$$h_e = 1587 h_c P/(P-p_a)^2 \qquad (2.9)$$

Respiratory heat exchange occurs directly between the core and the environment and is calculated from the following (Stewart 1982),

$$E_{res} = 0,0017MC_p^*(T_{wo}-T_{wb}) \qquad (2.10)$$

$$\text{where } T_{wo} = 32,6 + 0,066T_{db} + 0,2p_a \qquad (2.11)$$

The equivalent specific heat of most air, $C_p^*$ may be calculated from the following (Whillier 1977),

$$C_p^* = 1,6481e^{(0,0415T_{wb})} \qquad (2.12)$$

$E_{res}$ is significant only in cases of high ventilation rates in dry environments (Atkins 1975).

The heat balance equation,

$$M - W_o - E_{res} - R - C - E = S_t \qquad (2.13)$$

can be solved for a value of $\bar{T}_s$ at any instant in time provided that both physiological conductance, K and skin wettedness, W are known. The block $G_K(S)$ represents the transfer function between $T_{re}(S)$, $\bar{T}_s(S)$ and $M(S)-W_o(S)$ as inputs and K(S) as output. $G_{SR}(S)$ represents the transfer function between $T_{re}(S)$, $\bar{T}_s(S)$ and environmental conditions as inputs and W(S) as output. Together these two blocks constitute the controlling system.

The change in the weighted body temperature driving signal, $T_{mb}(S)$ can be calculated by integrating the body heat storage and dividing by the mass and specific heat of the body. The instantaneous rectal temperature can be calculated from a knowledge of $T_{mb}$ and $\bar{T}_s$.

It is clear from the literature reviewed that the use of the model as a dynamic predictor of the physiological responses to heat and work depend, to a large extent, on the accurate and complete description of $G_K(S)$ and $G_{SR}(S)$. The literature on overall models of human thermoregulation has indicated that sufficient attention to detail has not been devoted to the controlling system. Their use as accurate predictors of rectal temperature and other physiological responses to work and heat is therefore doubted.

The remainder of the current chapter is therefore directed at determining, by means of a more specific literature review, the current state of knowledge on physiological conductance and sweat rate. The two sections which follow report "state of the art" representations for $G_K(S)$ and $G_{SR}(S)$ as well as identifying specific areas of these two responses requiring further investigation.

## 2.4 Physiological Conductance - A Review

### 2.4.1 Definition of physiological conductance

As early as 1934, Burton (1934) and Burton and Bazett (1936) described the internal heat transfer coefficient in terms of a lumped parameter, physiological conductance, K. They expressed the heat flux between the core and shell as follows,

$$K(T_{cr}-\bar{T}_s) = M - W_o - E_{res} - S_t = M^* \qquad (2.14)$$

Implicit in the statement of this equality is the single cylinder core and shell representation of the human body. The driving force for the internal heat transfer is the difference between an assumed uniform core temperature and the mean skin temperature.

Two modes of heat transfer make up physiological conductance, tissue conduction and convection as a result of blood circulation. If the tissue conductance between core and shell is $h_t$ and the mass flow rate of blood is $\dot{m}_b$, physiological conductance may be written in terms of two components,

$$K = h_t + \dot{m}_b C_{pb} \qquad (2.15)$$

The tissue conductance component, $h_t$ accounts for approximately 20 per cent of the internal heat transfer at rest and a negligible amount during exercise (Atkins 1962). Since heat conduction takes place across an unperfused skin layer, the geometry of the core relative to the skin is important and could well change from rest to various levels of exercise (Nielsen 1969).

Most of the heat generated internally is transferred by way of vascular convection of warm blood through cooler skin vessels. Atkins (1975) has given a detailed account of the mechanisms through which the blood exchanges heat with body tissue, the possible existence of countercurrent heat exchange between veins and arteries and the operation of the pre-capillary sphincter and arteriovenous anastomoses. Paterson (1976) concluded that the site of delivery of warm blood to the periphery is under active control and therefore influences the thickness of the skin layer over which conduction occurs. The degree of dilation or constriction (tone) of the skin blood vessels is adjusted both locally and centrally by the thermoregulatory controller (Revow 1981). It follows therefore that physiological conductance is primarily a function of skin blood flow.

### 2.4.2 Empirical descriptions of physiological conductance in terms of physiological variables

In keeping with the original definition of physiological conductance derived by Burton (1934), physiologists have described the way in which K is controlled in terms of various physiological variables.

Mitchell (1972) compared the correlations between experimentally determined K and various individual and combined body temperatures. He concluded that the best correlation was obtained with skin temperature and the worst with rectal temperature. Correlations of K with mean body temperature and oesophageal temperature were highly significant and not different from each other. He concluded that it was difficult to confirm that the same inputs controlled the level of K during exercise and during rest. When men worked, their conductances increased above resting levels. He explained this observation by saying that the gain of the thermoregulatory system increased during exercise. He did not however establish a functional relationship between K, body temperatures and level of exercise.

Nielsen (1969) plotted physiological conductance against mean skin temperature at rest and at two levels of exercise. Their results indicate that for the same level of mean skin temperature, K increases from a value at rest to values which rise with increasing level of exercise. A relationship between K, mean skin temperature and metabolic rate was not mathematically derived.

