currently at: Gymnastik-och Idrottshögskolan
                Stockholm, Sweden

Abstract

SALTIN, B., A. P. GAGGE, U. BERGH, AND J. A. J. STOLWIJK. Body temperatures and sweating during exhaustive exercise. J. Appl. Physiol. (   ):   . 19 . Four normal healthy unclothed subjects exercised to exhaustion on a bicycle ergometer at 90, 100 and 115% of their maximal oxygen uptake. Ambient temperatures (T_a) ranged from 10° to 40°C (RH < 40%). Continuous observations of skin ($\bar{T}_{sk}$), rectal (T_{re}), esophageal (T_{es}) temperatures, skin sweating ($\underline{E}_{sk}$) and metabolic rate ($\underline{M}$) were made during exercise periods lasting 4-30 min. and for 15-20 min. during recovery. Muscle temperatures were sampled at end of exercise. In all our experiments $\bar{T}_{sk}$ was correlated with T_a (r > 0.95) and at a given T_a varied less than 1°C during rest, exercise and recovery. A consistent relationship between $\underline{E}_{sk}$ and body temperatures did not occur until after the first two minutes of exercise during which period $\underline{E}_{sk}$ was low or negligible. During the exercise phase, T_{re}, T_{es} and T_m appear to have equal significance when each is paired with $\bar{T}_{sk}$ in a multiple regression with $\underline{E}_{sk}$. During the recovery phase T_{re} appeared to be more significantly related to $\underline{E}_{sk}$ than T_{es} or T_m. The time to exhaustion was 15-20 min., 7-8 min. and approximately 4 min. for 90%, 100% and 115% maximum work respectively. Exhaustion was always associated with a working muscle temperature of 40°C.

REGULATORY SWEATING HAS BEEN PREDICTED with a reasonable reliability both at onset of work and during the steady state phase of submaximal exercise by a linear function of skin and internal body temperatures (11, 18, 19, 21). During the recovery from exercise no linear combination of these temperatures gave an adequate prediction of skin sweating (19). In these earlier reports the metabolic rates did not exceed 75% of the individual's maximal oxygen uptake, and the environmental temperatures varied between 10-30°C. Internal body temperatures, measured in the esophagus or rectally after 20-30 minutes of exercise were well related to the oxygen uptake during work expressed in percent of the subjects' maximal value (11, 18, 20), while mean skin temperature was a linear function of the ambient temperature (19, 21). The present investigation is being initiated to evaluate to what extent the above mentioned relationships hold at very high work rates. When heavy exercise is performed in a warm environment there is in addition to the nutritive blood flow to the muscles an increased blood flow to the skin. The question arises whether this increase results in either a lowered maximal oxygen uptake or in reduced work performance time or both. Finally, since the temperature in the exercising muscles reaches a level higher than any other in the body during heavy work (1, 18), what is the relation between exhaustion and muscle temperature?

Subjects

Selected anthropometric and exercise data for seven subjects in the

present experimental series are indicated in Table 1. The majority of the experiments reported here were performed on four subjects (U.B., Y.N., M.R. and B.S.) in the summer of 1969 in New Haven. The supplementary experiments on three subjects (R.N., L.W. and E.W.) were done at Stockholm. The work loads used for each subject are also listed in Table 1.

All subjects (except M.R. and Y.N.) had previously participated in regular endurance activities. However, before participating in any experimental runs in the present study, these two subjects trained 1/2 hour a day on the bicycle ergometer for three weeks prior to their experiments and increased their maximal oxygen uptake by 12 and 15% respectively. During the experimental period no significant change was observed in the maximal oxygen uptake in any of the subjects. The "leveling off criterion" (6) was used to establish maximal oxygen uptake. The reproducibility of the maximal oxygen uptake determinations expressed as a percent of the standard error of the mean was 3%. This value includes methodological errors as well as day-to-day variations in maximal oxygen uptake.

TABLE 1.   Anthropometric and Exercise Data.

Subj.	Age yr.	Height cm	Weight kg	Surface Area m^2	Highest observed values for Heart Rate, beats/min	Highest observed values for Oxygen uptake 1/min	Work loads (W) used[‡] 91% (87–93)	100% (97–105)	115% (110–126)
U.B.[*]	26	182	67	1.87	195	4.3	255	284	330
Y.N.[*]	29	165	63	1.69	200	3.4	182	210	–
M.R.[*]	21	178	67	1.83	188	3.9	240	270	300
B.S.[*]	34	187	90	2.18	179	5.6	345	390	–
$\bar{m}$	27	178	72	1.89	191	4.3	260	290	315
R.N.[†]	22	179	71	1.90	189	4.1	255	284	350
L.W.[†]	28	180	76	1.97	189	4.3	265	300	345
E.W.[†]	27	181	74	1.96	186	4.7	294	362	390
$\bar{m}$	26	180	74	1.94	188	4.4	268	315	361

[*]at New Haven
[†]at Stockholm

[‡]The experimental work loads were chosen by using the subjects normal mechanical work efficiency and by assuming a complete aerobic energy yield; the oxygen uptake demanded for the different work loads have been calculated and expressed in percent of each individual's maximal oxygen uptake (5).

Methods and Procedure

The methods and the procedures for the measurements of oxygen uptake, heart rate, rectal, esophageal and mean skin temperatures were the same as those reported before (19). Work was performed on a mechanically braked bicycle ergometer, placed on a Potter Platform Scale. The scale determined the rate of weight loss of the subjects, caused by evaporation from the body (19). The ambient air and wet bulb temperatures were recorded continuously. All the above measurements were produced every minute in the form of a millivolt output, which in turn was converted to digital form in an A-D converter and stored in the disc memory of an IBM 1131 computer during the entire experimental period. At the end of the experiment the computer, when supplied with the appropriate calibration constants, converted the basic millivolt data into the corresponding thermal units -- in this case, degrees centigrade and watts per square meter. In addition, it was possible to measure continuously the rate of weight loss, oxygen uptake and any single body temperature on separate millivolt strip recorders. Muscle temperature was measured in the lateral portion of the quadriceps muscle with the "needle" method (18) immediately at the end of the exercise. In the supplementary experiments at Stockholm the catheter technique (18) was also used to record continuously muscle temperature. Immediately after experimental runs blood lactate was determined by the modified Baker-Summerson method (22) on finger-tip blood.

Protocols

The New Haven climatic chamber was the same as used in our earlier studies. The present experiments were performed at four environmental temperatures, namely, 10°, 20°, 30° and 40°C, and the dew point temperature was normally below 15°C. The air movement around the subject in the chamber resulted in a combined heat transfer coefficient of 9.2 $W/(m^2 \cdot °C)$ when resting. While pedalling at 60 rpm the combined heat transfer coefficient rose to 11.2 $W/(m^2 \cdot °C)$ (10).

The protocol of a typical experiment was as follows. The subjects were dressed in shorts and gym shoes. The various thermocouples were placed on the subjects while sitting on the bicycle ergometer on the platform scale in the chamber. The initial exposure before maximal exercise was approximately 30 minutes. In the middle of this time period, the subjects exercised at 60 W (approx. 20% maximal O_2 uptake) for 10 minutes but stopped always 10 minutes before the start of the exhaustive exercise. The purpose of the pre-exercises was to avoid extreme body cooling prior to the maximal exercise such as occurs at 10° and 20°C. At least 5 minutes of data were recorded before the start of the maximal work. The subject then worked to exhaustion on a fixed work load that was calculated to demand 90, 100 or 115% of their maximal oxygen uptake (see Table 1). Each exercise period was followed by at least 15 minutes of rest. Oxygen uptake and weight loss were monitored continuously while the other data were recorded every minute. Complete runs at three ambient temperatures were performed on all four subjects at the 90 and 100% work load (WL). The highest work load (115% of max $\dot{V}O_2$) was performed only by UB and MR (see Table 1 and 2). Each subject performed only one experiment during the same day.

The protocol for the Stockholm experiments was identical to the one described above but was limited to 20°C. Oxygen uptake, heart rate, esophageal and muscle temperature were measured continuously.

Calculations

Our present methods are generally the same as the procedures reported in an earlier communication (19). For each minute of the experimental runs the heat partition is given by the following basic equation:

$$\underline{S} = \underline{M} - \underline{E} - \underline{W} - h\,(\overline{T}_{sk} - T_a) \quad , \quad \text{in } W/m^2 \tag{1}$$

where

$\underline{S}$ = rate of body heat storage ,

(+ for heating; − for cooling)

$\underline{M}$ = metabolic rate ,

$\underline{W}$ = work rate ,

h = combined heat transfer coefficient ,

and $\underline{E}$ = total evaporative heat loss ,

which was calculated from the rate of total body weight loss, as measured on the Potter balance.

The total loss $\underline{E}$ is divided into two components

$$\underline{E} = \underline{E}_{res} + \underline{E}_{sk} \quad , \quad\quad W/m^2 \tag{2}$$

where $\underline{E}_{res}$ = the respired vapor heat loss from the lungs ,

and $\underline{E}_{sk}$ = the total evaporative heat loss from the skin surface.

The respired loss $\underline{E}_{res}$ was calculated by the basic relation originally developed by Fanger (2) but with a correction factor F now added. Above a level of 65% of max $\dot{V}O_2$ ($\approx$ 2.6 1/min), the minute-volume, on which the respiratory vapor heat loss depends, is no longer linear with the metabolic rate but increases more than proportionally (17). The following relations have been developed for this study to fit the curve so reported.

$$\underline{E}_{res} = 0.0023\,\underline{M}\,(44.0 - \phi_a \cdot P_a) \cdot F \tag{3}$$

where ϕ_a = relative humidity in test chamber as a fraction,

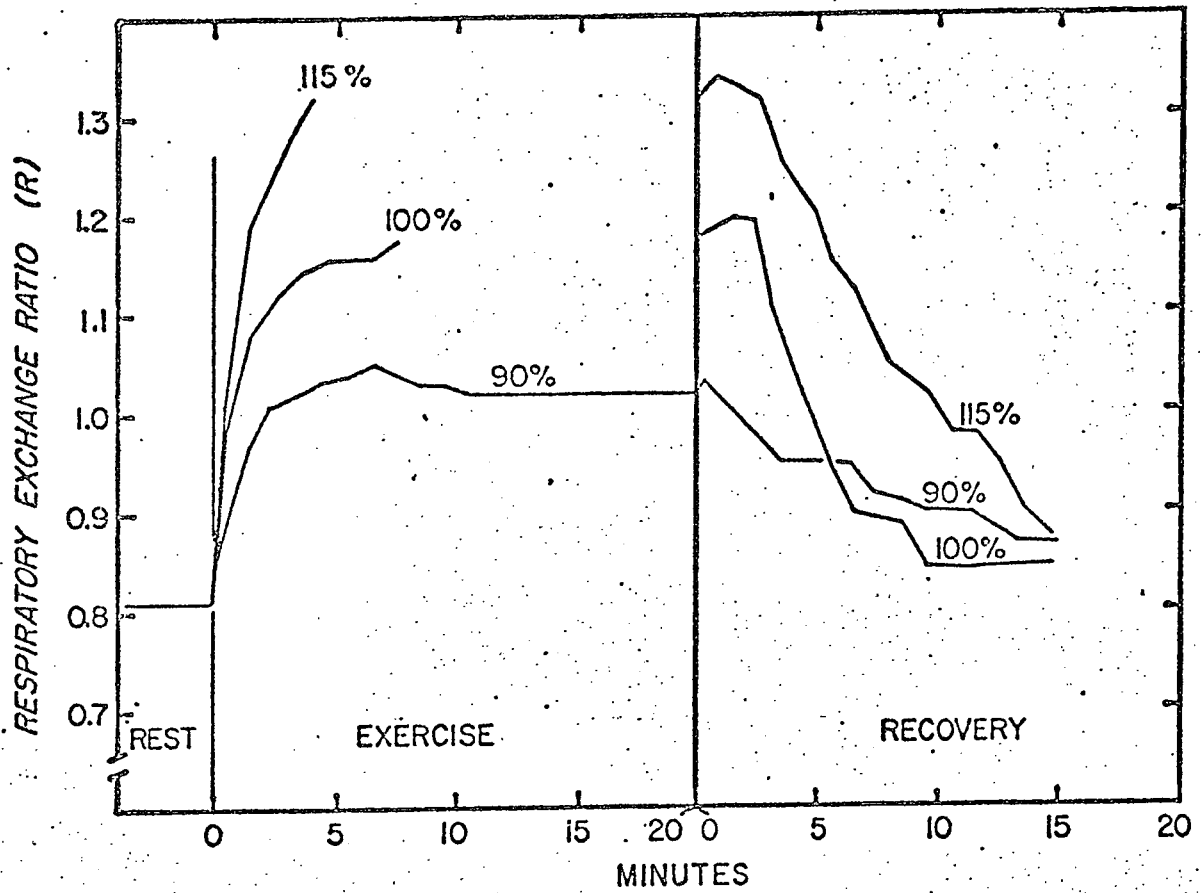and P_a = saturated vapor pressure at temperature T_a.

Fig. 1. Mean values of the instantaneous Respiratory Exchange Ratio (R) for three subjects (RN, LW and EW) observed during exercise to exhaustion for three levels indicated.

Below an oxygen uptake of 2.6 1/min, F = unity.

Above 2.6 1/min

$$F = 1 + 0.106 \, (\dot{V}_{O_2} - 2.6)^2 \qquad \text{N.D.} \qquad (4)$$

In eq. (4) $\dot{V}_{O_2}$ in liter/min is approximately equal to $(\underline{M} \cdot A_D)/(5.82 \cdot 60)$ where 5.8 W·hr is the metabolic heat equivalent of 1 liter of oxygen in watts (RQ = 1).

In addition to the respired vapor loss the observed rate of weight loss on the Potter Scale must also be corrected for the difference between the CO_2 loss over the O_2 gain. For each liter of oxygen consumed, when the Respiratory Exchange Ratio (R) of CO_2 to O_2 is unity, this net loss is (44-32)/22.4 or 0.536 g/liter where 44 and 32 are the respective molecular weights of CO_2 and O_2 in grams/mole and 22.4 are the liters/mole at (STP). For $\dot{V}_{O_2}$ in liters/min this correction for $\dot{w}$ in g/min on the Potter Scale is $0.536 \cdot (R) \cdot \dot{V}_{O_2}$. During exercise to exhaustion and recovery afterwards, the (R) varies considerably. The general pattern of R-changes, observed on the Stockholm subjects, for the standard protocol times at the three levels of exhaustive exercise is shown in Fig. 1. These general patterns were used to make minute-by-minute corrections of the observed $\dot{w}$ for the respired weight loss due to $CO_2 - O_2$ exchange.

The skin evaporation, E_{sk}, based on corrected measurements of $\dot{w}$ is therefore

$$E_{sk} = E - E_{res} \qquad (5)$$

In the present study the values of the coefficients h and h_c, which are necessary to calculate the dry heat exchange and the maximum evaporative heat loss from the skin surface E_{max}, were determined directly by the naphthalene sublimation method of Nishi and Gagge (10). The values used for the present study were:

a. while resting on bicycle saddle   h = 9.23; h_c = 4.0 $W/(m^2 \cdot {}^\circ C)$

b. while pedalling at 60 rpm   h = 11.23; h_c = 6.0 $W/(m^2 \cdot {}^\circ C)$.

Table 2. Mean values (±SE) for certain variables in four subjects (UB, YN, MR and BS) exercising to exhaustion.

Work load, W

			260		290		315
		Cond.	n = 4	Cond.	n = 4	Cond.	n = 2
Oxygen uptake	10°	A*	92±2		–		–
% of max $\dot{V}_{O_2}$	20°	B	90±2	D*	100±1	G*	100±2
	30°	C	92±2	E	99±1		–
	40°		–	F	100±1	H	100±1
Heart Rate	10°		175±4		–		–
beats/min	20°		177±3		181±3		181±2
	30°		181±3		182±2		–
	40°		–		182±2		183±3
Blood lactate	10°		10.1±1.4		–		–
mM/1	20°		11.4±1.2		12.7±1.1		13.0±2.0
	30°		12.0±0.8		13.6±0.6		–
	40°		–		13.0±1.4		14.8±1.6
Muscle Temperature	10°		39.3±0.3		–		–
°C	20°		39.6±0.2		39.6±0.2		38.8±0.4
	30°		39.8±0.2		39.8±0.15		–
	40°		–		39.9±0.15		39.2±0.2
Work Time	10°		100±3.8 (20.1)[+]		–		–
% t	20°		86±7.2		100±0 (8.3)[+]		100±0 (4.0)
	30°		81±2.7		97±2.9		–
	40°		–		84±3.1		100±0

*The capital letters denote the eight different conditions in which complete runs on the New Haven subjects were obtained.   .98‹

[+]The value within parenthesis denote the mean value for the work time in min. reached at each work load exercising in the lowest environmental temperature. The work time attained in the other environments are expressed in percent of this value.

Table 3. The effect of humidity and body preheating* on the maximal oxygen uptake and work time.

	Work time min	T_a °C	T_{wet} °C	$\bar{T}_{sk}$ °C	T_{re} °C	T_{es} °C	T_m °C	Oxygen uptake l/min	Heart rate beats/min.
	0			35.4	36.9	36.8	37.1		
	8.0	40.1	23.1	35.0	37.7	38.9	40.1	4.26	189
U.B.	0			36.2	37.0	36.9			
	8.2	41.8	32.2	36.1	37.9	39.2		4.34	190
	*0			35.2	37.5	37.4	38.3		
	6.3	40.0	21.8	35.0	38.5	38.8	40.5	4.01	190
	0			34.3	36.9	36.9	37.1		
	6.0	40.2	21.9	34.1	38.1	38.7	40.3	5.64	168
B.S.	0			35.2	37.2	36.9			
	6.0	41.7	30.4	35.3	38.2	38.8		5.62	172
	*0			34.7	37.6	37.7	38.4		
	4.5	40.3	21.5	34.5	38.9	39.0	40.4	5.19	174

*15 min. of work at 50% of Max $\dot{V}_{O_2}$ preceded the maximal exercise.

Results

All subjects worked to exhaustion in each experiment. It was then possible to evaluate to what extent maximal work time was affected by performing the assigned work load at different ambient temperatures. In each subject at the 90 and 100% loads the best work performance was achieved in the coldest environment (Table 2). By comparing T_a of 10° with 30°C, the mean value for the work time at the 90% work load fell from 20.1 to 16.3 minutes (or 19% drop); by comparing T_a of 20° with 40°C, the drop was from 8 to 6.3 minutes or 16% drop. At the highest work load (115%) a work time of 4 minutes was maintained by two subjects (UB and MR) at both 20° and 40°C. At the end of each exhaustive work the blood lactate concentration was above 8 mmoles per liter. No significant difference in the maximal blood lactate concentration was observed as an effect of differing ambient temperatures. However, slightly higher mean values of blood lactate were observed after work in the 30° and 40°C environment when compared to the 10° and 20°C environment.