MacDonald and Wyndham (1950) and Wyndham (1965) investigated the relationship between control actions and body temperatures. The relationships between K and rectal temperature and K and skin temperature were highly non-linear. The sensitivity of K to changes in rectal temperature was between 4 and 10 times greater than the

sensitivity of K to changes in skin temperature. The possible effect of metabolic rate was not considered. Wyndham et al (1965) plotted K against rectal temperature. The shape of the curve was sigmoidal (see Figure 2.5(a)) and indicated that K saturated at both low (36°C), and high (41°C) rectal temperatures. Experimentation lasted five hours and plots of K versus rectal temperature for each hour indicated that physiological conductance was independent of time after the first hour of exposure. No indication of the dynamics within the first sixty minutes, or the possible influence of metabolic rate was given.

The equation they derived has been adjusted so that K can be expressed in the units $W/m^2°C$,

$$K = 76,2 - 64,6e^{-0,114(T_{re}-36)^{2,46}} \qquad (2.16)$$

Wyndham and Atkins (1968) determined the average physiological conductance response of acclimatized men to rest and exercise over a wide range of environmental conditions. Their results show that physiological conductance varies with both rectal and mean skin temperatures. Cohen and Stewart (1982) have shown that when rectal and mean skin temperatures are combined into a weighted body temperature driving signal, $T_{mb}$ such that

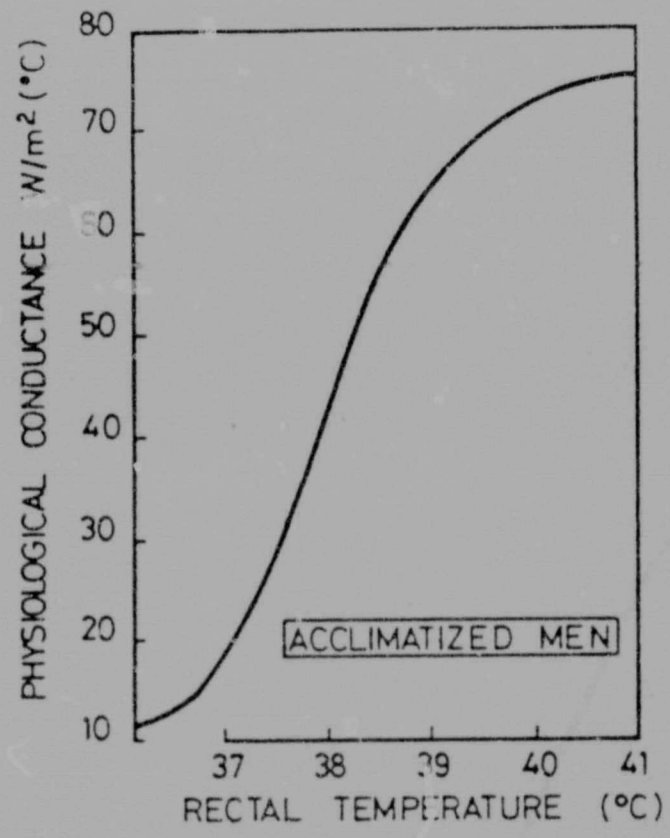
$$T_{mb} = 0,9T_{re} + 0,1\bar{T}_s \qquad (2.17)$$

the separate curves of Figure 2.5(b) appear to collapse into the single curve shown in Figure 2.6. The equation which describes the relationship depicted in the above Figure is as follows,

$$K = 10 + \frac{139}{(1+e^{-16,2X})} \qquad (2.18a)$$

$$\text{where } X = \frac{1}{(42-T_{mb})} - \frac{1}{(T_{mb}-34)} \qquad (2.18b)$$

a)




b)

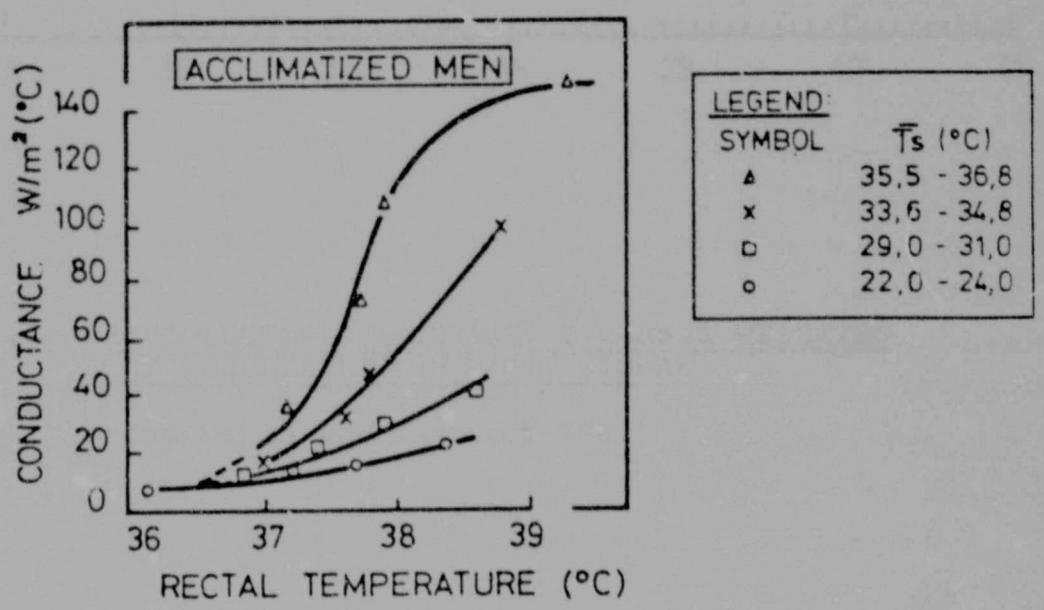



Figure 2.5 PHYSIOLOGICAL CONDUCTANCE AS A FUNCTION OF RECTAL TEMPERATURE

a) based on Wyndham et al (1965)

b) based on Wyndham and Atkins (1968)

(from Stewart 1981)

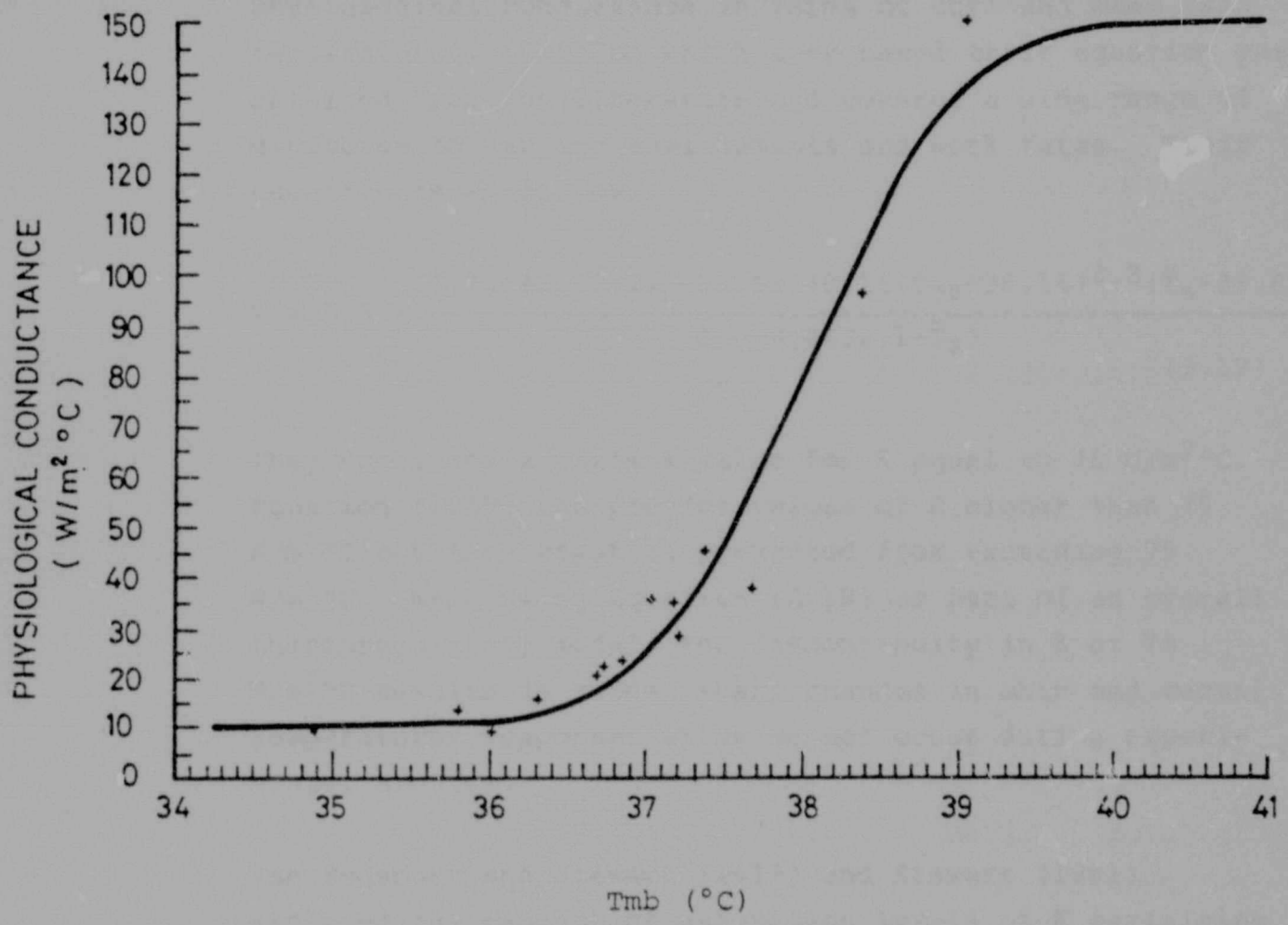



Figure 2.6 PHYSIOLOGICAL CONDUCTANCE VERSUS WEIGHTED BODY TEMPERATURE DRIVING SIGNAL

(from Cohen and Stewart 1982)

Unfortunately Wyndham and Atkins (1968) did not obtain a wide enough range of $T_{mb}$ for each metabolic group and it was therefore not possible to identify whether a metabolic rate effect was present in their data.