At onset of exercise oxygen uptake increased rapidly; the rate of increase was most pronounced with the heaviest work load. Environmental temperature had no effect on the acceleration of the oxygen uptake, nor did ambient temperature influenc the oxygen uptake attained at the end of the exhaustive exercise. Since a reduction in the maximal oxygen uptake has been reported earlier when exercising in very warm rooms (8, 13, 16), additional experiments were performed on subjects UB and BS to further elucidate this question: these two subjects exercised at maximal work (100% of max $\dot{V}O_2$) load three times at an ambient temperature of 40°C (Table 3). In the first two runs no prior warming-up was performed but the relative humidity was varied from around 20 up to 60%. On both occasions maximal oxygen uptake and normal work time was reached. In the third run the subjects exercised at 50% of their maximal
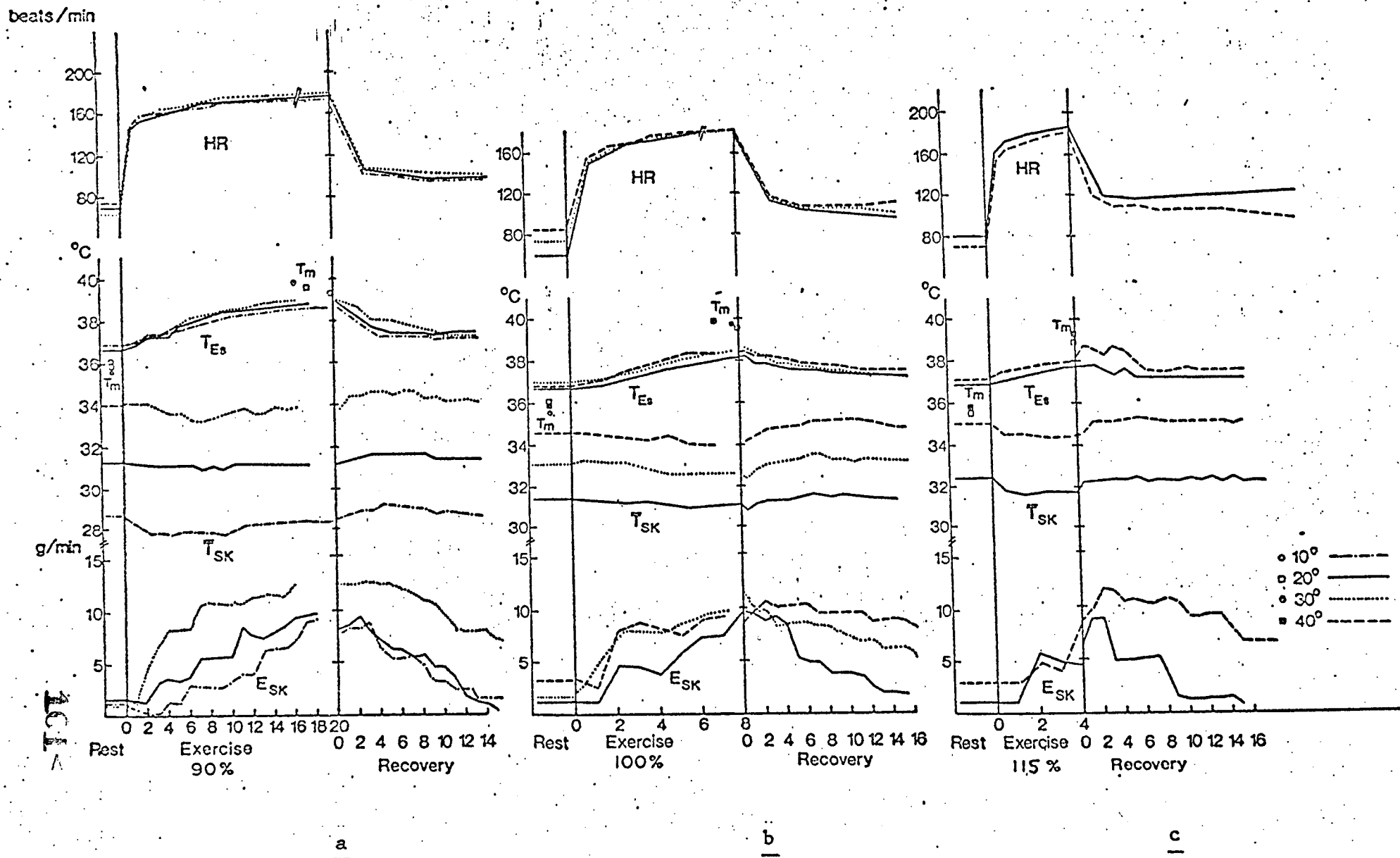
Figure 2a, 2b, 2c. Mean values for continuous observations of skin sweating (E_{sk}), mean skin temperature ($\bar{T}_{sk}$), esophageal temperature (T_e), and heart rate (HR) before, during and after exercise at exhaustive work loads performed at ambient temperatures from 10 to 40°C (a 90%, b 100% and c 115% of max $\dot{V}_{O_2}$). Included are also the temperatures determined in thigh muscle (T_m) just before and immediately after the exercise.
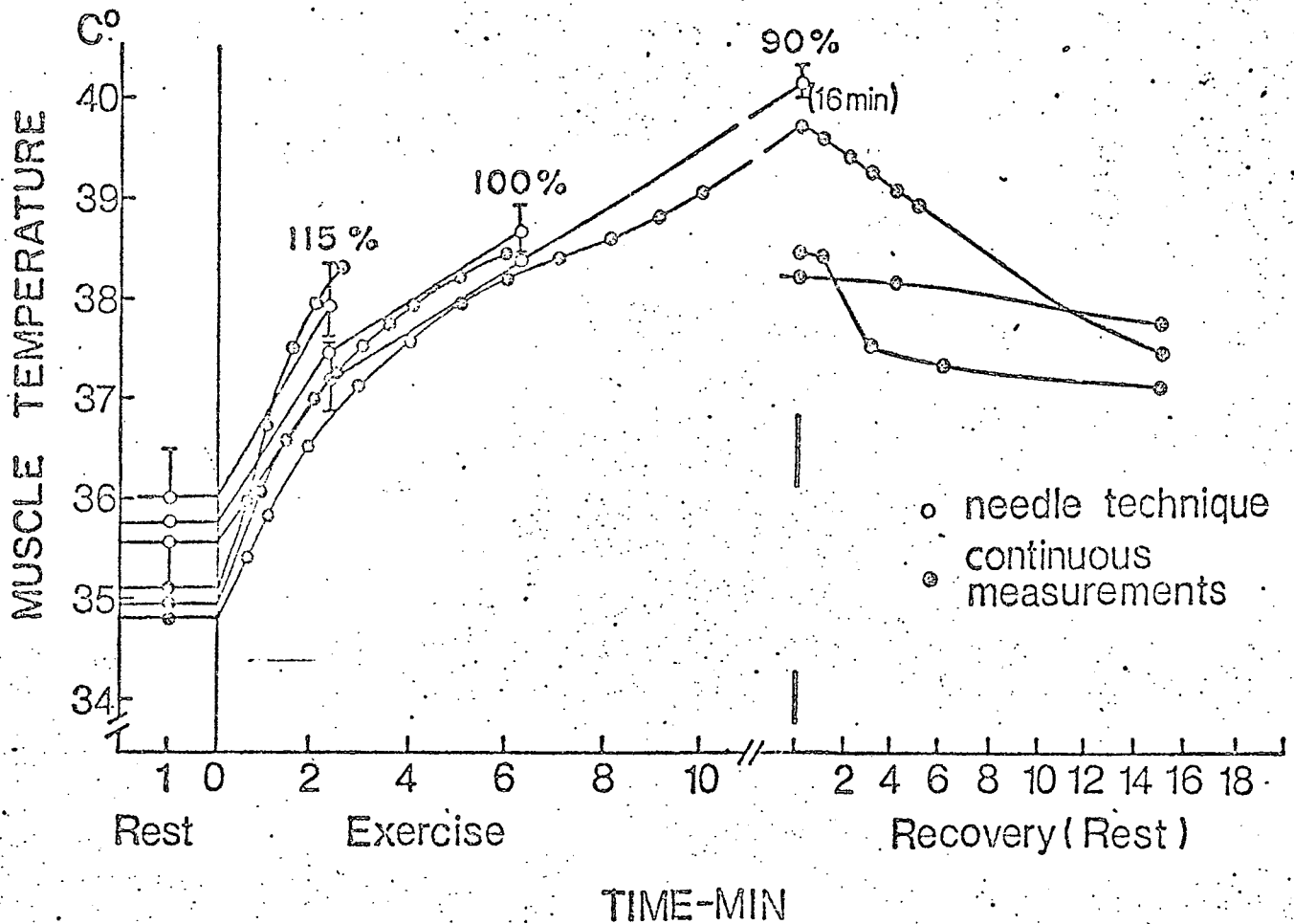
Fig. 3. The mean values for the highest observed temperature in the lateral portions of the quadriceps muscles determined in three subjects (RN, LW and EW) before, during and after work at three different work loads (90%, 100% and 115% max $\dot{V}_{O_2}$) at 20°C. It should be noticed that the measurements with the two different techniques to determine muscle temperature were performed in separate experiments.

oxygen uptake for 15 minutes, prior to the test run at 100% work load. In this later run an 8-10% reduction in the maximal oxygen uptake was observed in both subjects, and there was a noticeable reduction in work time.

Although the rate of oxygen uptake during maximal exercise without preheating, was unaffected by environmental temperature, the heart rate was influenced (Fig.2). In the warmer environments (30° and 40°C) heart rate accelerated faster at onset of exercise and reached a higher level at exhaustion. This rate did not return to normal as quickly during the recovery phase when compared with the experiments in the cooler environments (10° and 20°C).

At cessation of the exhaustive work, muscle temperatures reaching 39.8-39.9°C were observed for all subjects when exercising at the 90 and 100% work load and at 30° and 40°C. This high temperature was found also for subjects UB and BS working at the 90% WL in the 20°C environment. At the heaviest load, where work time was only 4 minutes, muscle temperatures only reached 39°C. These relationships are demonstrated in Table 2.

To evaluate the time course for the increase in muscle temperature during exhaustive exercise and analyze if there was a certain critical temperature level coinciding with exhaustion, additional experiments were performed on subjects RN, LW and EW in Sweden. The heavier work load caused a faster increase in the muscle temperature. However, a muscle temperature of 40°C was reached only at the lowest work load (90%), where work time was 20 minutes (Fig. 3). The needle and the catheter method of determining the muscle temperature produced identical results. Two subjects (RN, LW) also exercised at the highest and the lowest work load to exhaustion at 20°C with preheating which consisted of 15 minutes of work at 50% of max $\dot{V}O_2$. At the 90% work load with the longest work

time ($\simeq$ 20 minutes) muscle temperature was 40.1°C at the end of the exhaustive exercise regardless of preheating or not. At the highest work load (work time $\simeq$ 3.0 minutes) the final muscle temperature was 38.5°C without perheating and 39.2°C with warming-up exercise. It is worth mentioning that the warming-up procedure prior to maximal work improved the performance time about 20% at both 90% and 100% levels of work when exercising at 20°C. This fact is in line with the observed faster increase in the oxygen uptake at onset of exercise after warming-up.

In our earlier study (19) it was possible to show from continuous measurements that T_m rose immediately at the start of exercise and followed a fixed pattern in time for a given work load. As may be seen in Fig. 3 the upper limit for T_m is apparently 40.0°C ± 0.5°C and is relatively independent of the difference in maximal metabolic effort. For each of the eight conditions of the New Haven study it was then possible to estimate from start of exercise the probable values for T_m from the observed data at the end of exercise. No such precise predictions of T_m were possible during the recovery phase.

At onset of exercise esophageal temperature responded faster than the rectal, T_{re}; at exhaustion T_{es} was approximately 0.6°C above T_{re}. In fact T_{re} always reached its peak value one minute or two after the end of exercise. The same general pattern was observed for T_{es} and T_{re} at all environmental temperatures, but both reached slightly higher values in the two warmer environments (T_a 30° and 40°C). Body cooling in the recovery phase at higher ambient temperatures was very slow as indicated by the slow return to pre-exercising values for the core temperatures. During exercise on relative work loads up to 75% both T_{es} and T_{re} usually reach a plateau after 10 and 30 min. of exercise respectively. In our present study only T_{es} reached a plateau before end of the 90% work load.

The observed esophageal temperatures at end of 90% work level which lasted for 20 min. would also fall near the predicted regression line for T_{es} when related to oxygen uptake for submaximal exercise expressed as percent of max $\dot{V}O_2$ (20). Prewarming by exercise had little effect on the final T_{es} but did markedly affect the final T_{re} (see Table 3), which is a reflection of the longer time necessary for T_{re} to reach its level.

For all conditions studied only minor changes were noticed in mean skin temperature during the rest-exercise-recovery transients. The present results for $\bar{T}_{sk}$ thus follow almost exactly the pattern earlier observed (11, 19, 21). For the present series of experiments mean skin temperature during exhaustive exercise was more closely related to the ambient temperature ($r^2=0.92$) and was uninfluenced by the metabolic rate.

The increase in the regulatory skin sweating occurred within two minutes after onset of exercise (Fig. 2). This was the case also at the heaviest work load in the warmest environment. Regulatory sweating occurred at all work loads and environmental temperatures. It may then be worthwhile to compare in some detail how skin sweating (in gm/min), mean skin temperature ($\bar{T}_{sk}$), esophageal temperature (T_{es}) and heart rate varied just before, during and after the exhaustive exercise. Mean results for the different conditions (A-H) are illustrated in Fig. 2. Included are also the mean values for the muscle temperature obtained before and at exhaustion. After an initial delay of at least 1 min at onset of exercise, E_{sk} increased during the 3rd and 4th minute of exercise in all experimental situations. This increase was very marked in all conditions except for exercising at the 90% WL in the 10° and 20°C environment. In these cooler experiments a more gradual increase in E_{sk} during the whole exercise period was observed. At 30° and 40°C a rather small but further increase in

E_{sk} occurred after the first 3-5 minutes of exercise. At all work levels the rate of sweating at exhaustion was related to the ambient temperature, but only minor differences were observed between these different very high work levels. During the exercise phase for all conditions E_{sk} was less than E_{max}.

In the recovery phase, E_{sk} continued to increase slightly or stayed the same for 1-3 minutes as compared to the last minute of exercise and its return to pre-exercising values was gradual. This drop in E_{sk} did not occur, however, within 15 minutes of rest in any of the experiments performed at 30° and 40°C. In the present experiments E_{max}, i.e., the maximum rate of evaporation that can take place from the body surface at the observed $\bar{T}_s$, T_a and RH, was usually above the observed E_{sk} during both exercise and recovery.

As pointed out earlier exercise barely affected mean skin temperature at all. The largest variation ± 0.5°C in $\bar{T}_{sk}$ occurred at 10°C; above 30°C the variation was negligible. Esophageal temperature had approximately the same time lag as E_{sk} but increased gradually during the whole exercise period. A minor effect of the ambient temperature was noticed on T_{es} in the recovery phase. Although no continuous measurements of the muscle temperature were performed in the experiments illustrated in Fig. 1, we know from our previous studies and from the data in Fig. 2 that T_m increases very soon after onset of exercise and that the rate of increase is the highest during the first few minutes of work. In the recovery phase T_m approached 37°C within 15 minutes but never dropped to pre-exercise values.
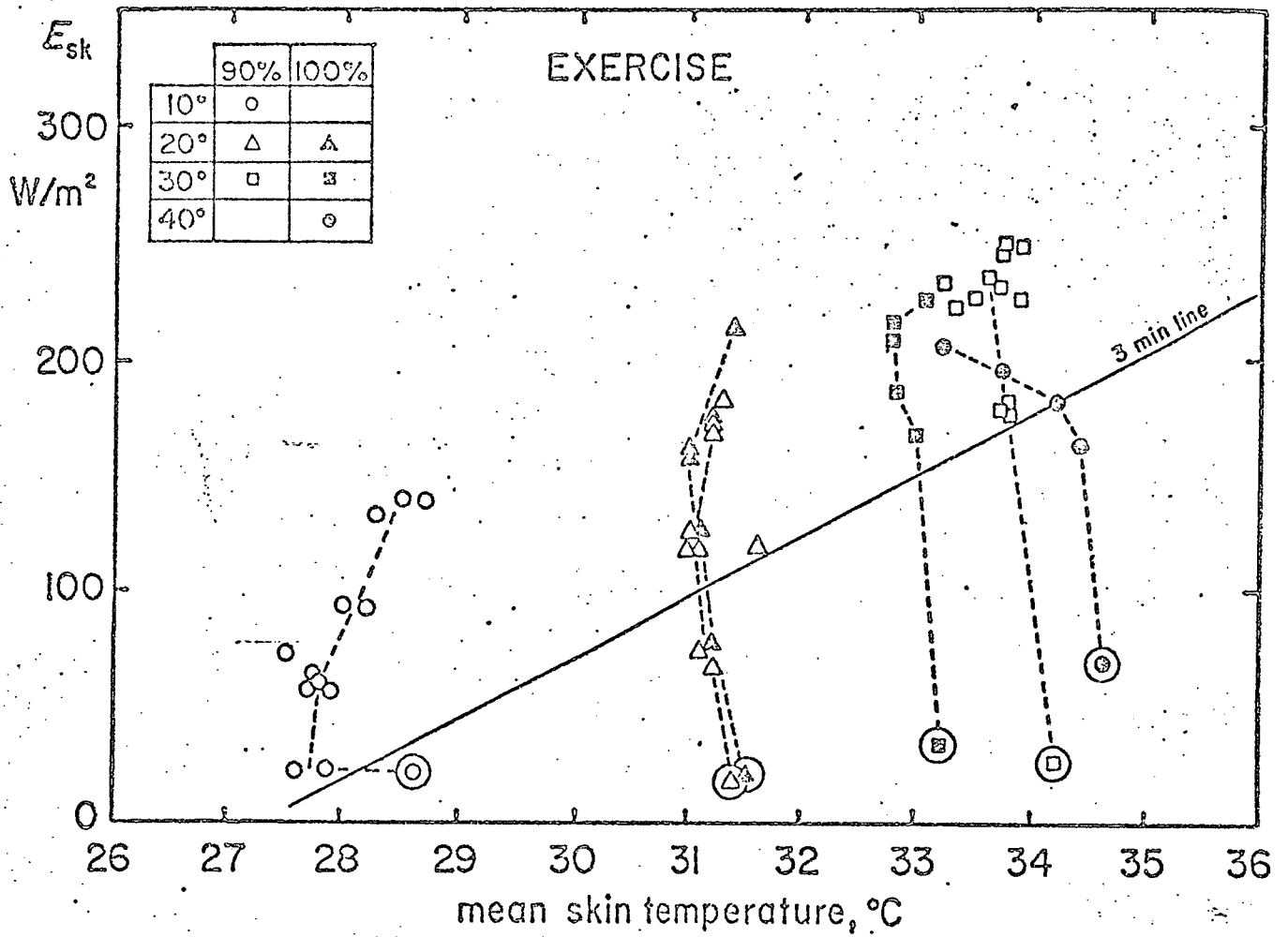
Fig. 4.   The variation of skin sweating with mean skin temperature during
          exercise. In this and Fig. 5, 6 and 7 the circled points represent
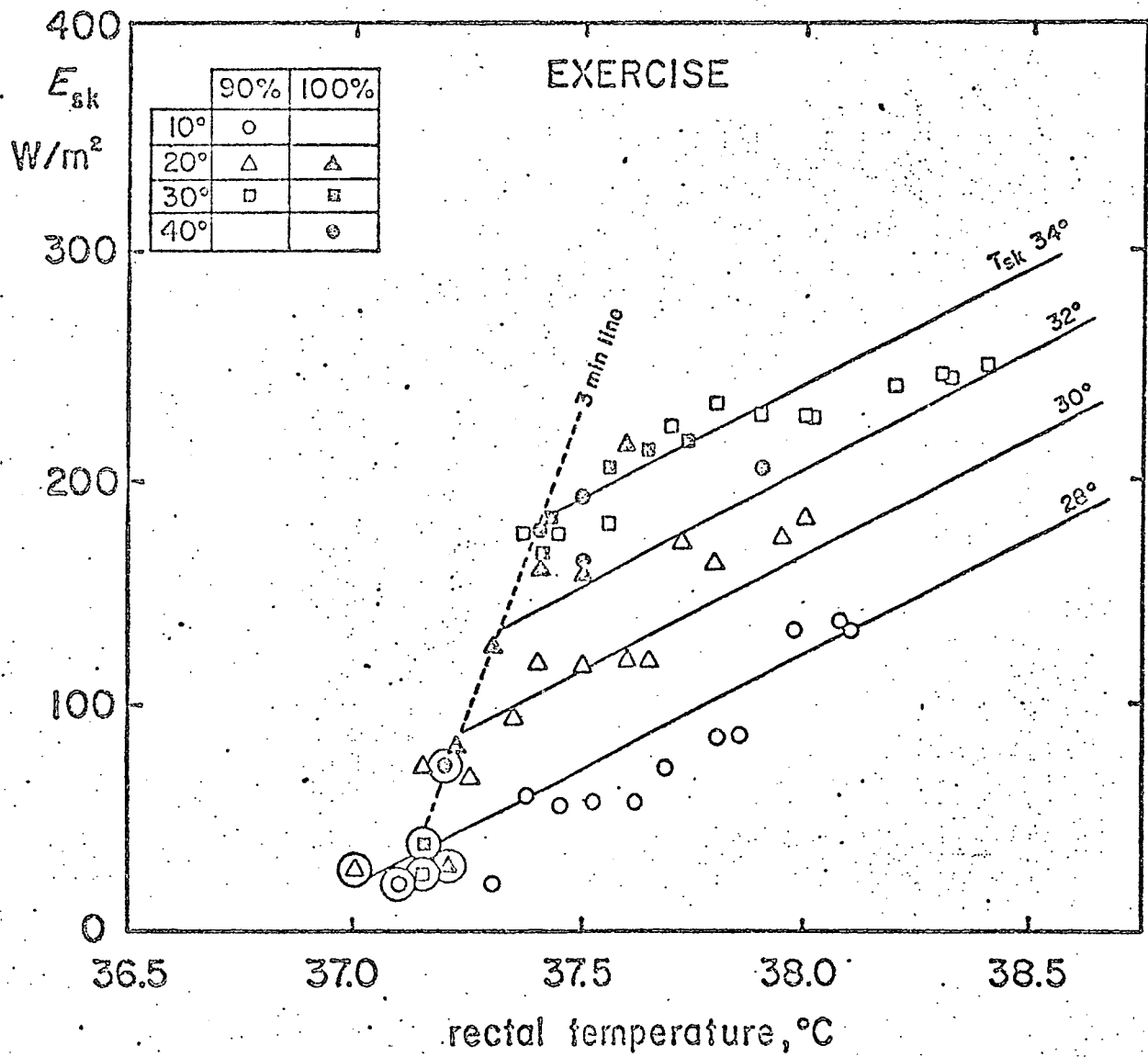   temperatures at the start of exercise.

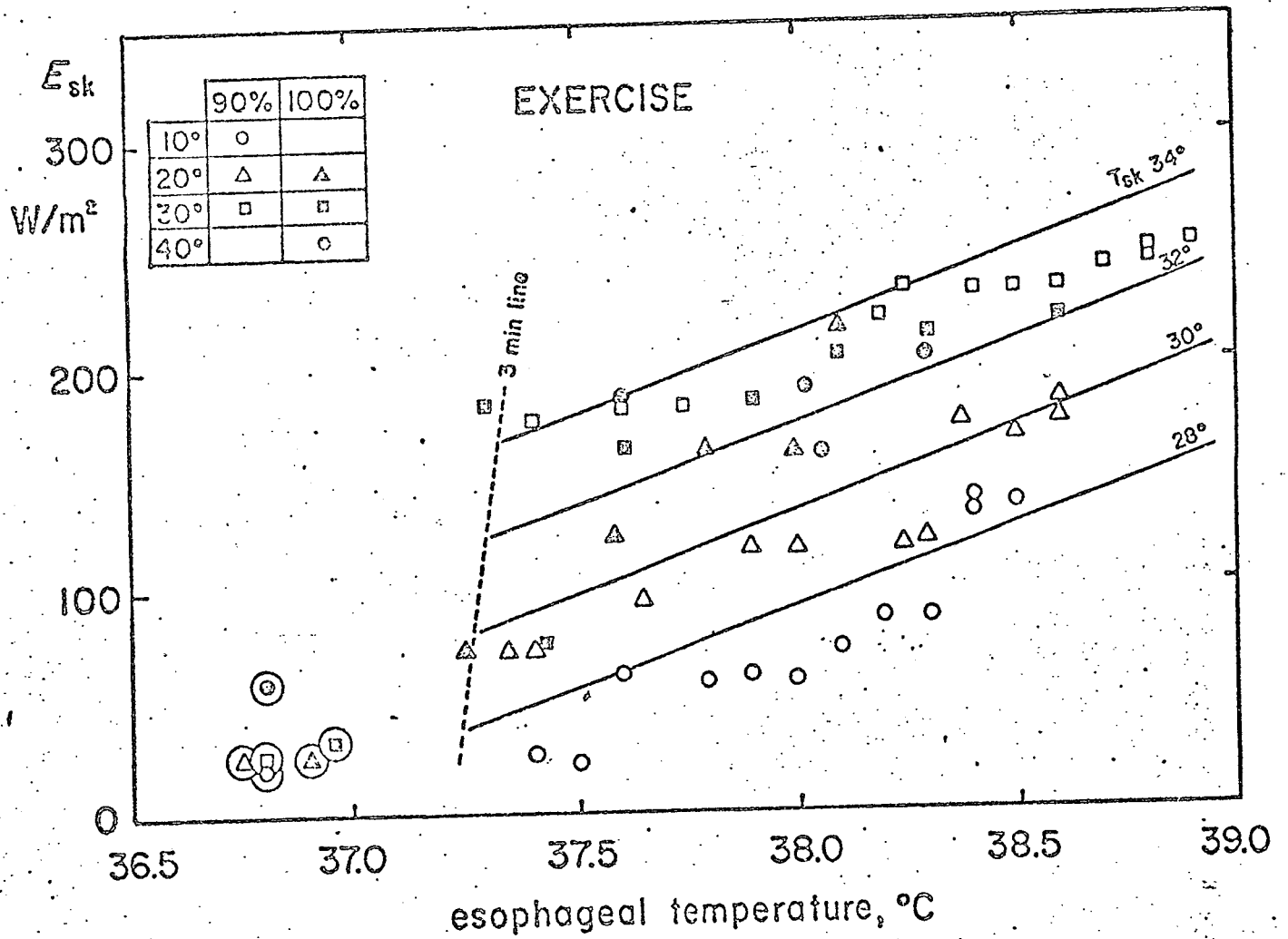Fig. 5. The variation of skin sweating with rectal temperature during exercise.