Azer and Hsu (1977) developed an equation describing physiological conductance in terms of core and mean skin temperatures. Data on which they based their equation was obtained from the literature and covered a wide range of exposures to varying environments and work rates. Their equation is as follows,

$$K = 5,3 + \frac{6,75+42,45(T_{cr}-36,98)+8,15(T_{cr}-35,15)^{0,8}(\bar{T}_s-33,8)}{1 + 0,4(32,1-\bar{T}_s)} \qquad (2.19)$$

They specified a maximum value for K equal to 75 $W/m^2°C$. Equation (2.19) can predict values of K higher than 75 $W/m^2°C$ but the output is prevented from exceeding 75 $W/m^2°C$. When using Equation (2.19) as part of an overall thermoregulatory model, the discontinuity in K at 75 $W/m^2°C$ results in sudden sharp changes in skin and rectal temperatures responses which do not occur during experimental studies.

Van Rensburg and Stewart (1977) and Stewart (1981) explored the concept of saturation levels of K pertaining to the highly non-linear form of the relationship between K and $T_{mb}$. The maximum level of K attained by resting men in experiments reported by Wyndham et al. (1968, 1970) was approximately 42 $W/m^2°C$. Both Figures 2.5(a) and 2.6 show that much higher levels of conductance may be achieved and it is clear that the saturation level of conductance must be variable and cannot be associated with a single level of body temperature. Stewart (1981) concluded that these observations raised the possibility that conductance might saturate at different levels for different metabolic rates. He went on to show that conductance could be

expressed as a function of rectal, skin temperatures and metabolic rate. Experimentation was carried out on a large number of unacclimatized men. Unfortunately not a wide enough range in $T_{mb}$ (computed from Equation (2.17)) was achieved and it was only possible to derive a functional relationship over a limited range of response. The empirical equation he derived is,

$$K = 22,46 - 0,2503M - 11,23(T_{mb}-36,5)^{0,5} + 0,3569M(T_{mb}-36,5)^{0,5} \qquad (2.20)$$

for $36,5 \leq T_{mb} \leq 39,3$

The saturation levels of K at low and high values of $T_{mb}$ could not be determined from the range of experimental data.

The abovementioned equations provide the means for predicting values of K from a knowledge of body temperatures. Stewart's (1981) equation includes M as another independent variable, a factor which has not been physiologically explained. The following theoretical analysis attempts to explain why K could be modulated by M.

During rest, the single cylinder core and shell model of the human body could be considered as being adequate. The basal metabolic energy production is distributed more or less evenly throughout the core. During steady state, a single temperature such as the rectal temperature is representative of core temperature and the calculation

$$K = \frac{M - W_o - E_{res}}{(T_{re}-\bar{T}_s)} \quad , S_t = 0 \qquad (2.21)$$

gives an accurate measure of physiological conductance.

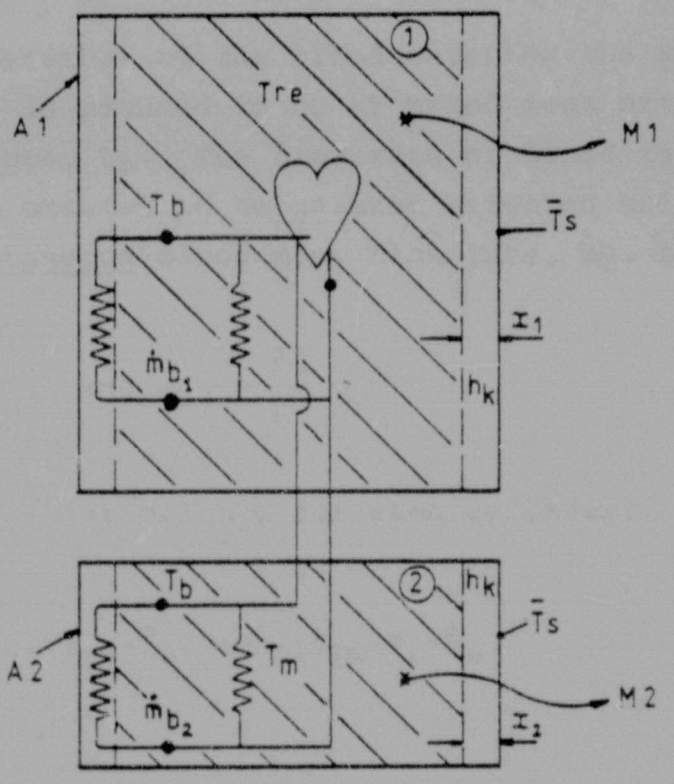
During exercise however, the single cylinder core and shell model does not adequately represent the human body. The metabolic energy over and above the basal level is produced in the muscles actively involved in exercise. As such regions of the core associated with these muscles will be at a higher temperature than the rest of the core. The average core temperature would not be equal to the rectal temperature, but could be somewhat higher depending on the level of exercise. The weighted body temperature driving signal, $T_{mb}$ calculated from a weighted sum of rectal and mean skin temperature could well be an underestimate of the true mean body temperature during exercise. The "error" involved in calculating K as in Equation (2.21) above would therefore depend on the extent to which regions of the core are hotter than the rectum.

During experimentation carried out in this study, exercise consisted of block stepping involving only the legs. The two cylinder model to be described was therefore constructed to mathematically determine the cause of the "error" mentioned above.