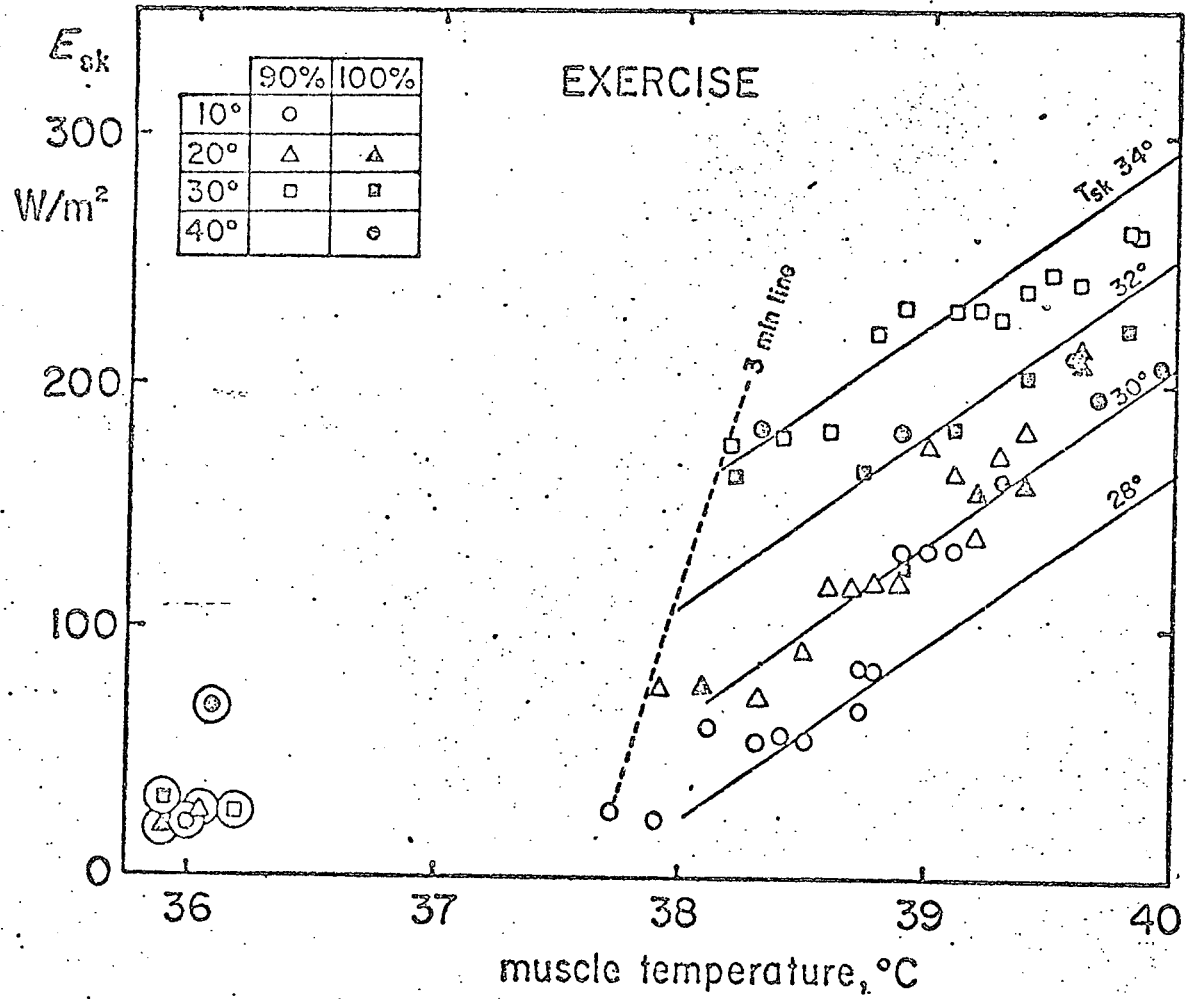Fig. 6.   The variation of skin sweating with T_{es} during exercise.

Fig. 7.   The variation of skin sweating E_{sk} and working muscle temperature T_{re} during exercise.

Figs. 4, 5, 6, 7   In Fig. 4, 5, 6 and 7, regulatory sweating E_{sk} (W/m^2) has been plotted against average skin, rectal, esophageal and muscle temperature. Each observation represents an average of three subjects (UB, BS, and MR) adjusted to a common time scale. The point for time 0 is circled; the second point plotted represents three minutes after the start of exercise. Thereafter all points for successive minutes are shown. A line indicating the sequence of observation has been drawn from the zero point through the third and fourth minutes. The readings during the transition period (1-2 min) were usually near zero. After the first three minutes of exercise a consistent relationship between E_{sk} and body temperature appears in each case. The data used are for 90% and 100% maximal work since these conditions cover at least seven minutes of exercise and represent over 3/4 of our observed data.

Fig. 4 for skin temperature shows again that the level of skin temperature is primarily a function of air temperature and varies little in time during the course of exercise. At 40°C the 1.5°C drop in skin temperature for the 100% exercise level was probably caused by the excessive sweating at the low humidity involved (40°C DB; 21°C Wet; 10°C Dew; and 15% RH).

During the first three minutes of exercise there was only 0.1-0.2°C change in rectal temperature, compared to almost 1°C for T_{es} and 2°C for T_m. After 3 minutes of exercise E_{sk} varied with T_{re}, T_{es} and T_m in the linear pattern one would expect if E_{sk} is a linear function of each and $\bar{T}_{sk}$. There appears to be a critical temperature for each body temperature above which the observed consistent relationship with E_{sk} appears. At 10°C the critical levels for T_{re}, T_{es} and T_m are 37°C, 37.3°C and 38°C respectively. In contrast at 30°C and 40°C the critical level for T_{es} seems unchanged while those for T_{re} and T_m rise slightly to 37.4°C and 38.2°C respectively.
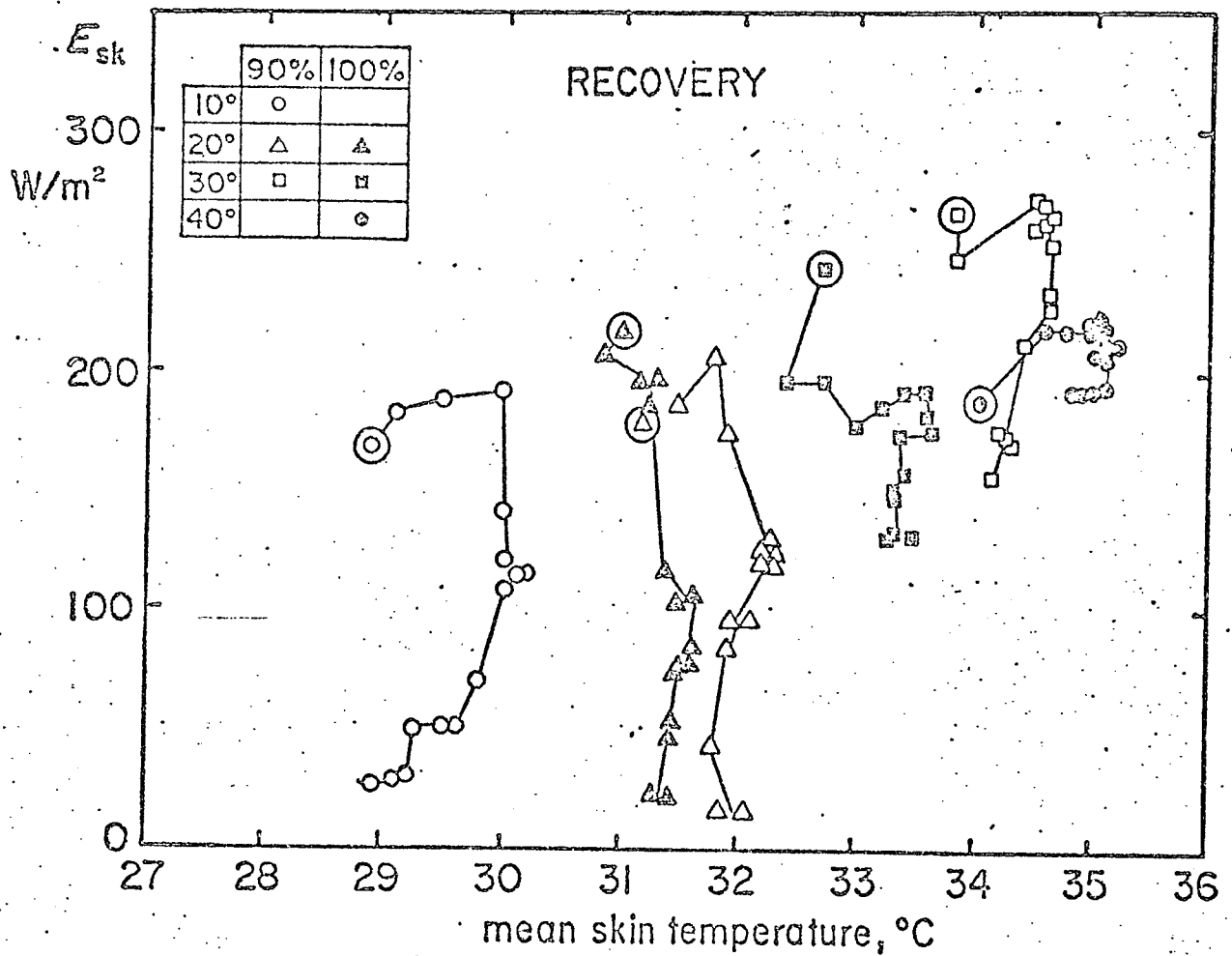
Fig. 8.   Skin Evaporative heat loss and skin temperature during recovery.
In this and Fig. 9 and 10 the circled points represent temperatures
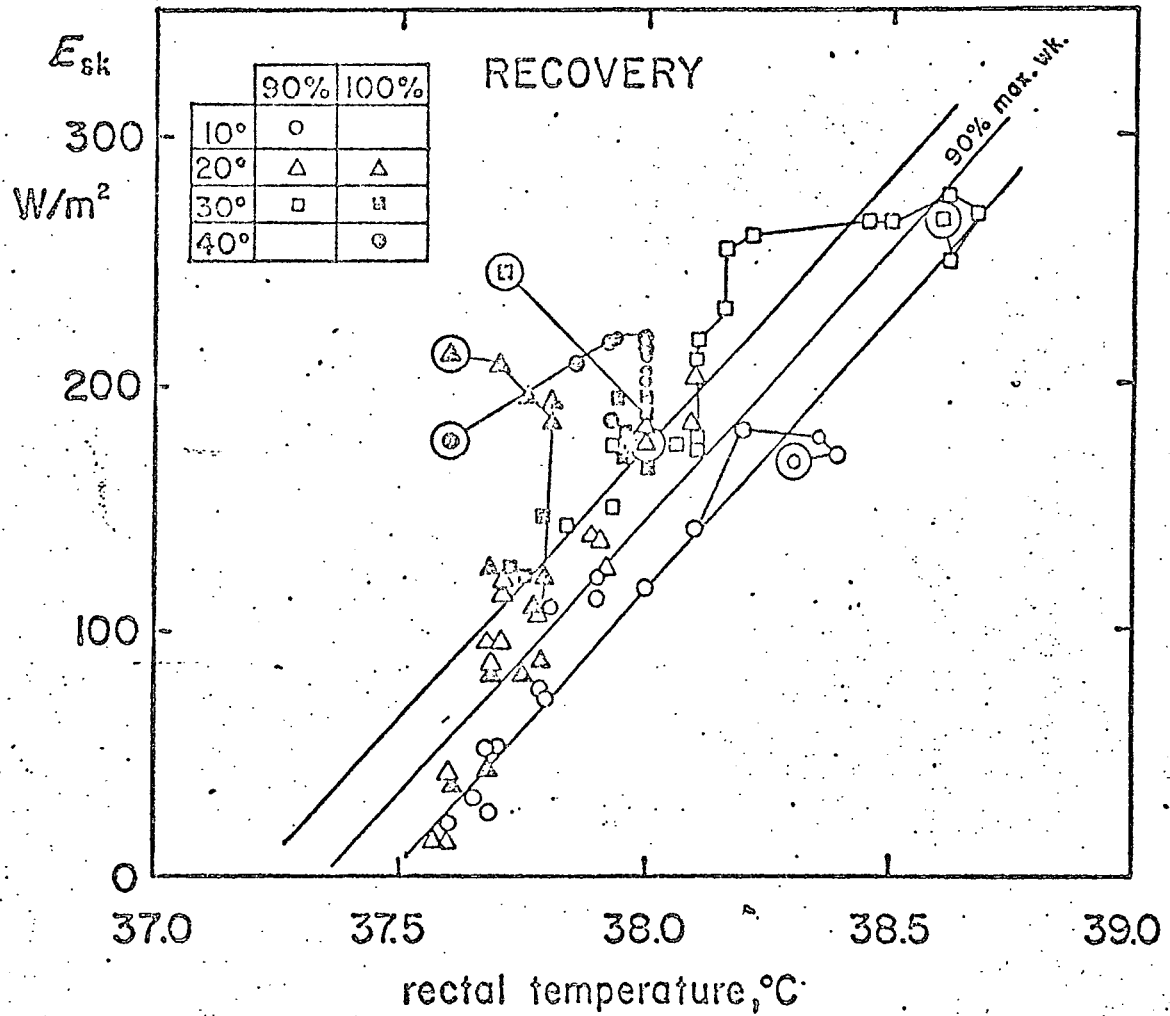at the start of recovery.

Fig. 9. The relation between skin evaporative loss $\underline{E}_{sk}$ and rectal tempera-
ture T_{re} during recovery. The regression line drawn for $\underline{E}_{sk} \sim T_{re}$
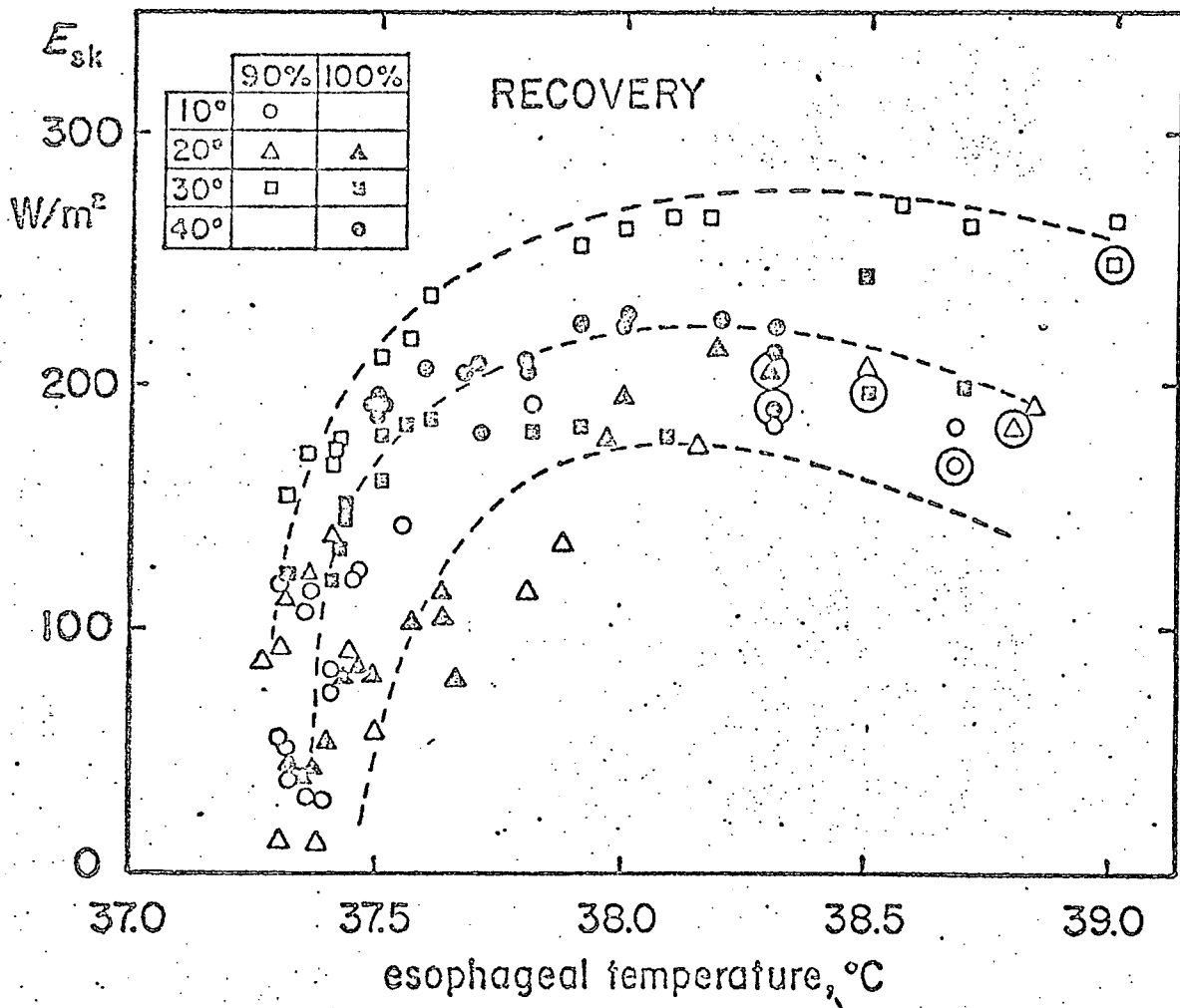represents only the 90% max. work data.

113<

Fig. 10. The relation between $\underline{E}_{sk}$ and esophageal temperature T_{es}. The median trend lines drawn have the statistical significance.

8,9,

During the recovery phase, shown in Fig. 8, 9 and 10, the relationships between E_{sk} and $\bar{T}_{sk}$, T_{re} and T_{es} are strikingly different in each case. In each plot the point for the start of recovery is circled and the trend sequence for the first few minutes of recovery is indicated by dotted lines. Again there was little change in $\bar{T}_{sk}$, while E_{sk} dropped from 250 W/m^2, although in each case $\bar{T}_{sk}$ rose approximately 1°C initially during the recovery period. For the 90% exercise data E_{sk} and T_{re} show the most consistent linear relationship over the whole recovery period. For the 100% exercise data, T_{re} continued to rise during the recovery period and after the 7th or 8th minute of recovery, the observed points joined the overall E_{sk} vs T_{re} trend observed for the 90% data. In contrast, the esophageal, T_{es}, cooled usually from 39°C down to 37.6°C before there was a sudden drop in E_{sk}. T_{es} did not significantly drop below 37.3°C during any 15 minute recovery period, regardless of the ambient temperature.

Statistical Analysis

In evaluating the significance of our data for the evaporative heat loss from the skin, E_{sk}, as it related to the four measured body temperatures $\bar{T}_{sk}$, T_{re}, T_{es} and T_m, we have looked at three key statistical indices – the Student T-test, the correlation coefficient R between individual variables and the square of the correlation coefficient (R^2). The T-test is defined as the ratio of the regression coefficient to its standard derivation and is an index of the significance of the chosen independent variables in affecting the dependent variable. R^2 is a measure of confidence for any regression equation and R^2 x 100 represents the approximate percentage of the observed data falling within the standard error of estimate (SEE) for the regression concerned.

Examination of Fig. 4 through 8 clearly shows that no significant correlation can be expected between E_{sk} and any one body temperature $(\bar{T}_{sk}$, T_{re}, T_{es} or $T_m)$, either during the exercise or recovery phases. The exception is a significant relationship between E_{sk} and T_{re} during the recovery phase for the 90% work data. This regression was found to be

$$E_{sk} = 222.2 \, (T_{re} - 37.35) \qquad \text{in W/m}^2 \qquad (6)$$

for which the Student T-test is 15; $R^2 = 0.82$; SEE = 34 W/m^2. Equation (6) has been plotted in Fig. 9. The inclusion of the 100% work data does not improve the correlation.

For the exercise phase during 90% work effort several multiple regressions between E_{sk} and body temperature are significant. They are summarized by the following equations in which an arbitrary value of 37°* has been used as a threshold for T_{re} or T_{es} in factoring the intercept of the regression equation. The calculated value of the $\bar{T}_{sk}$ threshold is thus free-floating. $\bar{E}_{sk}$ is given in W/m^2.

$$E_{sk} = 18.7 \, (\bar{T}_{sk} - 27.5) + 109.5 \, (T_{re} - 37. \, *) \qquad (7)$$

where $T = 11, 10$; $R^2 = 0.89$; SEE $= 24$.

$$E_{sk} = 19.1 \, (\bar{T}_{sk} - 28.1) + 78.7 \, (T_{es} - 37. \, *) \qquad (8)$$

where $T = 11, 10$; $R^2 = 0.87$; SEE $= 25$,

and

$$E_{sk} = 16.4 \, (\bar{T}_{sk} - 30.5) + 75.8 \, (T_m - 37. \, *) \qquad (9)$$

where $T = 10, 11$; $R^2 = 0.90$; SEE $= 22$.

For each regression at 90% work levels, the data for the first two minutes of exercise are omitted.

When the 100% work data are added the following significant regressions result:

$$E_{sk} = 20.1 \, (\bar{T}_{sk} - 26.8) + 99.5 \, (T_{re} - 37*) \qquad (10)$$

where $T = 13, 10$; $R^2 = 0.82$; SEE $= 26$.

$$E_{sk} = 19.8 \, (\bar{T}_{sk} - 27.4) + 70.2 \, (T_{es} - 37*) \qquad (11)$$

where $T = 13, 11$; $R^2 = 0.85$; SEE $= 25$,

and

$$E_{sk} = 15.3 \, (\bar{T}_{sk} - 29.3) + 64.5 \, (T_m - 37*) \qquad (12)$$

where $T = 9, 10$; $R^2 = 0.83$; SEE $= 26$.

Regression by eq. (10), (11) and (12) have been plotted on Figs. 5, 6 and 7 respectively for the levels of $\bar{T}_{sk}$ indicated.

During the recovery phase the only significant multiple regression occurred for $\bar{T}_{sk}$ and T_{re} at 90% work level and is given by

$$E_{sk} = 10.6 \, (\bar{T}_{sk} - 29.4) + 191.6 \, (T_{re} - 37.35), \qquad (13)$$

where $T = 5, 14$; $R^2 = 0.88$; SEE $= 28$.

In eq. (13), the threshold found statistically in eq. 6 above has been used. The confidence factor (R^2) for a multiple correlation between E_{sk} and $\bar{T}_{sk}$, T_{es} was only 0.64 and the regression is not listed.

A comparison of the above regressions for the exercise phase with those previously reported ([19]) for the submaximal level 25-75% shows that the same confidence levels occurred; here during the exercise phase the ratio of the regression coefficient for $\bar{T}_{sk}$ and T_{re} is 1:5; for $\bar{T}_{sk}$ and T_{es}, 1:3.5; and for $\bar{T}_{sk}$ and T_m, 1:4. In our early report the same three ratios were found to be 1:7.5, 1:10; and 1:4. In general our present regressions for maximal exercise show here a greater effect of $\bar{T}_{sk}$ in the control of sweating than at the lower submaximal level previously reported.
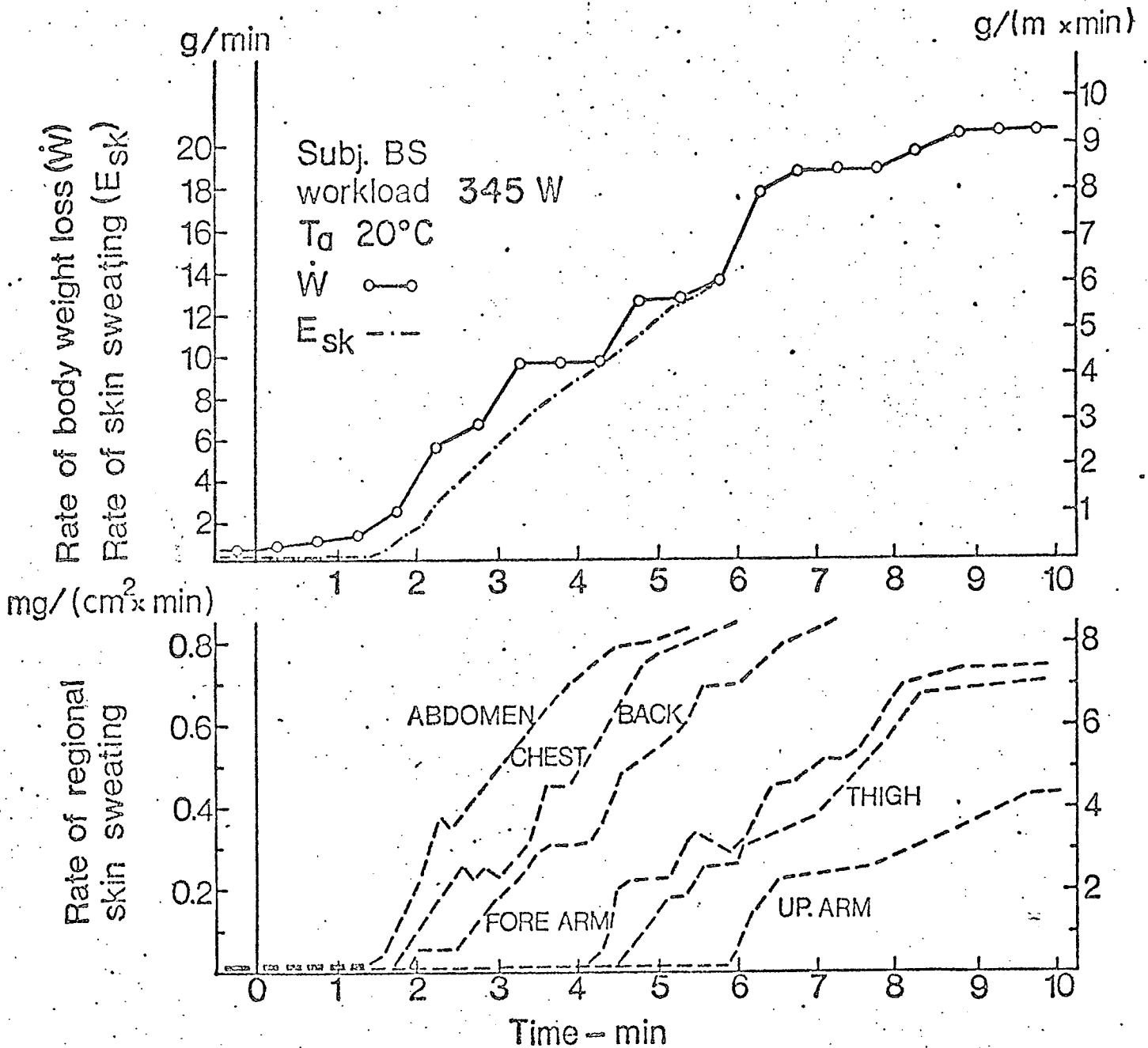
Fig. 11. Lower panel illustrates the rate of skin sweating over different areas of the body surface determined with resistance hygrometry (9). Upper panel gives the corresponding determinations of total body weight loss determined with the Potter Scale and calculated total skin sweating based on the data presented in the lower panel.

Discussion

At onset of very heavy exercise a time delay of at least 1 min. was observed before a significant increase in regulatory sweating occurred. This is in line with our previous results on submaximal exercise, where an increased rate of weight loss was first detected after 1.5 minutes of exercise. Due to the heavy work performed by the subjects in the present study there was usually a mechanical shift in the baseline of the weight record from the Potter scale going from rest to exercise, due to the sudden surge of exercise motion. If an increase in regulatory sweating had occurred during the first two minutes of exercise it would have been difficult to detect the true skin sweating with this technique. To clarify this question whether sweating started within the first minute of work, the resistance hygrometry method (2), as modified by Nadel et al (9), was used in special repeat runs on subj. BS while exercising at 90% maximal oxygen uptake. As can be seen in Fig.11 no increase in the rate of sweating could be detected from any area of the skin within the first minute of exercise. The cups indicate that the sweating, occurring over different areas of the body surface, started at different times after the exercise start and also varied in magnitude. When the area rates of sweating, as determined by the two methods, are expressed in comparable units $g/(m^2 \cdot min)$, the average of   cup readings weighted by area agreed well during the first minutes of the exercise with Potter Scale reading when corrected for E_{res} and $\dot{W}_{CO_2}$.

A question (19) we could not answer clearly before was whether the change in rate of weight loss ($\dot{w}$) corrected for respiratory vapor and CO_2 loss ($\dot{w}_{res}$) coincided with the start of sweat secretion ($\dot{S}$) or with the start of evaporative heat loss from the skin (E_{sk}). Since the cup method will always

indicate the presence of water or sweat on the skin surface, the present study shows that no sweat was found on the skin surface until after one minute of work. The exception happened when work is performed in the warm environment (40°C) where regulatory sweating was already present at rest before exercise. Even at 40°C a significant increase in E_{sk} did not occur until the second minute of work. Bullard (3) has recently suggested that the delay observed in skin sweating under different circumstances may partly be due to the filling of the duct of the gland with sweat and to its further transport to the skin surface. This may mean that sweat gland activity starts before any sweat is found on the skin surface. The time between the start of the sweat gland activity and when the first sweat may appear on the skin is only seconds in a neutral or warm condition (3). The present data, therefore, imply that there may be no drive for regulatory sweating immediately at onset of work. Further, the time delay (1-2 min) before onset of E_{sk} during exhaustive exercise seems to be more related to some internal temperatures of the body, since $\bar{T}_{sk}$ was constant during exercise at levels varying between 29° and 35°C.

The plots of E_{sk} vs the three major observable internal body temperatures ($\bar{T}_{sk}$, T_{re} and T_{es}) again show that both skin temperature and some internal body temperatures have a direct relationship on the control of sweating. Between the zero and third minute of exercise this relative rate of increase of E_{sk} with respect to either T_{es} or T_{re} is more a function of the air temperature T_a (and thus $\bar{T}_{sk}$). After this preliminary transitional period of maximal exercise, E_{sk} appears to be governed by a proportional control with both $\bar{T}_{sk}$ and T_{es} or T_{re} operating from some threshold.

Bullard (3) has shown the skin temperature may modify locally the drive for sweat secretion caused by an internal body temperature. Nadel et al. (9) have recently shown that such an effect would result in a relationship with the following form

$$E_{sk} = [\beta(\bar{T}_{sk} - 34) + \alpha(T_{es} - 36.5)]e^{(\bar{T}_{sk} - 34)/10} \tag{14}$$

Using our data for 90% maximum work at 10°C - 20°C - 30°C for a multiple regression between the ratio $E_{sk}/e^{(\bar{T}_{sk} - 34)/10}$ and $\bar{T}_{sk}$, T_{es}, the present data would show

$$E_{sk} = [6.9(\bar{T}_{sk} - 34^*) + 110.2 (\bar{T}_{es} - 36.4)]e^{(\bar{T}_{sk} - 34^*)/10^*}, \tag{15}$$

where the average SEE ∿ 27.

In eq. (15) the starred (*) values have been used to factor the intercept to derive a threshold value 36.4 for T_{es}. Our α/β ratio, i.e. 110/6.9, is lower than the 197/2 ratio reported by Nadel et al. This difference may be partially explained by the fact that for the current data body temperatures and sweat rates were rising steadily. The Nadel data, derived at a lower maximal work and after a longer exercise period, may represent a better "relative thermal equilibrium". By using our 90% data for the last 10 mins. of exercise when there may be a better equality between the rate of secretion (Ṡ) and the rate of weight loss ẇ, the predictive equation in the above format would be

$$E_{sk} = [115(T_{es} - 36.5)]e^{(\bar{T}_{sk} - 34.0)/10} \tag{16}$$

in which the β coefficient is negligible. For eq. (16) the standard deviation is 29. We do not consider our present data sufficiently precise to say which type function (eq. 8, 15 or 16) is a truer representation of sweating during these exhaustive exercises. For any case it is clear both $\bar{T}_{sk}$ and T_{es} have significant roles in the control of sweating.

During the recovery phase our data show that the falling esophageal temperature T_{es} has apparently little control over sweating until a temperature of approximately 37.5°C has been reached, after which there is a rapid drop in sweating. Even below 37.5°C T_{es} has little significant relationship, if any, with sweating. Rectal temperature appears to be the best single index of recovery sweating. This was specially for the 90% work load. If the first six minutes of the cooling curve for 100% work load were ignored, T_{re} below this level would also have been a good index of sweating for all temperatures over 10-40°C range. During recovery the threshold for cessation of sweating seems to be approximately 37.3°C for both T_{re} and T_{es}.

In an earlier study (19) we showed for a group of data covering three ranges of sub-maximal work (25, 50 and 75%) and for the three ambient temperatures (1) that $\bar{T}_{sk}$ was linear with ambient temperature and independent of metabolic rate; (2) that the internal body temperature (T_{es}) was linear with average metabolic rate or with the % $\dot{V}_{O_2}$ max; and (3) that E_{sk} was a linear function of both M and T_a. As seen in Figs. 1, 4 and 8 our present data confirm the first relationship. Only at the end of a 90% maximum work load the data for T_{es} (work time > 15) would agree with the second relation. By using all of our data for exercise for 10°-40°C, a confidence factor of only (R^2=0.64) could be obtained for a regression giving the third relationship.

Some reduction in the work performance was noticed when exercising in the warmest environments (T_a 30° and 40°C). This occurred without any significant reduction in the oxygen uptake. How this is brought about is unclear but a reduction in performance without a change in max $\dot{V}O_2$ has been observed before during similar conditions (15). At the end of exercise skin conductance varied between 45-55 W/m^2 at T_a of 10° and 20°C but was 60-70 W/m^2 when working at
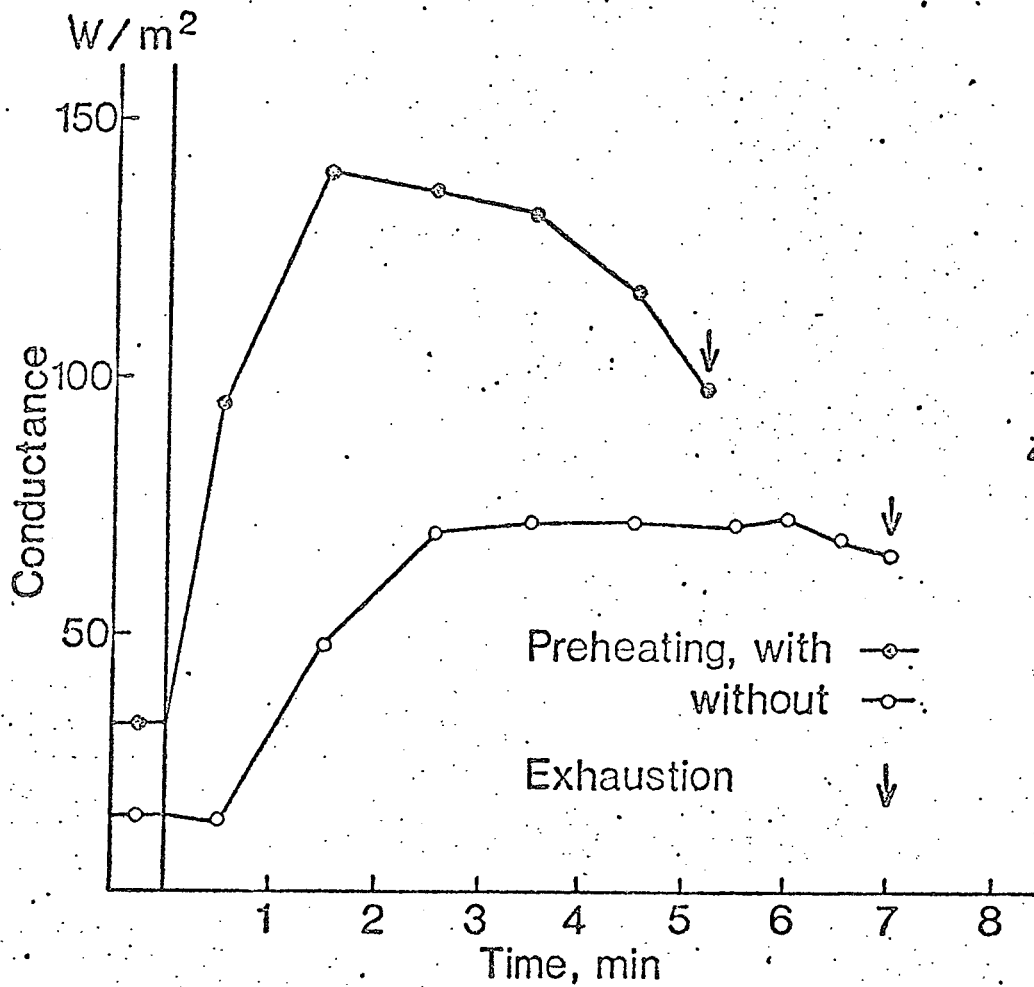
Fig. 12. Average skin conductances observed in subject UB and BS during maximal exercise at 40°C in two runs where one was preceded by warming up exercise.

ambient temperatures of 30° and 40°C. An increase in conductance means by definition an increase in skin blood flow. The situation thus may be as follows: when performing heavy exercise to exhaustion in a warm (as compared to a cool) environment the larger amount of the systemic flow distributed to the skin reduces the muscle blood flow (23) without any change in total cardiac output or significant change in $\bar{T}_{sk}$ (14). Anaerobic metabolites may then be accumulated faster in the exercising muscles in the warm environment causing the subject to stop earlier (7). As mentioned above, a reduction in max $\dot{V}O_2$ could not be observed just by exercising in a warm environment. A significant reduction in both maximal oxygen uptake and work performance was, however, observed when the subjects were preheated (15 min at 50% WL) and performed the maximal exercise at an ambient temperature of 40°C. The conductances observed during exercise in these latter situations were as high as 140 W/(m²·°C) but during the last minute of exercise went down to around 100 W/(m²·°C). This point is illustrated in

Fig. 12 Fig. 12 and should be compared with the peak value for conductance of 70 W/(m²·°C) reached at the end of the exercise when working in the same environment but without preheating. In the experiments with preheating only a small part of the large skin blood flow can be met by an increase in systemic cardiac output or reduced flow to organs such as the kidney and the liver. The main part must be taken from the muscles. Based on the observed differences in conductance the estimated difference in skin blood flow may be at least 2.5 l/min, which value agrees well with the observed reduction of 0.3-0.5 l/min in maximal oxygen uptake.

During submaximal exercise not leading to exhaustion within hours T_m, T_{es} and T_r reach a relative steady-state within 10-30 minutes of exercise (10, 11, 18, 20). In exhaustive exercise as used in the present study without warming-up these temperatures never attained a stable level. At 90% of max $\dot{V}O_2$ T_m and T_{es}

seemed, however, to be close to a leveling off at the end of 20 min exercise. Under all other conditions the subjects were exhausted before T_{re} and T_{es} reached very high temperature levels.

In conclusion when a subject starts exercise from a true resting state the heat stored in the body during the exhaustive exercise is not of sufficient magnitude to limit performance. On the other hand, muscle temperatures as high as $40.0° - 40.5°C$ were observed at the end of exercise. Since pre-heating of the subject did not result in significantly higher muscle temperatures at exhaustion, muscle temperature in range $40° - 40.5°C$ itself may be one limiting factor that can produce exhaustion during maximal exercise in humans.

References

1. Asmussen, E., and O. Bøje. Body temperature and capacity for work. Acta Physiol. Scand. 10:1-22, 1945.

2. Bullard, R. W. Continuous recording of sweat rate by resistance hygrometry. J. Appl. Physiol. 17:735-737, 1962.

3. Bullard, R. W. Studies on human sweat gland duct filling and skin hydration. J. Physiol. (Paris). In Press.

4. Fanger, P. O. Calculation of thermal comfort. ASHRAE Trans. 83:part II, 3.4.1-20, 1967.

5. Häggendahl, J., L. H. Hartley, and B. Saltin. Arterial noradrenaline concentration during exercise in relation to the relative work level. Scand. J. Clin. Lab. Invest. 26:337-342, 1970.

6. Hermansen, L., and B. Saltin. Oxygen uptake during treadmill and bicycle exercise. J. Appl. Physiol. 26:31-37, 1969.

7. Karlsson, J., and B. Saltin. Lactate, ATP and CP in working muscles during exhaustive exercise in man. J. Appl. Physiol. 29:598-602, 1970.

8. Klausen, K., D. B. Dill, E. E. Phillips, and D. McGregor. Metabolic reactions to work in the desert. J. Appl. Physiol. 22:292-296, 1967.

9. Nadel, E., R. W. Bullard, and J.A.J. Stolwijk. The importance of skin temperature in the regulation of sweating. J. Appl. Physiol. In Press.

10. Nielsen, M. Die Regulation der Körpertemperatur bei Muskelarbeit. Scand. Arch. Physiol. 79:193-230, 1938.

11. Nielsen, B. Thermoregulation in rest and exercise. Acta Physiol. Scand. Suppl. 323, 1969.

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

13. Pirnay, F., R. Deroanne, and J. M. Petit. Maximal oxygen consumption in a hot environment. J. Appl. Physiol. 28:642-645, 1970.

14. Rowell, L. B., J. A. Murray, G. L. Brengelmann, and K. K. Kraning. Human cardiovascular adjustments to rapid changes in skin temperature during exercise. Circulation Res. 24:711-724, 1969.

15. Saltin, B. Circulatory response to submaximal and maximal exercise after thermal dehydration. J. Appl. Physiol. 19:1125-1132, 1964.

16. Saltin, B. Circulatory adjustments and body temperature regulation during exercise. In: Physiological and Behavioral Temperature Regulation, edited by J. D. Hardy, et al., Springfield, Ill., Thomas, 1970, Chapter 23.

17. Saltin, B., and P-O Astrand. Maximal oxygen uptake in athletes. J. Appl. Physiol. 23:353-358, 1967.

18. Saltin, B., A. P. Gagge, and J. A. J. Stolwijk. Muscle temperature during submaximal exercise in man. J. Appl. Physiol. 25:679-688, 1968.

19. Saltin, B., A. P. Gagge, and J. A. J. Stolwijk. Body temperatures and sweating during thermal transients caused by exercise. J. Appl. Physiol. 28:318-327, 1970.

20. Saltin, B., and L. Hermansen. Esophageal, rectal and muscle temperature during exercise. J. Appl. Physiol. 21:1757-1762, 1966.

21. Stolwijk, J. A. J., B. Saltin, and A. P. Gagge. Physiological factors associated with sweating during exercise. J. Aerospace Med. 39:1101-1105, 1967.

22. Ström, G. The influence of anoxia on lactate utilization in man after prolonged muscular work. Acta Physiol. Scand. 17:440-448, 1949.