Consider the two cylinder model represented in Figure 2.7. The upper cylinder depicts the trunk of man and has a surface area $A_1$ and metabolic heat production equal to the basal rate, $M_1$. The lower cylinder represents the metabolically active legs having area $A_2$ and metabolic heat production $M_2$. Therefore the total surface area, A is equal to the sum $A_1+A_2$ and the total metabolic heat production, M is equal to $M_1+M_2$.

The shells of the two cylinders have equal thermal conductivity $h_k$ and equal thickness x. The conductances of the shells are therefore both equal to $h_k/x=h_t$.

The core of the upper cylinder is assumed to be at rectal temperature, $T_{re}$. The core of the lower cylinder is




$$A_1 + A_2 = A$$

$$M_1 + M_2 = M^*$$

$$X_1 + X_2 = X$$

Figure 2.7 TWO CYLINDER CORE AND SHELL MODEL OF MAN

assumed to be at muscle temperature, $T_m$. Both shells are assumed to be at the same mean skin temperature, $\bar{T}_s$

The temperature of the blood entering the shell of each cylinder is assumed to be at mixed mean arterial blood temperature, $T_b$. The flow rate of blood through each shell is assumed to be an area weighted multiple of the total cutaneous blood mass flow rate, $\dot{m}_b$, such that

$$\dot{m}_{b1} = \frac{A_1}{A}\dot{m}_b \text{ and } \dot{m}_{b2} = \frac{A_2}{A}\dot{m}_b \tag{2.22}$$

Writing a heat balance for each cylinder,

$$M_1 = \frac{A_1}{A} h_t(T_{re}-\bar{T}_s) + \dot{m}_{b1}C_{pb}(T_b-\bar{T}_s) \tag{2.23}$$

and

$$M_2 = \frac{A_2}{A} h_t(T_m-\bar{T}_s) + \dot{m}_{b2}C_{pb}(T_b-\bar{T}_s) \tag{2.24}$$

Substituting (2.22) into (2.23) and (2.24),

$$M_1 = \frac{A_1}{A} (h_t(T_{re}-\bar{T}_s) + \dot{m}_b C_{pb}(T_b-\bar{T}_s)) \tag{2.25}$$

and

$$M_2 = \frac{A_2}{A} (h_t(T_m-\bar{T}_s) + \dot{m}_b C_{pb}(T_b-\bar{T}_s)) \tag{2.26}$$

$$\text{Let } T_m = T_{re} + \Delta T_m \text{ and } T_b = T_{re} + \Delta T_b \tag{2.27}$$

Adding (2.25) and (2.26) and substituting (2.27) results in,

$$M_1+M_2=(T_{re}-\bar{T}_s)(h_t+\dot{m}_bC_{pb})+\Delta T_b\dot{m}_bC_{pb}+\frac{A_2}{A}\Delta T_m h_t \qquad (2.28)$$

The overall physiological conductance of the two cylinder model can be calculated from Equation (2.28) in the same way that it is calculated for the single cylinder model,

$$\frac{M_1+M_2}{(T_{re}-\bar{T}_s)} = K' = (h_t+\dot{m}_bC_{pb}) + \frac{\Delta T_m}{(T_{re}-\bar{T}_s)} h_t \frac{A_2}{A} + \frac{\Delta T_b\dot{m}_bC_{pb}}{(T_{re}-\bar{T}_s)} \qquad (2.29)$$

but from Equation (2.15), $h_t+\dot{m}_bC_{pb}$ is equal to K, the physiological conductance for the single cylinder core and shell model.

The value of physiological conductance calculated for the exercising man from a measure of rectal and mean skin temperatures and metabolic rate could therefore be in "error" by the quantity

$$\frac{\Delta T_m}{(T_{re}-\bar{T}_s)} h_t\frac{A_2}{A} + \frac{\Delta T_b}{(T_{re}-\bar{T}_s)} \dot{m}_bC_{pb} \qquad (2.30)$$

The first term represents the "error" in the conductive component of physiological conductance and the second term represents the "error" in the convective component of physiological conductance.

Saltin and Hermansen (1966) and Saltin et al. (1968) measured intramuscular temperature during leg exercise. Their data indicate that the magnitude of the difference between rectal and muscle temperatures could be as much as 3,5°C from rest to heavy exercise.

Their findings indicate that $T_m$ is a function of the metabolic rate,

$$\Delta T_m = f_1(M) \qquad (2.31)$$

Eichna et al. (1951) measured the difference between arterial and rectal temperature in resting subjects. Under steady state conditions, mean arterial temperature was 0,25°C lower than rectal temperature. During exercise, Saltin and Hermansen (1966) used oesophageal temperature as a measure of arterial blood temperature. They found that the difference between rectal and oesophageal temperature increased from rest to hard work by more than 0,1°C. $\Delta T_b$ could therefore also be expressed as a function of metabolic rate,

$$\Delta T_b = f_2(M) \qquad (2.32)$$

If Equations (2.31) and (2.32) are substituted back into Equation (2.29), it can be concluded that physiclogical conductance will be subject to a metabolic rate effect. The fact that both $\Delta T_m$ and $\Delta T_b$ increase with increasing metabolic rate indicates that physiological conductance will increase with increasing level of exercise. The preceding theoretical analysis has been confirmed experimentally by Stewart (1981) (see Equation (2.20)) for unacclimatized men.

It is easy to explain why the metabolic rate effect has been missed by other researchers. Experimentation carried out by Wyndham (1965), Wyndham et al. (1965) and Wyndham and Atkins (1968) did not achieve a range of $T_{mb}$ values for each rate of metabolic heat production. Instead, high $T_{mb}$ values were a result of low metabolic rates and cool environments. Their single curves of K plotted against either $T_{re}$, $\bar{T}_s$ or a weighted sum of $T_{re}$ and $\bar{T}_s$ were therefore founded upon pockets of data at different

metabolic rates giving the impression that K was not dependent on metabolic rate.

By combining the form of the empirical equations cited previously with the theoretical analysis presented above, it is possible to suggest the relationship between physiological conductance, the weighted body temperature driving signal and metabolic rate drawn qualitatively in Figure 2.8. It is the aim of the experimentation, discussed in Chapter 3, to quantify the relationship between K and M at low and high values of $T_{mb}$ as well as to determine the form of the equations relating K to $T_{mb}$.

## 2.5 Thermoregulatory Sweating - A Review

The secretion of sweat in order to wet the skin surface and the cooling derived from the evaporation of the sweat, is the major mode of heat loss in hot environmental conditions. In order to calculate the quantity of evaporative heat loss from Equation (2.6) the wettedness of the skin, W must be known. Accordingly the model in Figure 2.4 predicts W(S) as the output of the thermoregulatory sweating block, $G_{SR}(S)$. A complex equation relates W to the sweat rate under steady state conditions (see Section 5.2.4) but the same equation does not hold during transient conditions. Physiologists have concentrated rather on correlating sweat rate with various body temperatures during steady state.

### 2.5.1 Empirical descriptions of sweat rate in terms of physiological variables

Wyndham et al. (1954, 1965) expressed sweat rate in terms of rectal temperature. Hatch (1963) on the other hand correlated sweat rate with skin temperature. Benzinger (1964) postulated that sweating was purely dependent on internal body temperature,

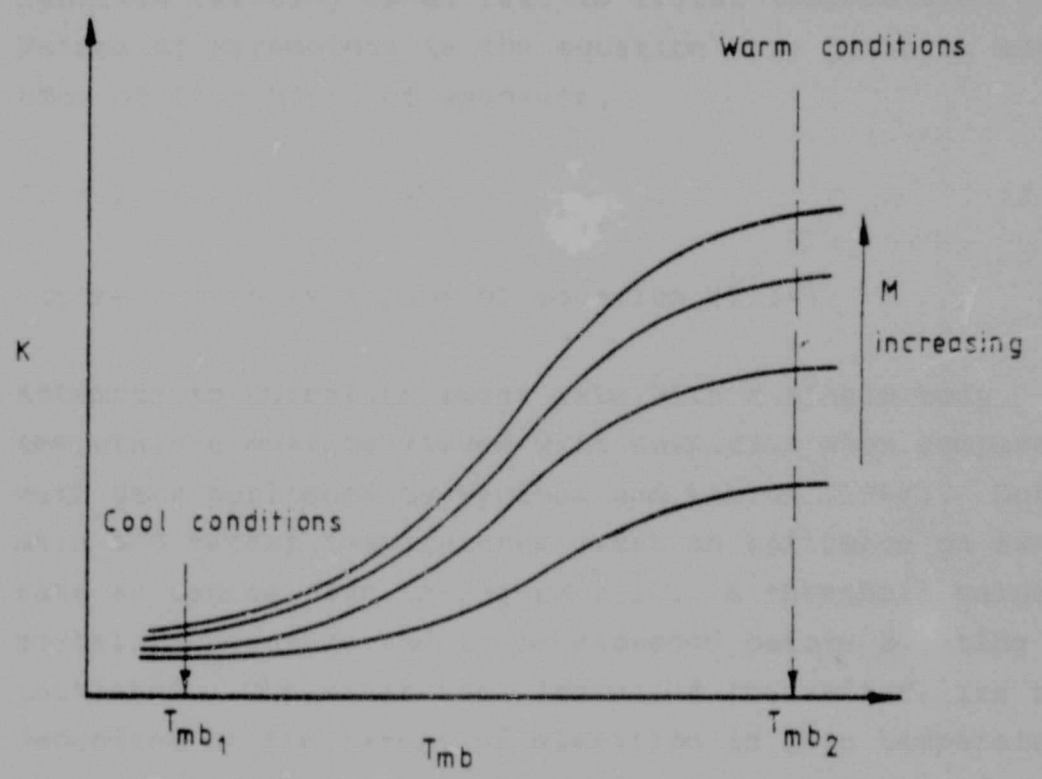



Figure 2.8 FORM OF THE PHYSIOLOGICAL CONDUCTANCE WEIGHTED BODY TEMPERATURE DRIVING SIGNAL RELATIONSHIP SHOWING THE EXPECTED INFLUENCE OF METABOLIC RATE

$$S_r = \alpha(T_{set} - T_{hy}) \qquad (2.33)$$

The most useful of these correlations was performed by Wyndham et al. (1966). They demonstrated a highly non-linear sweat rate response which became saturated at high rectal temperatures. They postulated a double exponential function relating sweat rate to rectal temperature. Values of parameters in the equation were given at each hour of five hours of exposure,

$$S_r = k_1(1-k_2 e^{k_3(k_4-T_{re})^n}) \qquad (2.34)$$

Figure 2.9 shows a plot of equation (2.34).