23. Williams, C. G., G. A. G. Bredell, C. H. Wyndham, N. B. Strydom, J. F. Morrison, J. Peter, P. W. Fleming, and J. S. Ward. Circulatory and metabolic reactions to work in heat. J. Appl. Physiol. 17:625-638, 1962.

Peripheral modifications to the central drive for sweating

ETHAN R. NADEL, JOHN W. MITCHELL, BENGT SALTIN, AND J. A. J. STOLWIJK

John B. Pierce Foundation Laboratory and Department of Epidemiology,
Yale University School of Medicine, New Haven, Connecticut 06519

NADEL, ETHAN R., JOHN W. MITCHELL, BENGT SALTIN, AND J. A. J. STOLWIJK. *Peripheral modifications to the central drive for sweating.* J. Appl. Physiol. 31(6): ▒▒▒–▒▒▒. 1971.—Three subjects each performed 15–20 bouts of 10-min bicycle ergometer exercise at 80% $\dot{V}o_{2\,max}$ in a 26° C ambient. This procedure imposed a consistent pattern of internal (esophageal) temperature (T_{es}) increase in the presence of a constant mean skin temperature ($\bar{T}_s$). Whole-body weight loss was continuously recorded and rate of evaporative loss due to sweating ($\dot{m}_{sw}$) was calculated during each minute of exercise. Local sweating rate was also continuously recorded from various skin areas and from the chest in the presence of local thermal clamps. It was confirmed that both local and total sweating are functions of internal temperature at a fixed $\bar{T}_s$. Each local skin area examined showed a characteristic T_{es} threshold above which sweat expulsion occurred and a characteristic proportional control constant. In most cases, an area which initiated sweating at a lower level of T_{es} also had a higher proportional control constant. Local heating of the skin decreased the T_{es} threshold for active sweating and increased the proportional control constant, and local cooling of the skin caused the opposite response. Thus, in the presence of a constant central drive for sweating, the sweating response could be modified at the periphery according to the area-specific characteristics and/or by local temperature. A point of zero central drive could be extrapolated from a conglomerate plot of local sweating rate against T_{es}, with both area and local temperature effects represented. Once the point of zero central drive was obtained, an approximation of the internal temperature set point could be predicted from a mathematical model.

sweating rate; evaporative weight loss; exercise; internal temperature; temperature regulation; mathematical model of regulation of sweating

---

RECENTLY WE DESCRIBED a conceptual model for the control of local sweating rate based on experiments in which the mean skin temperature of resting man was manipulated over a wide range (6). In this model the central drive for sweating was described as proportional to the sum of internal and mean skin temperature signals. This central drive was differentiated from the peripheral influence of local skin temperature, which was determined to be multiplicative with central drive in the prediction of local sweating rate. Observations that all skin areas over the body do not initiate sweating simultaneously and that different skin areas display different sweating rates in the presence of a given central sweating drive (4) indicate that area influences as well as local thermal influences affect the control system.

The present study was undertaken to examine these peripheral influences upon the central drive for sweating. A constant pattern of central drive increase was induced by exercise, and locational differences in local sweating rate as well as sweating modifications in the same area due to local temperature differences were studied. It was of interest to observe the pattern of recruitment of sweating from the initially nonsweating individual during exercise and to determine whether the model, which had been derived under resting conditions, could account for observations during the on-transient of exercise.

METHOD

Three male subjects, described in Table 1, each performed 15–20 bouts of 10-min bicycle ergometer exercise at a constant work load approximating 80 percent $\dot{V}o_{2\,max}$. Subjects were clothed only in shorts and athletic shoes. Ambient conditions in the environmental room were constant at 26° C, 40% rh, and minimal air movement. Experiments were performed at the same time of day and were always preceded by 20 min of rest to standardize the onset conditions in an attempt to minimize natural day-to-day physiological variations. The procedure of a brief bout of heavy exercise in a relatively neutral ambient was selected as a condition where sweating would be induced from a non-sweating individual within a relatively short time. Of the thermal variables shown to be important in the regulation of sweating, only the internal temperature would vary in this procedure. Thus, the initiation and changes in sweating rate could be related to the level of internal temperature in the present studies.

The bicycle ergometer was located on a Potter platform scale (see ref 11 for a more detailed description), with a 1-g sensitivity. A continuous record of body weight was obtained on a pen recorder while the subject was seated on the bicycle ergometer during rest and exercise. Minor

TABLE 1. *Anthropometric and exercise data for the subjects*

Subj	Age, yr	Ht, cm	Wt, kg	DuBois Body Surface Area, m²	$\dot{V}o_{2\,max}$, L/min STPD	Work Load, W	Exptl Work Load, % $\dot{V}o_{2\,max}$
JM	35	185	85	2.07	4.45	270	86
EN	29	170	82	1.95	4.00	245	80
BS	35	187	81	2.07	5.30	300	78

fluctuations in the weight record which resulted from pedaling during exercise were regular and were readily averaged over 1-min periods. The best smooth curve was drawn through the weight record of each experiment and rate of total weight loss ± 5% was then calculated for each minute of exercise by differentiation of the curve. The balance was calibrated prior to each experiment.

Oxygen consumption during the exercise was measured by an open-circuit technique with a Beckman paramagnetic oxygen analyzer. Rate of whole-body weight loss due to evaporation of sweat ($\dot{m}_{sw}$) was obtained by subtracting the rate of water loss from the respiratory tract and the excess weight of CO_2 over O_2 from the total weight loss. In separate experiments it has been shown that the respiratory quotient (R) rapidly approached a value of 1.0 or higher within the first 2 min of work at 90% $\dot{V}_{O_2 max}$. After the second minute of work there were no significant changes in R. During the present study where work around 80% $\dot{V}_{O_2 max}$ was performed, a steady-state R value of 0.96 was assumed throughout the work bout for the correction for CO_2 over O_2.

Respiratory water loss was measured directly in two of the subjects over a wide range of exercise conditions by humidity sensor and wet-dry bulb thermocouple sampling of the expired air. Fanger's (2) equation was confirmed for prediction of evaporation from the respiratory tract (E_{res}), as follows:

$$E_{res} = 0.0023 \dot{M} (44-P_a)$$

where M = metabolic rate (in W) and P_a = water vapor pressure in inspired air (in mm Hg).

Local sweating rate was recorded from a 12-cm² capsule on selected skin areas by a resistance hygrometry technique previously described (6). Humidity sensing elements were individually calibrated by pumping distilled water at known rates into the sealed capsules, and calibrations were periodically checked. Time lag of the system due to transport delay was 3 sec. Two sites of measurement of local sweating rate were utilized. In each experiment a capsule was placed on the right chest as a consistent reference. In separate experiments, local sweating rate was also recorded from the ventral thigh surface, abdomen, between the scapulae (back), volar surface of the forearm, lateral surface of the upper arm, and the left chest.

Since the local skin temperature under the capsule (T_{sl}) has been shown to exert a peripheral modifying influence upon local sweat gland activity (1, 6, 8), the local sweating rate from the contralateral chest was measured in separate experiments during which skin temperature under one capsule was clamped at different levels. Local thermal clamps were obtained by perfusing the upper layer of a two-layered copper sweat collection capsule with warm or cold water. Local skin temperatures beneath both capsules were continuously monitored by thermocouple during all experiments.

Each subject had internal temperature continuously recorded from a thermocouple in the esophagus at the level of the heart during three to five experiments. This yielded a standard T_{es} vs. time pattern which was used for the remainder of the experiments. Temperatures from 10 skin
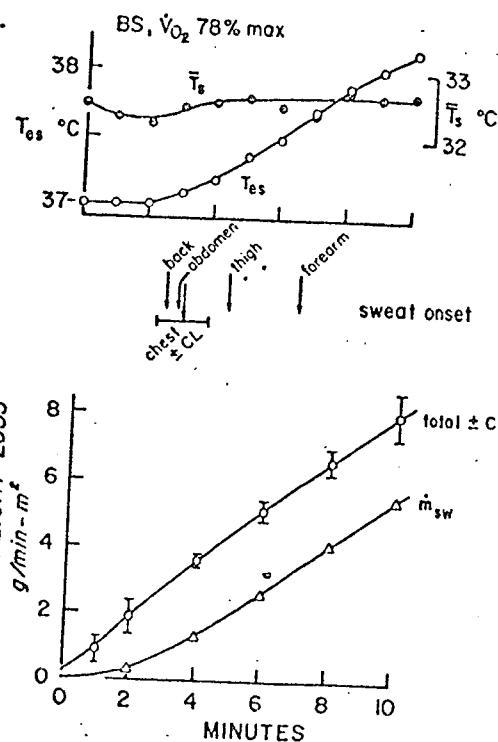

FIG. 1. Representative average thermal and weight loss (±95% confidence limits) data from one subject during 10 min of standard exercise. Time of onset of local sweating indicated by arrows.

surface loci were obtained once per minute throughout the exercise from thermocouple recordings and average skin temperature was calculated from a modified Hardy-Dubois formula as previously described (6).

RESULTS

Figure 1 illustrates the average thermal and whole-body weight loss data and times of onset of local sweating from different skin areas over the 10 min of standard exercise. Onset of local sweating for local areas from individual experiments and the average onset of sweat secretion from the chest (±95% confidence limits) from all experiments are shown. Esophageal temperature (T_{es}) began to increase after approximately 2 min of exercise and continued to increase throughout the duration at a relatively constant rate. The pattern of $\dot{m}_{sw}$ tended to follow that of T_{es} during the 10 min of exercise and local sweating rates from the different skin areas also tended to be related to T_{es} once sweating was initiated. Mean skin temperature ($\bar{T}_s$) was essentially constant during the exercise bout.

In Fig. 2 total and local sweating rates are plotted as a function of T_{es}. Each skin area displayed a characteristic T_{es} threshold above which sweat expulsion occurred and a characteristic proportional control constant. In most skin areas investigated, an area which initiated sweating at a lower level of T_{es} also had a higher proportional control constant (higher gain control).

Although the observation of a higher gain control associated with lower sweating threshold was consistent between subjects, the three subjects did not show inter-individual consistency in their hierarchies of sweat onset.
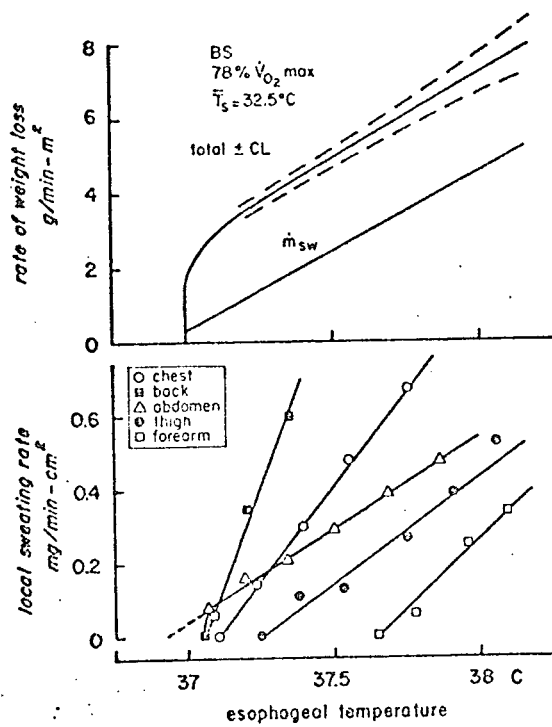
FIG. 2. Total (±95% confidence limits) and local sweating rates from one subject as a function of esophageal temperature during 10 min of standard exercise.

Subject JM began sweating at lower levels of T_{es} than the other subjects from nearly every skin area, with the thigh and abdomen the first areas from which active sweating was recorded. Under similar conditions, no active sweating was initiated from *subject BS* until his T_{es} was 37.10° C, which was 0.30° C higher than when sweating was initiated from *subject JM*. Sweating was first initiated from the back and abdomen in *subject BS*, in contrast to the pattern from *subject JM*. Subject EN showed a sweat onset-T_{es} pattern that was intermediate between the other two subjects, but sweating from his abdomen was late in appearance in comparison with that of *subjects JM* and *BS*.

Heating or cooling the skin under the sweat collection capsule modified the sweating response to the increased central drive associated with the heavy exercise bout. Figure 3 illustrates this effect of local skin temperature upon local chest sweating rate as a function of T_{es}. Heating the skin decreased the T_{es} threshold for active sweating and increased the gain of the sweating rate-T_{es} relationship. Conversely, cooling the skin increased the T_{es} threshold and decreased the gain.

DISCUSSION

The observations of Hertzman et al. (4) that the rates and pattern of recruitment of thermoregulatory sweating varied markedly over the body were confirmed in the present study. However, their report (4) of a caudal to rostral sequence in recruitment of sweating during rest which ascended the body as the thermoregulatory demands increased was not confirmed during the thermal stress of bicycle ergometer exercise. Recruitment of sweating from different areas in the present study was not entirely con-

sistent between individuals; there was a tendency for the back, chest, and abdomen to begin sweating sooner and at greater rates than the arms or legs, although *subject JM* showed the greatest sweating activity from the thigh. Differences between the thermal load imposed in Hertzman's study, which consisted of resting exposures to high ambient temperatures, and in the present study were considerable, suggesting that recruitment of sweating areas may vary according to the position of the subject (pressure effects can be considerable (5)), the activity level, or the method of inducing the increase in load.

The central drive for sweating has been previously described as a summation of thermal signals from the body core (brain temperature) and skin (mean skin temperature) as follows (6, 12):

$$\text{central drive} = \alpha(T_{es} - T_{es_0}) + \beta(\bar{T}_s - \bar{T}_{s_0}) \quad (1)$$

Where α and β are proportional control constants, the value of α is approximately 10 times that of β, and T_{es_0} and $\bar{T}_{s_0}$ are threshold temperatures. In the present series of experiments where $\bar{T}_s$ did not vary, changes in the central drive for sweating were directly related to changes in the internal temperature. Thus, the relationship between local sweating rate and T_{es} is analogous to that between sweating rate and central drive. The plot of $\dot{m}_{sw}$ vs. T_{es} (Fig. 2) also suggests the relation between $\dot{m}_{sw}$ and central drive, with the complication that evaporation in the transient may not be equivalent to sweat secretory rate because of storage of secreted sweat on the skin surface or dripping phenomena.
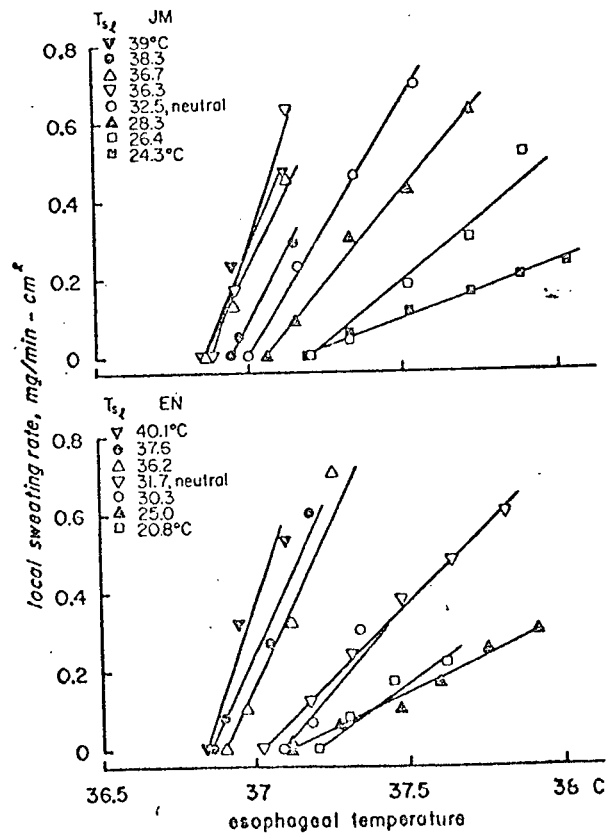

FIG. 3. Effect of local skin temperature on local chest sweating rate as a function of esophageal temperature. Data points taken at 1-min interval.

As shown in Fig. 2, each local skin area satisfied the requirements of the model described above, as each area increased its sweating rate linearly with increased central drive. Locational differences in the gain and onset temperature were attributed to local factors, such as the density of sweat glands under the capsule or the density and distribution of nerve branchlets in contact with the glands. The density of sweat glands may not be as important as the distribution of innervation, for there were no correlations between sweating rate and area density as reported by Szabó (13). Thus, each local skin area was seen to have a characteristic proportional control constant and internal temperature threshold above with active sweating occurred.

The relation between $\dot{m}_{sw}$ and T_{es} also satisfied the requirements of the model, as total evaporative water loss from sweating approximated a linear relationship to T_{es} over the range tested. This was less obvious in *subject JM*, whose $\dot{m}_{sw}$ approached E_{max} at a very rapid rate, resulting in the suppression of sweating rate by water standing on the skin (3). E_{max} was 15.5 g/min, calculated for bicycle ergometer exercise at 60 rpm under the present ambient conditions using data of Nishi and Gagge (7).

Nonthermal increases in local sweating rate that have been reported immediately at the onset of exercise (14) were not observed in the present conditions (Fig. 1), presumably because there was no ongoing sweating activity prior to exercise. In separate experiments in warmer ambients, this phenomenon has been documented in this laboratory. Most of the increase in rate of whole-body weight loss in the first minutes of exercise, prior to any significant increase in T_{es}, was the result of elevated E_{res} associated with the increment in ventilation and the excess weight loss of CO_2 produced over O_2 consumed, which also becomes increasingly significant during heavier exercise. Additional whole-body losses not directly accounted for by local sweating records in the first minutes of exercise may result from increased air velocity from pedaling causing increased evaporation of water lying on or near the skin surface (estimated to be 0.2 g/min-m²) and/or evaporation of sweat secreted from the axilla, face, or other areas not examined (estimated to be less than 0.8 g/min-m²).

The effect of local skin temperature (T_{sl}) has been described as having a peripheral influence which modifies the output from the central controller in the determination of local sweating rate (6). The peripheral (local skin temperature) influence was found to be multiplicative with central drive in the prediction of sweating as follows:

$$\text{local sweating rate} = (\text{central drive}) \, e^{(T_{sl}-\bar{T}_{s0})/\delta} \qquad (2)$$

where the value of δ was 9.1 (6). In the present series of experiments the peripheral influence of local skin temperature modifying the output from the central controller was confirmed as illustrated in Fig. 3, where the central drive is analogous to T_{es}.

By use of the complete model, *equation 2*, the value of δ could be determined from the data in Fig. 3 by taking the partial derivative of sweating rate with respect to T_{es} while T_s and T_{sl} were constant in the presence of different T_{sl} values. This relation is expressed as follows:
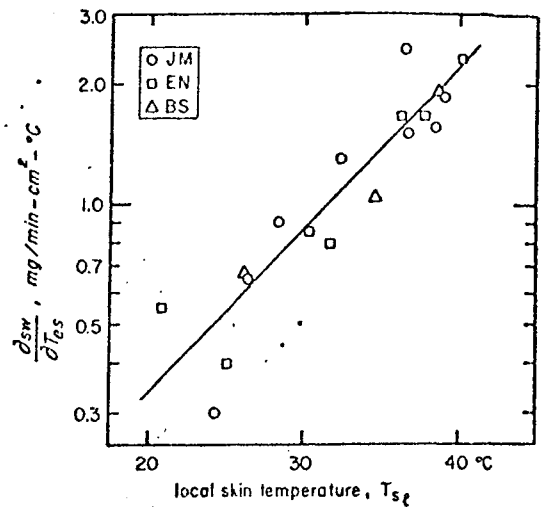

FIG. 4. Partial derivative of sweating rate with respect to T_{es} while T_s and T_{sl} were constant in the presence of different T_{sl} values.

$$\partial \text{sw}/\partial T_{es} = \alpha e^{(T_{sl}-\bar{T}_{s0})/\delta} \qquad (3)$$

This expression yields a linear relation between sw and T_{sl} when plotted on semilog coordinates, permitting the ready calculation of δ from the slope of the plot as seen in Fig. 4.

$$\ln(\partial \text{sw}/\partial T_{es}) = (\ln \alpha - \bar{T}_{s0}/\delta) + (1/\delta)(T_{sl}) \qquad (4)$$

The consistency among the three subjects over the range of local skin temperatures examined also supported the hypothesis (6, 8) that the local skin temperature effect upon local sweating rate was peripheral, exerting its influence at the neuroglandular junction where physicochemical laws tend to govern the reaction rate rather than within the central controller. The value of δ from the least-squares regression analysis of the pooled data was 10.7 ± 0.3 (95% confidence limits), a value similar to that previously determined from the tests in which T_{es} was held constant (6).

Although the mean value of α can be obtained from the data in Fig. 4, the confidence limits are very broad due to the fact that a small alteration in the slope between subjects can result in a relatively large shift in the intercept. The mean value of α for chest sweating rate was found to be 1.25 ± 0.38 (95% confidence limits) mg/min-cm²-°C; the potentially large differences in α between subjects indicate potentially significant interindividual variability in the central integrator.

Since active sweating can be observed in some areas but not in others at the same level of central drive for sweating, and since active sweating can be induced in any skin area at a specific level of central drive by local heating of the skin, it was apparent that a central drive for sweating can exist without the external secretion of sweat. Ogawa (9, 10) has described some characteristics of this subthreshold drive, including an apparently linear correlation between sweating rate and number of sweat expulsions per minute, where a forearm sweating rate of —0.07 mg/min-cm² was obtained by extrapolation to zero expulsions per minute.
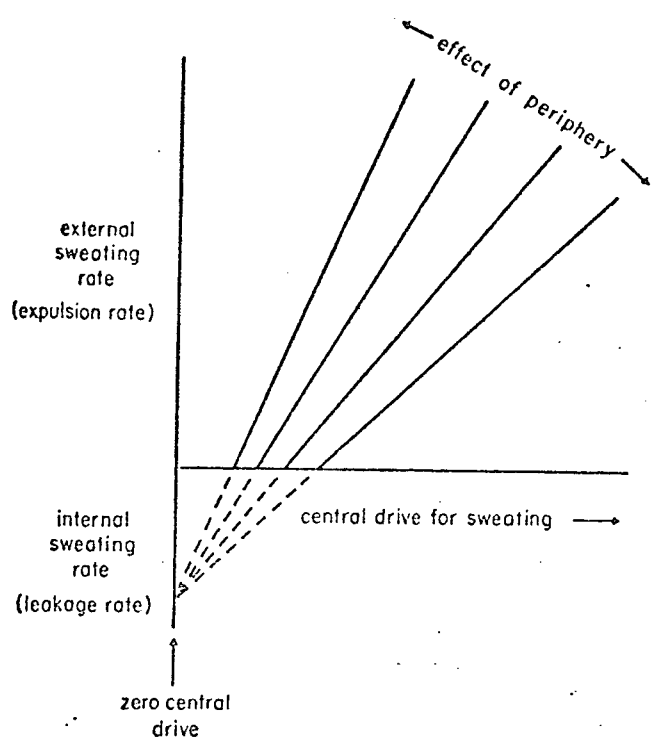
FIG. 5. Idealized model of total sweating rate (sum of internal and external sweating rates) as a function of central drive, illustrating effect of periphery modifying total rate.