Attempts to correlate sweat rate with a single body temperature must be viewed with suspicion when compared with data published by Wyndham and Atkins (1968). Both skin and rectal temperatures exert an influence on sweat rate as can be seen in Figure 2.10. A threshold value of rectal temperature had to be exceeded before sweating was initiated. The sweat rate increased thereafter, its rate depending on the extent of elevation in skin temperature. They quantified the sweat rate response in the region where skin temperature inhibited sweating,

$$S_r = (T_{re}-36,5)(0,1+0,455e^{0,27(\bar{T}_s-33)}) \qquad (2.35)$$

Since the rate at which the body can secrete sweat must be subject to some upper limit, eventually a point will be reached where the body will no longer be able to increase its sweat output in response to an increase in body temperatures. The sigmoidal shape of the curves in Figures 2.9 and 2.10 emphasize this point. However in much of the literature, a linear function has been used to describe the relationship between sweat rate and body temperature. Stolwijk and Hardy (1966) postulated the following linear expression for evaporative heat loss in terms of deviations from internal and surface temperatures,

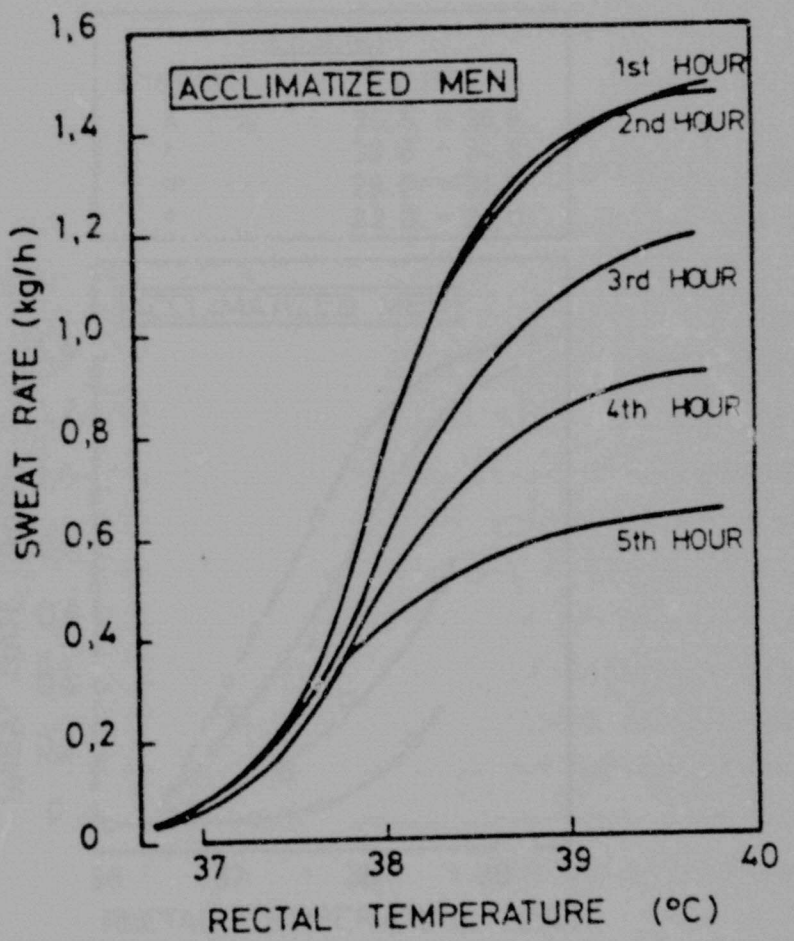



Figure 2.9 SWEAT RATE RESPONSE CURVES DETERMINED FOR VARIOUS PERIODS OF HEAT EXPOSURE. BASED ON WYNDHAM ET AL (1966)

(from Stewart 1981)