In the present study the rate of external sweat secretion has been shown to be directly proportional to the central drive for sweating, with the threshold and gain of the sweating response a characteristic of the periphery (whether the peripheral effect is a local temperature effect or an area effect). With the assumption, as Ogawa and Bullard (10) have suggested that the rate of internal sweat secretion (sweat production by the glands without expulsion of sweat, where reabsorption or leakage > total secretion) is also proportional to the central drive for sweating, extrapolation of the linear sweating rate vs. central drive plots should be possible below the line of zero external sweating. Further-

more, since both area and local temperature effects upon the slope of the sweating rate-central drive relationship are peripherally mediated, the extrapolation of these plots should converge to a theoretical point at which central drive is zero, as illustrated in Fig. 5. From such a graph, one could predict 1) the true central temperature threshold for sweating at any level of mean skin temperature, and 2) the rate of sweat secretion necessary to achieve external sweating.

Figure 6 illustrates a conglomerate plot of local sweating rate against T_{es} for all three subjects, with both area and local temperature effects represented. In the case of each subject the theoretical point of zero central drive occurs at a discrete point, as given in Table 2.

It is theoretically possible to calculate the value of T_{es_0} in equation 1 for each subject, an approximation of the internal temperature set point. The assumptions made in this estimation are 1) $\delta = 10.7°$ C for each subject, 2) the value of β is approximately equal to 0.10 that of α (6), and 3) T_{s_0} for each subject = 34.0° C (6). Table 2 illustrates the values of these constants and the value of the internal temperature set point for sweating drive for each subject.

TABLE 2. Values of constants in model for chest sweating

Subj	At Zero Central Drive		α	δ	T_{es_0}, °C
	$\bar{T}_s$, °C	T_{es}, °C			
JM	32.0	36.75	1.30	10.7	36.55
EN	31.0	36.75	1.24	10.7	36.45
BS	32.5	36.90	1.18	10.7	36.75

Where α and δ obtained from equation 4: $\beta \simeq 0.1 \alpha$, $T_{s_0} = 34.0$, and T_{es_0} calculated by difference at zero central drive.

The point of zero central drive occurred at a sweating rate of -0.20 mg/min-cm^2 for each subject, as shown in Fig. 6. This rate of sweat secretion represents the internal leakage or reabsorption rate, or the rate of secretion by the sweat gland that must be surpassed in order to have external sweating. It cannot be determined from the data of the
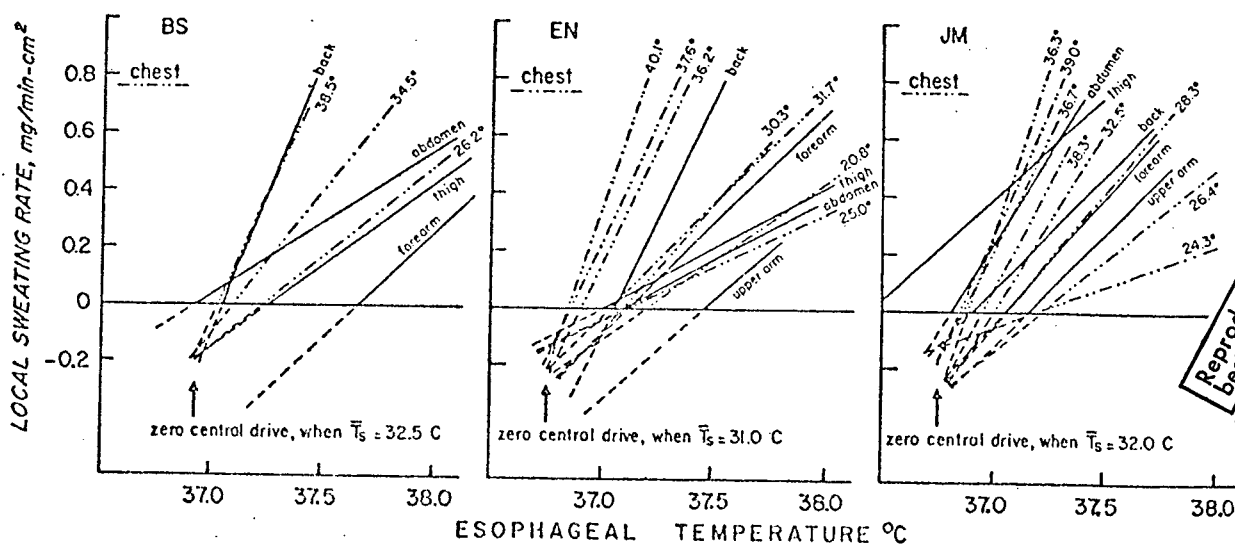
FIG. 6. Extrapolation to zero central drive from conglomerate plot of local sweating rate as a function of esophageal temperature, illustrating both area and local temperature effects on sweating rate.

present study whether glandular sweat secretory rates below 0.20 mg/min-cm² do not result in sweat expulsion from the ducts as a result of water reabsorption back into the duct or water leakage out of the duct, but the value of 0.20 mg/min-cm² may be looked upon as a constant leakage. This value is about one third that suggested by Ogawa and Bullard (10). By use of the data of Kuno (5) for the height of the duct (0.2 cm), diameter of the duct (30–60 μ), and density of sweat glands on the skin (0.006 cm² skin surrounding each duct), one can show that for a step change in glandular secretory rate from 0 to 0.20 mg/min-cm² sweating would not appear on the skin for 1.5 min if there were no leakage. If there were a constant leakage of 0.20 mg/min-cm², a step change in total secretion from 0 to 0.40 mg/min-cm² of internal plus external sweating would be necessary to achieve a comparable time lag, a lag on the order of those seen in the present study.

The mathematical model describing the regulation of sweating should be modified by the addition of a constant leakage term and a term accounting for locational differences (ϵ_l), as follows:

$$\text{local sweating rate} = \epsilon_l[\alpha(T_{es} - T_{es_0})$$
$$+ \beta(\bar{T}_s - \bar{T}_{s_0})]e^{(T_{sl}-\bar{T}_{s_0})/\delta} - \text{leakage} \quad (5)$$

A model that represents data from the present study appears as follows:

chest sweating rate (mg/min-cm²)

$$= [1.25(T_{es} - 36.60)$$
$$+ 0.12(\bar{T}_s - 34.0)]e^{(T_{sl}-34.0)/10.7} - 0.20 \quad (6)$$

The structure of the model determined from the present studies, using exercise to induce a change in load, was consistent with that determined from resting subjects. However, there are special cases which should be examined more thoroughly before the model can be completely accepted. It has yet to be established that the model is valid during all transients of exercise. The present results suggest that this is the case, but transients in a wider range of conditions would have to be examined for confirmation including off-transients. The nonthermal sweating observed at the onset of exercise in individuals already sweating (14) suggests a possible exception to the model in subjects in warm ambients or in preheated individuals. It further appears that the model should be valid during the steady state of positive work, but this would also have to be verified by experimentation over a wide range of exercise and ambient conditions.

The technique of measurement of local sweating rate in these further assessments of the model is a valuable tool for monitoring efferent activity, since estimates of sweating rate by measurement of rate of weight loss in the transient may be complicated by storage of water on the skin, adjustments in respiratory losses, or dripping. However, caution should be utilized in translating local sweating information to the whole body, because local E_{max} values are markedly increased under the sweat collection capsule. Establishment of the time relation between area-weighted sums of local sweat secretory rates and whole-body evaporative losses due to sweating during the transient of exercise would be an important challenge to the model.

REFERENCES

1. Bullard, R. W., M. R. Banerjee, and B. A. MacIntyre. The role of skin in negative feedback regulation of eccrine sweating. *Intern. J. Biometeorol.* 11: 93–104, 1967.
2. Fanger, P. O. *Thermal Comfort.* Copenhagen: Danish Technical Press, 1970.
3. Hertig, B. A., M. L. Riedesel, and H. S. Belding. Sweating in hot baths. *J. Appl. Physiol.* 16: 647–651, 1961.
4. Hertzman, A. B., W. C. Randall, C. N. Peiss, and R. Seckendorf. Regional rates of evaporation from the skin at various environmental temperatures. *J. Appl. Physiol.* 5: 153–161, 1953.
5. Kuno, Y. *Human Perspiration.* Springfield, Ill.: Thomas, 1956.
6. 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.
7. Nishi, Y., and A. P. Gagge. Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. *J. Appl. Physiol.* 29: 830–838, 1970.
8. Ogawa, T. Local effect of skin temperature on threshold concentration of suderific agents. *J. Appl. Physiol.* 28: 18–22, 1970.
9. Ogawa, T. Subthreshold sudomotor nerve impulses for generalized sweating in a cool environment. *Tohoku J. Exptl. Med.* 100: 255–261, 1970.
10. Ogawa, T. and R. W. Bullard. Sudomotor activity with and without generalized sweating. *J. Physiol., Paris* In press.
11. Saltin, B., A. P. Gagge, and J. A. J. Stolwijk. Body temperatures and sweating during thermal transients caused by exercise. *J. Appl. Physiol.* 28: 318–327, 1970.
12. Stolwijk, J. A. J., B. Saltin, and A. P. Gagge. Physiological factors associated with sweating during exercise. *Aerospace Med.* 39: 1101–1105, 1968.
13. Szabó, G. The number of eccrine sweat glands in human skin. In: *Advances in Biology of Skin*, edited by W. Montagna, R. A. Ellis, and A. F. Silver. New York: Pergamon, 1962, vol. 3.
14. Van Beaumont, W., and R. W. Bullard. Sweating: its rapid response to work. *Science* 141: 643–656, 1963.

MODIFICATION OF CENTRAL SWEATING DRIVE

AT THE PERIPHERY

J.A.J. Stolwijk[*], E.R. Nadel[*], J.W. Mitchell[**] and B. Saltin[***]

Please address editorial correspondence to:

Dr. J.A.J. Stolwijk
John B. Pierce Foundation Laboratory
290 Congress Avenue
New Haven, Connecticut 06519 U.S.A.

[*] John B. Pierce Foundation Laboratory and Department of Epidemiology
Yale University School of Medicine, New Haven, Connecticut 06510 U.S.A.

[**] Presently at Department of Mechanical Engineering, University of Wisconsin,
Madison, Wisconsin 53706 U.S.A.

[***] Presently at Gymnastik-och idrottshögskolan, Fysiologiska institutionen,
Lidingovagen 1, 11433 Stockholm, Sweden

ABSTRACT

Three subjects each performed 15-20 bouts of ten-minute bicycle ergometer exercise at 80 % $\dot{V}_{O_2}$ max in a 26°C environment. Esophageal and skin temperatures, total evaporative loss and local sweating rates over various 12 cm^2 areas of the body were continuously followed during the exercise transient. Sweating thresholds and patterns over the body and on the chest in the presence of different local skin temperatures were followed as functions of esophageal temperature (T_{es}). In most cases an area which initiated sweating at a lower level of T_{es} also had a higher proportional control constant. Local heating of the skin decreased the T_{es} threshold for active sweating and increased the proportional control constant, with local cooling of the skin eliciting the opposite response. Thus, the local sweating response to a constant increase in central drive for sweating could be modified at the periphery according to 1) the locality characteristics and 2) local temperature.

INTRODUCTION

Observations that all skin areas over the body do not begin sweating simultaneously in the presence of a given thermal load and that different skin areas exhibit different sweating activities under a constant load (Hertzman, Randall, Peiss, and Seckendorf, 1953) suggest that the central nervous system drive for sweating can be modified at the periphery according to area influences. It has also been demonstrated that local skin temperature can modify the local sweating rate in the presence of a constant central nervous system drive (Bullard, Banerjee and MacIntyre, 1967; Ogawa, 1970; Nadel, Bullard and Stolwijk, 1971). The present study was intended to describe the pattern of recruitment of sweating from an initially non-sweating individual during the first minutes of exercise. The study was further intended to examine the relationship between these peripheral influences (locality and local temperature) and the central drive for sweating upon local sweating rates over the body.

METHOD

Three male subjects each performed 15-20 bouts of 10-minute bicycle ergometer exercise at 80 % $\dot{V}_{O_2}$ max in an ambient of 26°C DB, 17°C WB, with minimal air movement. This procedure was selected as a condition which would induce sweating from a non-sweating individual within a brief period. Of the thermal variables shown to be important in the regulation of sweating, only the level of internal temperature would be altered by this procedure. Thus, the initiation and changes in sweating rate could be directly related to the level of internal temperature.

Local sweating rate was recorded in each experiment from a 12 cm^2 capsule on two selected skin areas by a resistance hygrometry technique previously described (Nadel, Bullard and Stolwijk, 1971). In each experiment a capsule was placed on the right chest as a consistent reference. In separate experiments local sweating rate was also recorded from the ventral thigh surface, abdomen, back, volar surface of the forearm, and the lateral surface of the upper arm. Local sweating rate from the contralateral chest was measured in additional experiments during which skin temperature under one capsule was clamped at different levels by perfusing the upper chamber of a two-chambered sweat collection capsule with warm or cold water. Local skin temperatures beneath both capsules were continuously recorded during all experiments.

The bicycle ergometer was located on a Potter platform scale (Saltin, Gagge and Stolwijk, 1970), which permitted continuous recording of body weight ±0.5 gram during rest and exercise. The best smooth curve was drawn through the

weight record of each experiment and the rate of total body weight loss was then calculated for each minute of exercise by differentiation of the curve. Rate of whole body weight loss due to evaporation of sweat ($\dot{m}_{sw}$) was calculated by subtracting the rate of water loss from the respiratory tract (Mitchell, Nadel and Stolwijk, 1971) and the excess weight of CO_2 expired over O_2 taken in, assuming $\underline{R}$ = 1 (Nadel, Mitchell, Saltin and Stolwijk, 1971) from the rate of whole body weight loss.

Each subject had internal temperature continuously recorded from a thermocouple in the esophagus at the level of the right atrium in three to five experiments, providing a standard T_{es} vs. time relationship. Temperatures from ten skin surface loci were obtained each minute of exercise from thermocouple recordings and mean skin temperature was calculated from a modified Hardy-Dubois equation (Nadel, Bullard and Stolwijk, 1971).

RESULTS

Esophageal temperature (T_{es}) began to increase after approximately two minutes of exercise and the rate of increase was relatively constant throughout the duration. The pattern of $\dot{m}_{sw}$ tended to follow that of T_{es} during the ten minutes of exercise. Local sweating was initiated at different times in the specific areas examined, with sweating on the back, chest, and abdomen tending to onset earlier than on the forearm and upper arm. Sweat secretion from the thigh was observed relatively early in two subjects and late in the third. Once sweating was initiated from a particular area, local sweating rate also tended to be

related to T_{es}. Mean skin temperature $(\overline{T}_s)$ was essentially constant during the exercise bout. Thus, with $\overline{T}_s$ constant the central sweating drive was entirely provided by the level of internal temperature.

When considered against T_{es}, each skin area was found to have a characteristic T_{es} threshold above which sweat expulsion occurred and a characteristic proportional control constant. In most areas investigated, an area which began sweating at a lower level of T_{es} also demonstrated a higher proportional control constant (higher gain).

Heating or cooling the skin under the sweat collection capsule modified the local sweating response to the increase in central drive. Heating a local area of skin on the chest decreased its T_{es} threshold for sweat expulsion and increased the gain of the sweating rate - T_{es} relationship. Conversely, cooling the skin resulted in an increase in its T_{es} threshold for sweating and a decrease in the proportional control constant.

DISCUSSION

The present study confirmed the observations of Hertzman, Randall, Peiss and Seckendorf (1953) that the rates and pattern of recruitment of thermoregulatory sweating varied significantly over the body. However, in exercising man the recruitment was not caudal to rostral, as they reported for resting man, and the pattern of recruitment in exercising man did show some inter-individual inconsistencies. It may be concluded that recruitment of sweating areas can vary according to the particular increase in load, the activity level or the position of the subject.

Each local skin area increased its sweating rate linearly with increased T_{es} once sweating was initiated. The central drive for sweating has previously been described as a summation of thermal inputs from the body core (brain temperature) and periphery ($\overline{T}_s$) as follows (Stolwijk, Saltin and Gagge, 1968; Nadel, Bullard and Stolwijk, 1971):

$$\text{central drive} = \alpha (T_{es} - T_{es_o}) + \beta (\overline{T}_s - \overline{T}_{s_o}) \qquad (1)$$

where α and β represent proportional control constants and T_{es_o} and $\overline{T}_{s_o}$ represent threshold temperatures. In the present series of experiments where $\overline{T}_s$ was relatively constant, the increases in the central drive for sweating were directly resultant from increases in T_{es}. Therefore, in the present study, T_{es} changes can be thought of as analogous to central drive changes. Thus, each local skin area satisfied the requirements of this model, as sweating was linearly related to central drive for sweating above a threshold level.

Each local skin area was observed to have its characteristic gain and T_{es} threshold above which active sweating occurred. Locational variability in gain and T_{es} threshold was attributed to the distribution of local nervous system innervation serving the glands rather than to density of sweat glands, as local sweating activity appeared to be unrelated to area density (Szabo, 1962).

The gain of the entire system was higher than for most of the local areas involved, suggesting that the sum of the sweat capsule (local) measurements of sweating rate might be greater than the whole body evaporative weight loss at any time. Since the local measurements involve evaporation of all water underneath the capsule, resulting in a dry skin, it is hypothesized that the effect of water

standing on the skin during whole body measurements tends to suppress sweating (Hertig, Riedesel and Belding, 1961). Preliminary evidence (Nadel, Mitchell and Stolwijk, 1971) obtained from experiments in which the evaporative heat transfer coefficient was increased two to three times tends to verify this concept, although quantification of this factor has not yet been realized.

The local skin temperature effect upon local sweating rate has been described multiplicative with central drive, as follows (Nadel, Bullard and Stolwijk, 1971):

$$\text{local sweating rate} = (\text{central drive})\, e^{(T_{s_\ell} - \overline{T}_{s_o})/\delta} \qquad (2)$$

The value of δ in the present study was determined by taking the partial derivative of chest sweating rate with respect to T_{es} while $\overline{T}_s$ and $\overline{T}_{s_\ell}$ were constant in the presence of different T_{s_ℓ} values. The value of δ from the least squares regression - analysis of the pooled data of the three subjects was found to be 10.7 $\pm$ 0.3 (95 % confidence limits). This value was comparable to the value of 9.1 previously determined from studies in which T_{es} was held constant and $\overline{T}_s$ manipulated (Nadel, Bullard and Stolwijk, 1971). The relatively low variability between subjects further supports the notion (Ogawa, 1970; Nadel, Bullard and Stolwijk, 1971) that the local temperature effect is peripheral rather than exerting its influence in some manner within the central controller.

Figure 1a demonstrates the peripheral effect of both local temperature upon chest sweating rate and the locality effect upon sweating rate in the presence of a constant central drive. It is apparent that 1) both peripheral effects are multiplicative with central drive and 2) a central drive for sweating can exist without the external secretion of sweat. The latter should be obvious since sweating can
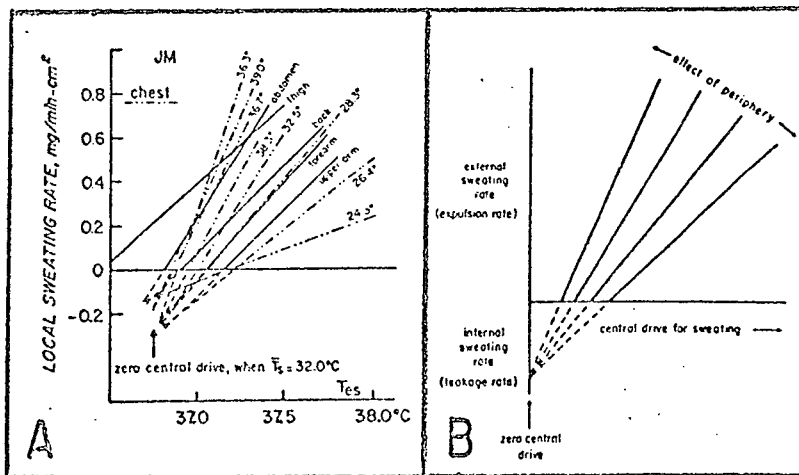
Figure 1a   Conglomerate plot of local sweating rate as a function of esophageal

temperature with $\bar{T}_s$ constant, illustrating both locality and local

temperature effects upon local sweating rate.

Figure 1b   Idealized model of local sweating rate (sum of internal and external

rates) as a function of central drive for sweating, showing modify-

ing effect of periphery (locality and/or local temperature effects).

be observed in certain areas prior to its appearance in others at the same intensity of central drive and since sweating can be induced from a non-sweating skin surface by heating of that area.

In Figure 1a the linear sweating rate - T_{es} relationships have been extrapolated to a hypothetical point of intersection, representing the locus of zero central drive. As shown in Figure 1b, the influences of the periphery, whether they are thermal or locational, can superimpose their effect upon any level of central drive resulting in coordinated shifts in both proportional control constant and threshold above which active sweating occurred. In the presence of a central drive for sweating without expulsion of sweat, the rate of reabsorption (or leakage) must exceed the rate of total secretion from the population of sweat glands. The extrapolation to zero central drive (Figure 1a) reveals that maximum reabsorption rates can be as great as 0.20 mg/min-cm^2, a value about three times that predicted by Ogawa and Bullard (1971) from their extrapolation of sweating rate vs. rate of sweat expulsions. However, the value of 0.20 mg/min-cm^2 is theoretically possible based upon computations of time lags for sweating to appear and the height, diameter and density of sweat ducts in the epidermis (Szabo, 1962).

In light of the above information, the model describing the regulation of rate of local sweat secretion should be expanded to include a reabsorption (or leakage) term, a locality term and a local wetted area term. The more complete model could then be represented as follows:

$$\dot{m}_{secr} = \emptyset_\ell \, \varepsilon_\ell \, A_\ell [\alpha(T_{es} - 36.5) + \beta(\overline{T}_s - 34.0)] e^{(\overline{T}_s - 34)/10.7} - \dot{m}_r \qquad (3)$$

where $\dot{m}_{secr} = \dot{m}_{sw} + \dot{m}_{storage}$

$$\emptyset_\ell = f \text{ (local wetted area)}$$

$$\varepsilon_\ell = F \text{ (locality)}$$

$$A_\ell = \text{area}$$

$$36.5 = \text{predicted } T_{es_0}$$

$$a = \text{approximately } 10\beta$$

$$\dot{m}_r = \text{rate of reabsorption (leakage)}$$

$$\overline{T}_s = \overline{T}_s - \gamma(d\overline{T}_s/dt - r_0)$$

In addition, acclimation effects must be considered as should the inter-action between the various effects in the control of sweating. It is apparent that certain factors must act in opposing directions, i.e. storage of sweat on the skin acting to reduce sweating rate, and this interaction could cause great difficulty in an attempt to isolate and quantify each of the factors.

REFERENCES

BULLARD, R.W., BANERJEE, M.R. and MACINTYRE, B.A. (1967): The role of the skin in negative feedback regulation of eccrine sweating. Int.J. Biometeorol., 11: 93-104.

HERTIG, B.A., RIEDESEL, M.L. and BELDING, H.S. (1961): Sweating in hot baths. J. Appl. Physiol., 16: 647-651.

HERTZMAN, A.B., RANDALL, W.C., PEISS, C.N. and SECKENDORF, R. (1953): Regional rates of evaporation from the skin at various environmental temperatures. J. Appl. Physiol., 5: 153-161.

MITCHELL, J.W., NADEL, E.R. and STOLWIJK, J.A.J. (1971): Respiratory weight losses during exercise. J. Appl. Physiol., submitted for publication.

NADEL, E.R., BULLARD, R.W. and STOLWIJK, J.A.J. (1971): Importance of skin temperature in the regulation of sweating. J. Appl. Physiol., 31(1).

NADEL, E.R., MITCHELL, J.W., SALTIN, B. and STOLWIJK, J.A.J. (1971): Peripheral modifications to the central drive for sweating. J. Appl. Physiol., submitted for publication.

NADEL, E.R., MITCHELL, J.W. and STOLWIJK, J.A.J. (1971): Control of local and total sweating during exercise transients. Int. J. Biometeorol., This proceeding.

OGAWA, T. (1970): Local effect of skin temperature on threshold concentration of sudorific agents. J. Appl. Physiol., 28: 18-22.

OGAWA, T. and BULLARD, R.W. (1971): Sudomotor activity with and without generalized sweating. J. Physiol. (Paris), In press.

SALTIN, B., GAGGE, A.P. and STOLWIJK, J.A.J. (1970): Body temperatures and sweating during thermal transients caused by exercise. J. Appl. Physiol., 28: 318-327.

STOLWIJK, J.A.J., SALTIN, B. and GAGGE, A.P. (1968): Physiological factors associated with sweating during exercise. Aerospace Med., 39: 1101-1105.

SZABÓ, G. (1962): The number of eccrine sweat glands in human skin. In: Advances in Biology of Skin, Volume 3. W. Montagna, R.A. Ellis and A.F. Silver (ed.) Pergamon, New York.

CONTROL OF LOCAL AND TOTAL SWEATING

DURING EXERCISE TRANSIENTS

E.R. Nadel[*], J.W. Mitchell[**] and J.A.J. Stolwijk[*]

[*] John B. Pierce Foundation Laboratory and Department of
Epidemiology, Yale University School of Medicine, New
Haven, Connecticut 06519 U.S.A.

[**] Presently at Department of Mechanical Engineering,
University of Wisconsin, Madison, Wisconsin 53706 U.S.A.

148<

Please address editorial correspondence to:

Dr. Ethan R. Nadel
John B. Pierce Foundation Laboratory
290 Congress Avenue
New Haven, Connecticut 06519 U.S.A.

ABSTRACT

Thermal and sweating parameters were continuously followed during the transient and steady state phases of three levels of bicycle ergometer exercise in 10, 20, 30 and 40°C ambients. A mathematical model describing the interrelations of the important thermal variables in the determination of local and whole body sweating rates was developed from the combined steady state information. The whole body model was shown to be inadequate when applied to the transient, however, being underpredictive in moderately heavy to heavy sweating situations. This was attributed to partial suppression of sweating rate by water standing on the skin during the steady state. It was concluded that there are several factors which interact during the steady state and therefore cannot be partitioned, but during the transient of exercise separation of these factors is possible. The model describing sweating rate was expanded to include these factors.

INTRODUCTION

It is generally agreed that during the steady state of exercise internal temperature is a function of relative workload (Saltin and Hermansen, 1966) and mean skin temperature is a function of ambient temperature (Nielsen and Nielsen, 1965; Stolwijk, Saltin and Gagge, 1968). Internal and mean skin temperatures have been implicated in the determination of the central drive for sweating in a recent proposal by Nadel, Bullard and Stolwijk (1971) in which the regulation of sweating rate is described by a summation model representing central drive modified by a local skin temperature factor in the following form:

$$\text{sweating rate} = [\alpha \, (T_{es} - 36.7) + \beta \, (\bar{T}_s - 34.0)] \, e^{(T_{s_\ell} - 34)/10} \tag{1}$$

The present study was designed to test this model during the transient of exercise in an attempt to better understand the interrelations between the various thermal inputs directing the regulation of sweating. During a change in load it was expected that the possible involvement of factors such as shifting set temperatures, non-thermal inputs, system lags or skin wettedness could be readily evaluated.

METHOD

Three healthy male subjects each performed 40-minute bicycle ergometer exercise bouts at work loads approximating 35, 50 and 65% $\dot{V}_{O_2}$ max in ambients of 10, 20 and 30°C and at 35 and 50% $\dot{V}_{O_2}$ max in an ambient of 40°C. Ambient water vapor pressure was constant around 10 mm Hg and air velocity was minimal. Subjects were clothed only in shorts and athletic shoes. Experiments were always

performed in the afternoon.

The bicycle ergometer was located on a Potter platform scale (Saltin, Gagge and Stolwijk, 1970) with a one gram sensitivity. A continuous record of body weight was obtained on a pen recorder while the subject was seated on the ergometer. The minor fluctuations in the record resulting from pedalling were regular and were readily averaged by fitting the best smooth curve through the record. Rate of total weight loss $\pm$ 0.5 g/min·m^2 was then calculated for rest and each minute of exercise by differentiation of the curve. Rate of total weight loss was corrected to rate of weight loss due to evaporation of sweat ($\dot{m}_{sw}$) by subtracting the rate of water loss from the respiratory tract and the excess weight of CO_2 expired over O_2 taken in (Mitchell, Nadel and Stolwijk, 1971).

Local sweating rate was continuously monitored in each experiment from 12 cm^2 ventilated capsules on the volar surface of the forearm and on the lateral surface of the upper arm, utilizing a resistance hygrometry technique previously described (Nadel, Bullard and Stolwijk, 1971). Humidity sensors were periodically calibrated by pumping distilled water at known rates into the sealed sweat collection capsules. Time lag of the system due to transport delay was three seconds.

Internal temperature was continuously monitored in each experiment from a thermocouple in the esophagus at the level of the right atrium. Average skin temperature was calculated each minute throughout each experiment from appropriate weighting of ten skin surface temperatures (Nadel, Bullard and Stolwijk, 1971). Since local skin temperature has been shown to exert an important influence on sweat gland activity (Bullard, Banerjee and MacIntyre, 1967; Ogawa, 1970; Nadel, Bullard and Stolwijk, 1971), local skin temperatures beneath both sweat collection capsules were continuously recorded during all experiments.

RESULTS

During the relatively steady state of exercise (30-40 minutes), esophageal temperature was observed to be independent of ambient temperature over the range 10-40°C and directly related to the relative workload according to the following relation:

$$T_{es} = 37.03 + 1.46 \ (\% \ \dot{V}_{O_2} max/100) \tag{2}$$

This relation was slightly lower than that reported by Saltin and Hermansen (1966), a fact which may, in part, be attributed to two of the subjects having been well acclimated to exercise.

Mean skin temperature during the steady state of exercise was related to ambient temperature and independent of internal temperature (and, therefore, workload). This observation was in accord with previous observations of Nielsen and Nielsen (1965) and Stolwijk, Saltin and Gagge (1968).

Energy balances computed during the steady state of exercise revealed that storage of heat was significant in all subjects only during the 50% $\dot{V}_{O_2}$ max exercise at 40°C. The combined heat transfer coefficient (h) during exercise was determined to be 9.5 W/m$^2 \cdot$°C, with the convective heat transfer coefficient (h_c) for bicycle ergometer exercise pedalling at 60 RPM then found to be 5.2 W/m$^2 \cdot$°C, a slightly lower value than Nishi and Gagge (1970) reported from naphthalene sublimation experiments.

The thermal and sweating data during the steady state when $\dot{m}_{sw} = \dot{m}_{secr}$, were fit to the model shown in eq. 1 by a two-parameter regression analysis, with the following results:

$$\dot{m}_{sw} \ (g/min \cdot m^2) = [4.82 \ (T_{es}-36.7) + 0.36 \ (\bar{T}_s-34.0)] \ e^{(\bar{T}_s-34)/10} \tag{3}$$
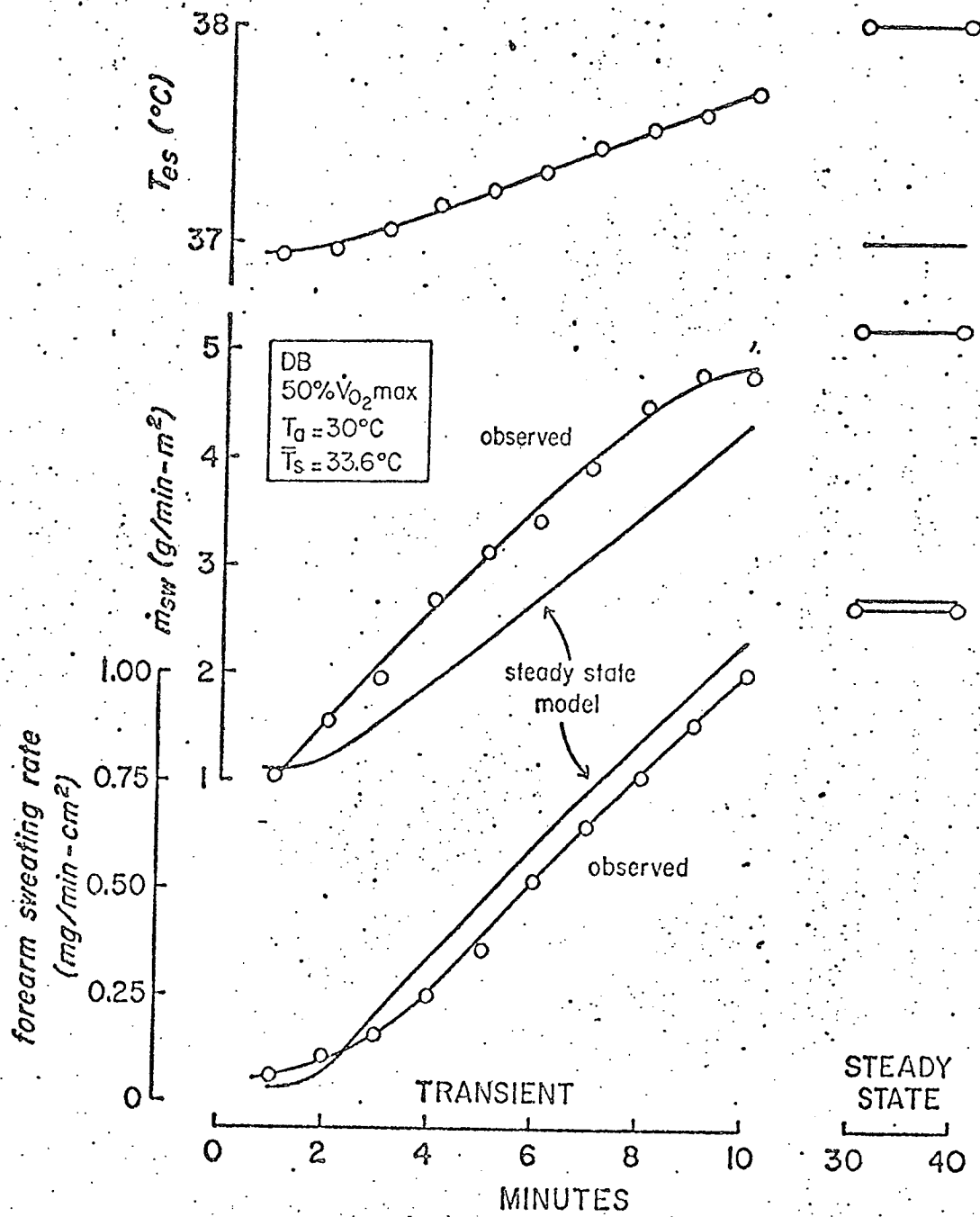
Figure 1. Observed and predicted whole body sweating rates ($\dot{m}_{sw}$) and forearm sweating rates during transient and steady state phases of moderate exercise in a 30°C ambient.

The values for $\dot{m}_{sw}$ in the present study were in close agreement to those previously calculated (Nadel, Bullard and Stolwijk, 1971) from the data of several studies. For the combined upper and lower arm local sweating rates, the best fit was as follows:

$$\text{arm sweating rate } (\text{mg/min} \cdot \text{cm}^2) = [1.46 \, (T_{es}-36.95) + 0.14 \, (\bar{T}_s-34.0)] \, e^{(T_{s_\ell}-34)/10} \qquad (4)$$

The above solutions obtained during the relative steady state of exercise were then compared to the observed data during the on-transient of exercise of each experiment. The ability of Eq. 3 to predict the observed $\dot{m}_{sw}$ was quite good in the 10 and 20°C ambients under all exercise conditions tested and also good during the 35% $\dot{V}_{O_2}$ max test in the 30 and 40°C ambients. However, the model was underpredictive in the warmer ambients or at heavier work conditions, indicating that the steady state model for prediction of $\dot{m}_{sw}$ was not wholly adequate during the transient.

On the other hand, local sweating rate from the arm could be adequately predicted during the transient of exercise from the model derived from the steady state data (Eq. 4). Figure 1 illustrates the closeness of fit between observed and predicted local sweating data from one subject working at 50% $\dot{V}_{O_2}$ max in a 30°C ambient. In this condition, the steady state model describing the control of $\dot{m}_{sw}$ proved to be underpredictive.

DISCUSSION

In the relative steady state of exercise, both whole body ($\dot{m}_{sw}$) and local (arm) sweating rates could be accurately described by a summation model, with the effect of local skin temperature multiplied by the summation term. In this model the internal temperature error signal multiplied by its proportional con-

trol constant plus a mean skin temperature error signal times its proportional control constant is envisioned as determining the central drive for sweating. The peripheral modifying (multiplicative) effect of local skin temperature has been shown to be an exponential with a Q_{10} of 2.7 to 3 (Nadel, Bullard and Stolwijk, 1971; Stolwijk, Nadel, Mitchell and Saltin, 1971).

The value of α, the internal temperature proportional control constant, was found to be approximately ten times that of β, the skin temperature proportional control constant, in the description of both whole body and local sweating rates. This supported the concept that deviations in internal temperature are about ten times as important as mean skin temperature changes in the determination of the central sweating drive.

The gain constants found in the model describing the control of local sweating rate for the arm when extended to the whole body were on the order of three times those found in the whole body model. Since the arm is characteristically lower in its sweating capacity than much of the rest of the body (Stolwijk, Nadel, Mitchell and Saltin, 1971), it would appear that hygrometric measurements of local rate of sweat secretion are not directly transferable to the whole body. The hygrometric technique requires evaporation of all water beneath the capsule, resulting in a dry skin. Thus, it was postulated that the effect of water standing on the skin was to suppress the rate of sweat secretion, as intimated by Hertig, Riedesel and Belding (1961). This concept was confirmed as follows:

One of the subjects exercised at 55% $\dot{V}_{O_2}$ max in an ambient of 34°C DB, 24°C WB. At the end of 30 minutes, after a relative steady state in thermal and sweating rate parameters had been achieved, a large fan was directed onto the subject, increasing the evaporative heat transfer coefficient by a factor of two
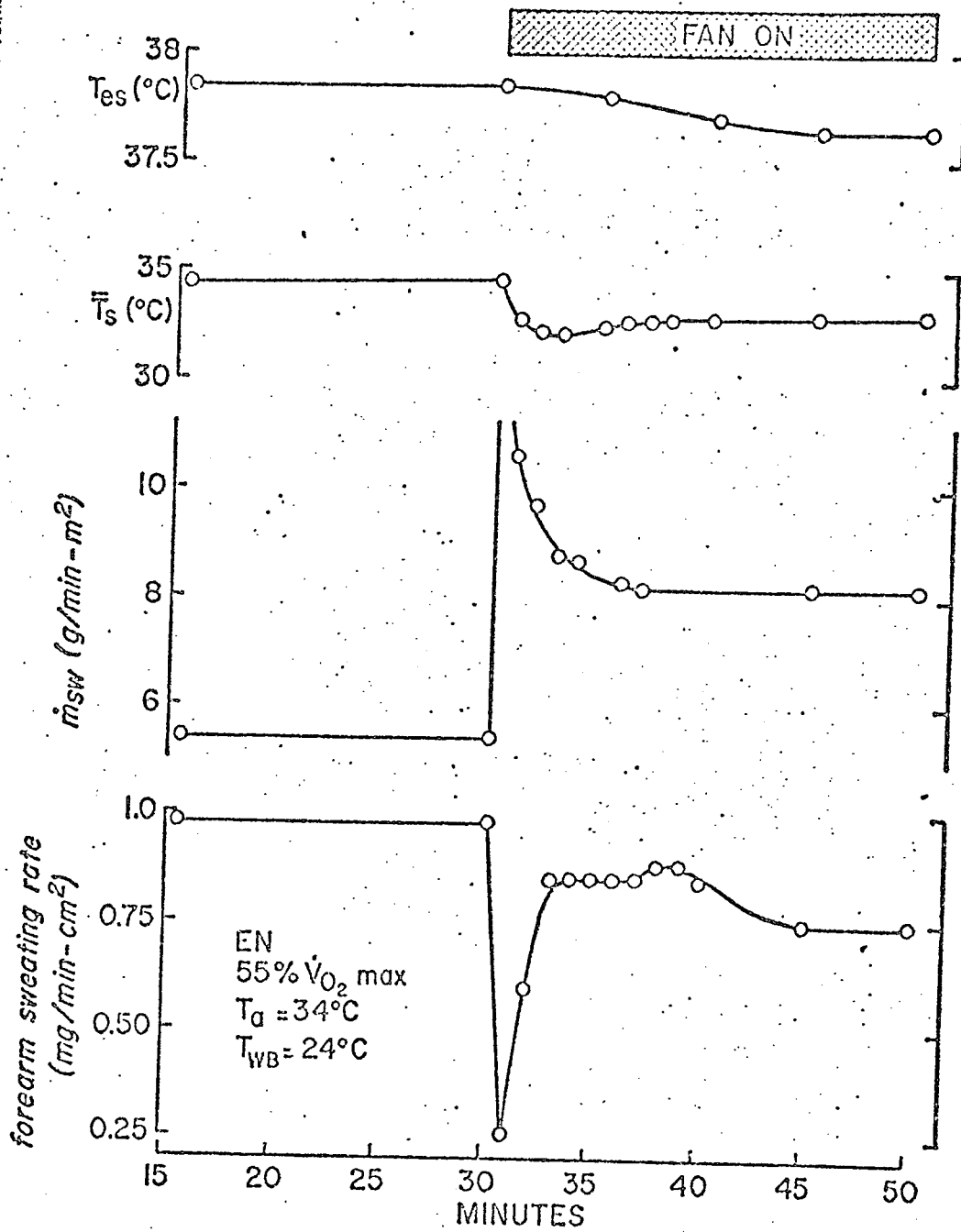
Figure 2. Example of drier skin resulting in increased whole body sweating rate despite lowered central sweating drive.

to three and decreasing the skin wettedness from 72% to between 30 and 40%. As shown in Fig. 2, the subject attained a new steady state in 15-20 minutes after the transient response to the increase in air velocity. In the new steady state, T_{es} had dropped from 37.85 to 37.65°C and $\bar{T}_s$ had dropped from 34.5 to 33.0°C. Local sweating rate from the forearm, reflecting the decrease in central drive, also dropped from 0.98 to 0.75 mg/min·cm^2. However, in the presence of the drier skin and despite the lower central sweating drive, $\dot{m}_{sw}$ increased from 5.6 to 8.0 g/min·m^2. Thus, it was clear that the water standing on the skin prior to exposure to the fan had suppressed sweating rate over the body.

This observation explains why the model was more adequate during lower workloads in cooler ambients. These conditions do not place as great a drive on the sweating system and the skin wettedness remained at relatively low levels. In warmer ambients or during heavier workloads, observed steady state sweating rates were the result of partial suppression by water standing on the skin. During the exercise transient before a high level of skin wettedness had been reached, sweating rate was not yet suppressed and therefore temporarily exceeded the rates predicted from the steady state model.