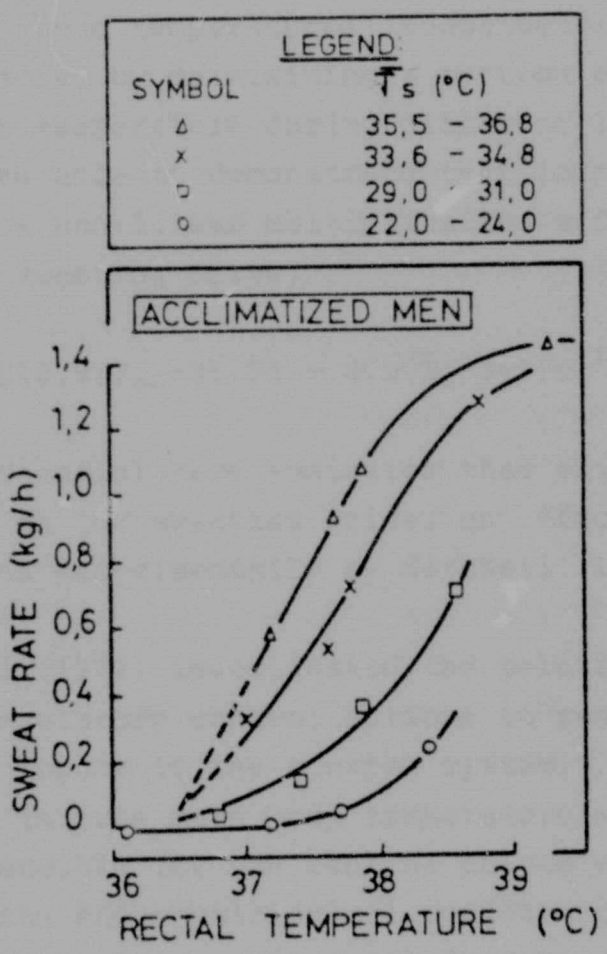



Figure 2.10 SWEAT RATE VERSUS RECTAL TEMPERATURE AT VARIOUS LEVELS OF MEAN SKIN TEMPERATURE. BASED ON WYNDHAM ET AL (1966) (from Stewart 1981)

$$E = 5,8 + 93(T_e - 37,18) + 21(\bar{T}_s - 33) \quad (2.36)$$

Gagge et al. (1974) combined skin and rectal temperature, using weightings of 0,2 and 0,8 respectively, into a representative mean body temperature. Sweat rate was plotted as a linear function of mean body temperature.

Nadel et al (1971a, 1971b) attempted to identify the relative importance of core and skin temperatures by varying these temperatures independently of each other. Furthermore, by maintaining a portion of the skin at constant temperature during different levels of heating, they were able to demonstrate that local skin temperature exerted a non-linear multiplicative effect on the linear central sweating drive,

$$S_r = 220[0,9(T_{es} - 36,7) + 0,1(\bar{T}_s - 34)]e^{((\bar{T}_s - 34)/10)} \quad (2.37)$$

The exponential term indicates that skin temperature acts locally on the sweating drive, an effect which has been confirmed experimentally by Mitchell (1972).

Mitchell (1972) investigated the relationships between thermoregulatory control actions in resting men and various inputs to the control system. He concluded that changes in true mean body temperature were most likely to be responsible for the central drives which control both sweat rate and physiological conductance. The correlation between sweat rate and mean body temperature was higher than between sweat rate and rectal temperature.

Bullard, Banerjee et al. (1970) investigated, by experimentation the influence of skin temperature on the stimulation of the sweat gland. Two mechanisms were believed to exist. One is local and directly affects the function of the sweat gland, the second is of a neural nature involving thermal receptors and the integration of these afferent signals by the central nervous system. The model

which they postulated utilizes hypothalamic proportional control with the modification that skin temperature acts to determine the reference level. Their model is expressed mathematically as follows,

$$E = \alpha(T_{set}(N)-T_{hy})L \qquad (2.38)$$

where $T_{set}(N)$ = function of the mean weighted skin temperature

$L$ = function of local skin temperature.

They observed further that the rate of sweating was depressed when the subject's legs were cooled in water, the extent of this depression was proportional to the rate of cooling of the surface and the area cooled.

It has been demonstrated by Wyndham et al. (1966) that the sweat rate of an individual may decrease with time even though body temperatures continue to rise. Similar results have been found by other researchers (see Kerslake 1972). Two mechanisms are thought to be responsible for this decay of sweating with time, sweat gland fatigue and the skin wettedness effect (hidromeiosis). The contributions of both the above mechanisms to the suppression of sweating is discussed at length in Chapter 5.

Nadel and Stolwijk (1973) have included a parameter $\phi$ as a multiplier on Equation (2.37) which reduces the sweat rate as skin wettedness increases,

$$S_r = \phi[\alpha(T_{es}-36,7) + \beta(\bar{T}_s-34)]e^{(\bar{T}_s-34)/10} \qquad (2.39)$$

The wettedness suppression factor $\phi$ is a function of the wettedness divided by the drive as shown in Figure 5.9. Equation (2.39) does not however explain the decay in sweat rate which occurs with time, an observation which is clear in the experimental results discussed in Chapter 5.

Stewart (1981) after a comprehensive review of the literature reached the following important conclusion: "For a given thermoregulatory stimulus, and provided a certain level of skin wettedness has been exceeded, sweat rate decreases exponentially with time and the rate of decrease depends on the skin wettedness". The implication is that, whereas sweat rate has been expressed as a function of rectal and skin temperatures (and more recently skin wettedness has been included) the effects of time per se, and of the cumulative duration of skin wettedness, should be included.

From the review just presented it is clear that there is neither a qualitative nor quantitative equation which includes all of the abovementioned effects. It can be concluded therefore that no comprehensive model of thermoregulatory sweating exists although some of the individual components have been investigated.

## 2.6 Discussion

In respect of the literature just reviewed, and in the light of the requirements for a thermoregulatory model discussed in Chapter 1, the first objective of the study can be evaluated and the scope and direction which has been adopted in order to achieve the second and third objectives is outlined.

Existing quantitative formulations of composite thermoregulatory models cannot be used to accurately predict the rectal temperature response of acclimatized men required to work, in underground mining conditions, for extensive periods of time. The reasons are;

(i) present models rely on experimentation carried out for short time periods. Long and short term transient response and predictions of steady state temperatures have been based on inadequate short term data,

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