It has become apparent that there are several factors which interact in the regulation of sweating rate during the steady state and are therefore indistinguishable. Only during a transient can these factors be separated and evaluated. In consideration of the evidence from the comparison of local and whole body models and the fan test, a more complete model of the regulation of whole body sweating rate appears as follows:

$$\dot{m}_{secr} = \phi\psi \left[\alpha' \left(T_{es} - 36.7 \right) + \beta' \left(\bar{T}_s - 34.0 \right) \right] e^{(\bar{T}_s - 34)/10} + \dot{m}_{nt} \tag{5}$$

where

$$\dot{m}_{secr} = \dot{m}_{sw} + \dot{m}_{storage}$$

$\dot{m}_{storage}$ = rate of accumulation of water on the skin

ϕ = f (wetted area)

ψ = f (acclimation)

α' and β' may be as much as three times the values of α and β found during the steady with wetted skin (Eq. 3)

$\dot{m}_{nt}$ = f (non-thermal inputs, e.g. sympathetic nervous system activity)

The values of ϕ should have an inverse function with skin wettedness and range from around 0.2 in an extremely wet situation to 1.0 in a totally dry situation. It is postulated that the acclimation factor ranges from 1 to perhaps 3, a value representative of a highly acclimated individual. It is clear, however, that the regulatory system for sweating is more sensitive than had been previously assessed, and factors associated with ambient conditions are those that limit the efficiency of this system. In fact, many of the older concepts of the regulation of body temperature during exercise may have to be re-examined under a greater variety of ambient conditions to determine the actual sensitivity of the sweat regulatory mechanism and the extent to which it is limited by the environment. It is also clear that skin wettedness, degree of heat acclimation and skin temperature all need to be accounted for in studies of the central drive.

REFERENCES

BULLARD, R.W., BANERJEE, M.R. and MACINTYRE, B.A. (1967): The role of the skin in negative feedback regulation of eccrine sweating. Int. J. Biometeorol., 11: 93-104.

HERTIG, B.A., RIEDESEL, M.L. and BELDING, H.S. (1961): Sweating in hot baths. J. Appl. Physiol., 16: 647-651.

MITCHELL, J.W., NADEL, E.R., and STOLWIJK, J.A.J. (1971): Respiratory weight losses during exercise. J. Appl. Physiol., submitted for publication.

NADEL, E.R., BULLARD, R.W. and STOLWIJK, J.A.J. (1971): Importance of skin temperature in the regulation of sweating. J. Appl. Physiol., 31(1).

NIELSEN, B. and NIELSEN, M. (1965): On the regulation of sweat secretion in exercise. Acta Physiol. Scand. 64: 314-322.

NISHI, Y. and GAGGE, A.P. (1970): Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. J. Appl. Physiol. 29: 830-838.

OGAWA, T. (1970): Local effect of skin temperature on threshold concentration of sudorific agents. J. Appl. Physiol., 28: 18-22.

SALTIN, B., GAGGE, A.P. and STOLWIJK, J.A.J. (1970): Body temperature and sweating during thermal transients caused by exercise. J. Appl. Physiol., 28: 318-327.

SALTIN, B. and HERMANSEN, L. (1966): Esophageal, rectal and muscle temperature during exercise. J. Appl. Physiol., 21: 1757-1762.

STOLWIJK, J.A.J., NADEL, E.R., MITCHELL, J.W. and SALTIN, B. (1971): Modification of central sweating drive at the periphery. Int. J. Biometeorol., This proceeding.

Respiratory Weight Losses During Exercise

John W. Mitchell

Ethan R. Nadel

Jan A. J. Stolwijk

John B. Pierce Foundation Laboratory
Department of Epidemiology
Yale University School of Medicine
New Haven, Connecticut 06519

Please address editorial correspondence to:

Dr. John W. Mitchell
John B. Pierce Foundation Laboratory
290 Congress Avenue
New Haven, Connecticut 06519

Running Head: Respiratory weight losses during exercise

Abstract

Evaporative water loss from the respiratory tract was determined over a wide range of exercise. The humidity of the expired air was the same at all levels of exercise and equal to that measured at rest. The rate of respiratory water loss during exercise was found to be consistent with the following published relationship based upon data obtained during resting conditions

$$\dot{m}_e = 0.019 \ \dot{V}_{O_2} \ (44 - p_a)$$

where $\dot{m}_e$ is the rate of evaporative water loss (g/min), $\dot{V}_{O_2}$ is the oxygen up-take (L/min STPD), and p_a is water vapor pressure (mm Hg). The rate of weight loss during exercise due to $CO_2 - O_2$ exchange was calculated. For exercise at oxygen consumption rates exceeding 1.5 L/min in a dry environment with a water vapor pressure of 10 mm Hg, the total rate of weight loss via the respiratory tract is on the order of 2 to 5 g/min.

In many studies concerned with the regulation of sweating, evaporative losses attributed to sweating have been estimated by intermittent whole body weighing. Although it has been acknowledged that whole body evaporative weight losses only approximate sweating losses, there have been no determinations made of the respiratory weight losses during exercise. These terms could become increasingly significant during exercise when marked increases in respiratory activities occur.

Fanger (2) has proposed an equation for estimating respiratory water loss during exercise. It is based on the experimental results obtained by McCutchan and Taylor (4) for the humidity of expired air in resting subjects as functions of inspired air temperature and humidity over wide ranges. The change in humidity was found to be given by

$$w_{ex} - w_{in} = 0.029 - 0.8 \, w_{in} \tag{1}$$

where w_{ex} and w_{in} are the absolute humidities of expired and inspired air in grams of water per gram of air. Fanger (2) combined Eq. 1 with a relation between ventilatory exchange and oxygen uptake during rest and exercise to derive the following equation for predicting respiratory heat loss (E_{res})

$$E_{res} = 0.0023 \, M \, (44 - p_a) \tag{2}$$

where M is the metabolic rate (W) and p_a is the ambient water vapor pressure (mm Hg). The corresponding rate of water loss is given by

$$\dot{m}_e = 0.019 \, \dot{V}_{O_2} \, (44 - p_a) \tag{3}$$

where $\dot{m}_e$ is the rate of evaporative water loss in the expired air (g/min) and $\dot{V}_{O_2}$ is the oxygen uptake (L/min STPD).

The respiratory weight loss given by Eq. 3 is based on humidity data obtained from resting subjects, yet it has been used extensively to account for respiratory

water loss during exercise. There are major alterations in ventilation during exercise, and there are no physical nor physiological reasons why Eq. 1 would then be applicable. The present study was undertaken to determine the validity of these equations under exercise conditions. An additional purpose of this study was to evaluate the significance of weight loss resulting from excess CO_2 eliminated over O_2 taken in during exercise in relation to total body weight loss.

Methods

Respiratory water loss was determined from four subjects (Table 1) during steady state bicycle exercise in four environments. The work rates were approximately 30, 50, 65, and 80 percent of maximum oxygen consumption. Ambient conditions were 20, 26, 30, and 37°C with a water vapor pressure between 7 and 10 mm Hg. Low humidity environments were selected to maximize the humidity changes during breathing and to accentuate any differences between resting and exercise values.

In the 37°C environment, the inspired and expired air humidities were measured by two methods. The first employed standard wet and dry bulb thermocouples placed in the expired air stream 0.5 meters downstream from the Collins valve. In the second method, 500 ml/min of expired air was drawn through a resistance hygrometry circuit (5), and the humidity determined directly. Ventilatory volume was measured by a balanced spirometer.

In the 20, 26, and 30°C environments, the ambient air temperature is below the dew point of the expired air, and condensation in the expired air lines would invalidate the humidity readings obtained by either of the two methods. For these tests, the expired air was diluted with a high flow rate of ambient air. Flow rate and wet and dry bulb temperatures of the air stream were measured. The relative humidity of the expired air could not be determined from these

Table 1

Physical characteristics of the subjects

	Age, yr	Height, cm	Weight, kg	Dubois body surface area, m^2	$\dot{V}_{O_2}$ max, L/min STPD
RG	28	174	68	1.82	3.0
MK	24	188	84	2.10	4.1
JM	36	185	85	2.07	4.5
EN	29	170	80	1.92	4.2

measurements alone, but the total evaporative water loss could be directly calculate

In these conditions the air flow was drawn through a Beckman paramagnetic oxygen

analyzer for determination of oxygen uptake.

Results

The humidity of the expired air in the 37°C environment is shown in Fig. 1.
The water content of the expired air was essentially constant over the entire
work range and equal to the resting value given by Eq. 1. Also shown for com-
parison is the water content for air saturated at body temperature. Since the
expired air conditions are 35°C and 80% rh, it is apparent that the expired air
does not become saturated even during heavy exercise.

The rate of water loss from the respiratory tract is presented in Fig. 2.
The results for all four environments were obtained directly from the measured
values of the change in humidity and flow rate. With the exception of the high
work load test for RG, these data were within 0.25 g/min of the relationship
proposed by Fanger (Eq. 3) over the entire exercise range. Since the ambient
water vapor pressure varied only slightly (7 - 10 mm Hg) for all test conditions,
there is basically only one relationship from Eq. 3 as shown on Fig. 2.

Discussion

McCutchan and Taylor (4) found that over the range of 20 to 90°C and 0 to
100% rh the expired air is always unsaturated for an inspired air humidity less
than 100%. For inspired temperatures less than about 55°C, the expired air
temperature is less than body temperature. The mechanism which prevents satura-
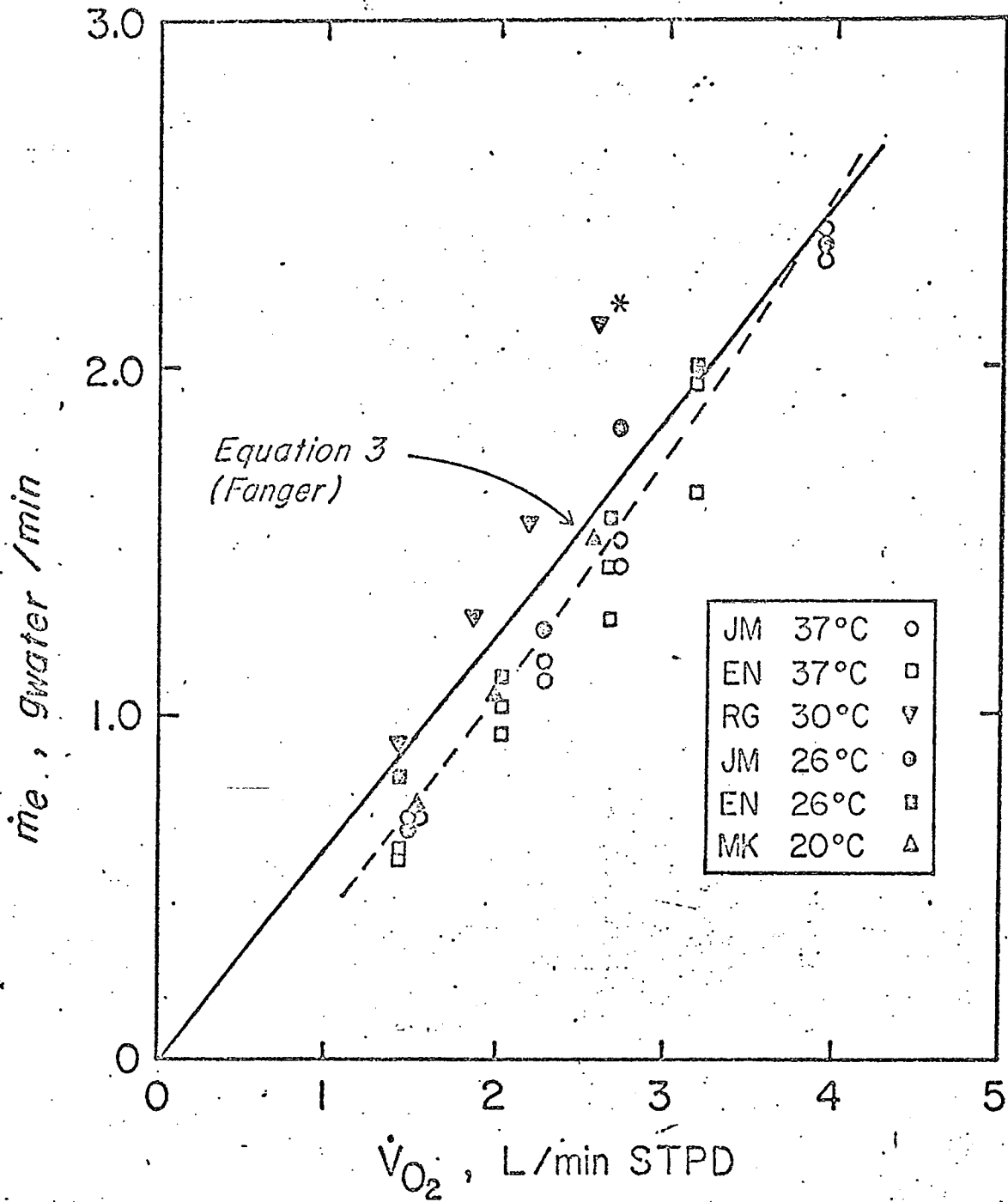tion of the expired air is the cooling by evaporation of the nasal and oral

Figure 2: Evaporative water loss ($\dot{m}_e$) from the respiratory tract as a
function of oxygen consumption ($\dot{V}_{O_2}$) during exercise.
Starred point (RG) was taken at 90 percent $\dot{V}_{O_2}$ max, where
ventilation – $\dot{V}_{O_2}$ ratio was significantly elevated (see text).
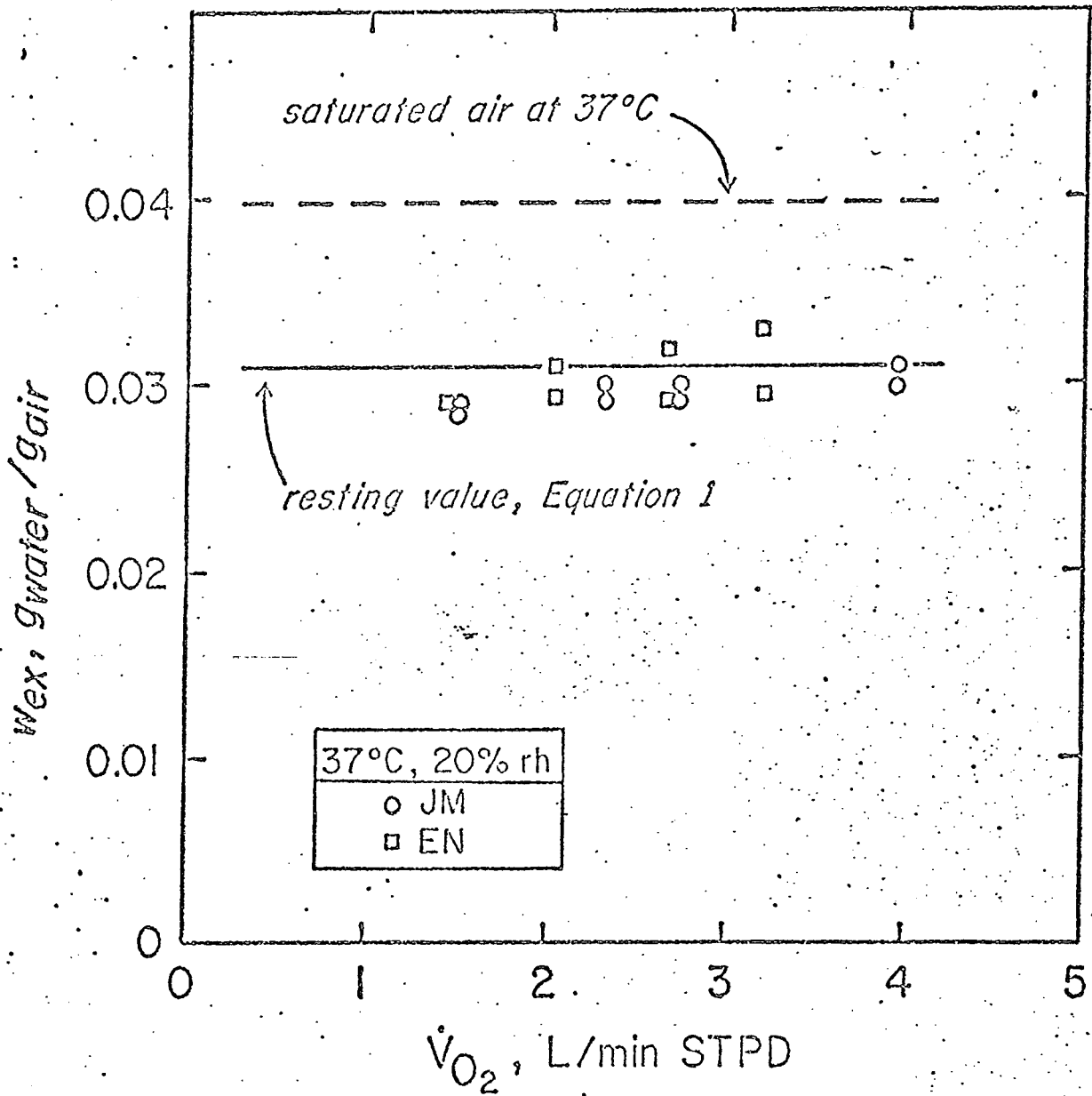
Figure 1: Absolute humidity of expired air (w_{ex}) as a function of oxygen consumption ($\dot{V}_{O_2}$) during exercise.

passages upon inspiration with a resultant cooling and dehumidification of the air upon expiration. Ingelstedt (3) has shown that under rest conditions the expired air in the laryngeal passage is essentially saturated at body temperature by the lungs for inspired air at temperatures as low as 0°C. Seeley (7) determined the humidity and temperature at various positions in the nasal passage, and demonstrated that cooling and dehumidification of the air occur during expiration. In addition to cooling by the surfaces, there is further cooling and dehumidification of the saturated air due to the mixing of the saturated tidal volume air (approximately two-thirds of the total inspired air) and the dead space air. The change in ventilatory patterns during exercise might be expected to alter these relationships.

It was surprising to find that the humidity of the expired air was the same during rest and exercise, since exercise is associated with a change from nasal to oral breathing, an increase in ventilatory flow rate by an order of magnitude, and a reduction in dead space from approximately 33% to less than 10% of the tidal volume. During exercise, the increased flow results in lower surface temperature and humidities in the respiratory passages due to increased evaporation. Nearly all of the inspired air will become saturated at body temperature since the respiratory dead space is a negligible fraction of the tidal volume. Consequently, the flow of air passing over the cooler and drier surfaces would result in a lower temperature and humidity in the expired air; however, a greater percentage of the air is saturated due to the increased tidal volume. Thus, during exercise there are apparently compensating processes that result in a humidity in the expired air that is essentially the same as that during rest.

As shown in Fig. 2, the average water loss data for moderate work loads lie

below the values given by Eq. 3 by 15-20 percent. This indicates that there is a difference in respiratory water loss as a function of O_2 - uptake between exercise and rest, but this difference is small. Fanger's relation generally agrees better with the data at higher work loads. The relation between ventilatory flow rate and oxygen uptake is essentially linear up to approximately 70 percent of $\dot{V}_{O_2}$ max, with an increased ratio of ventilation to oxygen uptake above this level (1). The increase in ventilation - $\dot{V}_{O_2}$ ratio at high work rates would be accompanied by increased water loss above the predicted linear relation (Eq. 3) at the higher work loads. Thus, the actual relation between water loss and oxygen uptake is not linear as proposed by Fanger, but of the form indicated by the dashed curve in Fig. 2.

During the course of this study, the weight loss associated with the excess CO_2 in the expired air was computed. The respiratory quotient R is the ratio of the moles of CO_2 eliminated to moles of O_2 taken up, and varies from a resting value of about 0.82 to greater than 1.0 during exercise (6). The rate of body weight loss resulting from the difference between the weight of CO_2 eliminated and O_2 taken up is

$$\dot{m}_r = \dot{V}_{O_2} \, (\underline{R} \cdot \rho_{CO_2} - \rho_{O_2}) \tag{4}$$

where $\dot{m}_r$ is in g/min and ρ_{CO_2} and ρ_{O_2} are the densities of carbon dioxide (1.96 g/L STPD) and oxygen (1.43 g/L STPD). For exercise assuming $\underline{R} = 1$, the weight loss becomes

$$\dot{m}_r = 0.53 \, \dot{V}_{O_2} \, .$$

It was concluded that during exercise up to about 80% of maximum aerobic capacity, the rate of water loss from the respiratory tract may be satisfactorily predicted using Eq. 3. The differences between the data of the present study and Eq. 3 are small enough (approximately 0.25 g/min) to be within the uncertainty

associated with whole body sweating rate determinations. The rate of weight loss during exercise due to differences in weight between expired CO_2 and O_2 uptake may be calculated from Eq. 4 or 5 (when $R = 1$). The respiratory losses can be an appreciable fraction of the measured whole body weight loss rates during exercise. For typical exercise conditions, the respiratory weight loss due to evaporation of water and due to $CO_2 - O_2$ differences are on the order of 1 to 2 g/min each.

Present address of J. W. Mitchell: Department of Mechanical Engineering,

University of Wisconsin, Madison, Wisconsin.

References

1. Astrand, P. O., and K. Rodahl, <u>Textbook of Work Physiology</u>, New York, McGraw Hill, 1970, p. 208.

2. Fanger, P. O., <u>Thermal Comfort</u>, Copenhagen, Danish Technical Press, 1970.

3. Ingelstedt, S., Studies on the conditioning of air in the respiratory tract. <u>Acta Oto-Laryn.</u>, Supp. 131, 1956.

4. McCutchan, F. W. and C. L. Taylor, Respiratory heat exchange with varying temperature and humidity of inspired air. <u>J. Appl. Physiol.</u>, 4: 121-135, 1951.

5. Nadel, E. R., R. W. Bullard, and J.A.J. Stolwijk, The importance of skin temperature in the regulation of sweating. <u>J. Appl. Physiol.</u>,31:80-87, 197

6. Saltin, B., A. P. Gagge, U. Bergh, and J.A.J. Stolwijk, Body temperatures and sweating during exhaustive exercise. <u>J. Appl. Physiol.</u>, submitted for publication.

7. Seeley, L. E., Study of changes in the temperature and water vapor content of respired air in the nasal passage.' <u>Trans. ASHVE</u> 46: 259-290, 1940.