

PROPERTIES OF THE HUMAN THERMOSTAT:  
RESULTS FROM MATHEMATICAL AND EXPERIMENTAL ANALYSES

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INTRODUCTION

Prediction of human thermoregulatory performance in heat and cold depends on detailed knowledge both of the passive thermal system and the active thermostat(1). Insight into the structure of the latter is still insufficient. However, it has become evident that a set of assumptions according to the kind and weighting of afferent inputs and the spatial distribution of effector mechanisms has to be made. In order to evaluate the minimal structural and functional requirements to the thermostat both mathematical and experimental analyses have been carried out.

RESULTS

Essential properties become evident already by using a one-cylinder-one-dimensional model of human thermoregulation for homogeneous thermal load. The passive system is essentially based on the following differential equation taking into account metabolic heat production, conduction and convection via circulation:

$$\rho(r)c(r)\frac{\partial T(r,t)}{\partial t} = M(r,t) + \frac{1}{r} \left[ s \lambda(r)r \frac{\partial T(r,t)}{\partial r} \right] / \frac{\partial r} + Bf(r,t) \cdot \rho_b \cdot c_b (T(r,t) - T_b),$$

where  $t$ =time coordinate,  $r$ =radial cylinder coordinate  $0 \leq r \leq R_{max}$ ,  $\rho$ =density,  $c$ =specific heat,  $T$ =temperature,  $M$ =metabolic heat production per volumetric unit,  $\lambda$ =heat conductivity,  $Bf$ =blood flow per volumetric unit, index  $b$ =blood. The boundary condition at the surface and at the central axis are

$$-\lambda \cdot \frac{\partial T}{\partial r} \Big|_{r=R_{max}} = \alpha \cdot (T_s - T_a) + Q_s$$

$$\frac{\partial T}{\partial r} \Big|_{r=0} = 0,$$

where  $\alpha$ =heat transfer coefficient, index  $a$  = air,  $Q_s$ =heat flow due to evaporation, index  $s$  = skin. According to an approach supported by many experimental results (2) the following essentials for the controller are assumed:

1. The integrative afferent signal  $f$  is proportional to the weighted sum of a central ("core temperature  $T_c$ ") and a peripheral ("mean skin temperature  $T_s$ ") signal.
2. A linear superposition of these signals sufficiently describes the system within its normal operating range.
3. All three effector systems have a minimal threshold and a maximal capacity of responses. Hence the afferent signal is:

$$f = (1-\tau)T_c + \tau T_s - f_0,$$

where  $f_0$  is the afferent signal in the indifferent status and  $\tau$  and  $1-\tau$  the weights of the central and the peripheral signals. Then the output  $y_i$  of the three effector systems is, using a gain factor

$$g_i: y_i = -g_i f + y_{i, \min}$$

$i = 1$ : metabolic heat production

$i = 2$ : blood flow

$i = 3$ : sweat production.

The derivative of core temperature with respect to ambient temperature  $dT_c/dT_a$  is a good measure of controller performance. The smaller this value the better is the compensation of disturbances by environmental temperatures. There is a high performance for  $0.1 \leq \tau < 0.25$  and  $100 \text{ W}^\circ\text{C}^{-1} \leq g_1 \leq 200 \text{ W}^\circ\text{C}^{-1}$ , and at the

same time a low sensitivity to morphological and environmental changes. This purely theoretical conclusion fits well to experimentally determined values of  $\tau$ . In order to obtain a good compatibility of computed and measured core and skin temperatures within a range of environmental temperature from 10°C to 50°C, the

following set of parameters was finally used:  $t_0=0.13$ ,  $g_1=115 \text{ W } ^\circ\text{C}^{-1}$ ,  $g_2=3450 \text{ W } ^\circ\text{C}^{-1}$ ,  $g_3=20.1 \text{ g } \text{m}^{-2} \text{ s}^{-1} \text{ } ^\circ\text{C}^{-1}$ ,  $y_{1\text{min}}=70 \text{ W}$ ,  $y_{2\text{min}}=1 \text{ ml } (100 \text{ g})^{-1} (60 \text{ s})^{-1}$ ,  $y_{3\text{min}}=300 \text{ g } \text{m}^{-2} \text{ d}^{-1}$ ,  $y_{1\text{max}}=300 \text{ W}$ ,  $y_{2\text{max}}=90 \text{ ml } (100 \text{ g})^{-1} (60 \text{ s})^{-1}$ ,  $y_{3\text{max}}=5000 \text{ g } \text{m}^{-2} \text{ d}^{-1}$ ,  $f_0=36.5^\circ\text{C}(3)$ . In particular the effect of inhomogeneous distribution of heat production and blood flow, the influence of body fat content, of controller gains, of weight of skin temperature feedback and of depth of peripheral receptors on the dynamic performance were analysed.

## CONCLUSIONS

increase of peripheral blood flow evokes essentially both an increase of energy requirement in the cold and a quicker system response. Differing rates of increase of metabolic heat production are the consequence of differing body fat content. The weight of skin temperature feedback can be limited to 5...20 %, because values outside this range evoke dynamic responses incompatible with the experiments. The actual value can only be determined if there is a correct assumption for the depth of the skin receptors. The use of measured superficial skin temperatures brings about an underestimation of the peripheral afferent signal. Of the controller gains it is primarily the gain of the metabolic controller which affects the dynamic response of the system. The experimental fact of a delayed onset of sweat production after a transition from cold to heat is the consequence of a high gain of the vasomotor system. When using a three-dimensional model (4,5) of the true geometry of the body with a grid of 0.5 to 0.1 cm it was concluded that it is essential to take into account the spatial distribution of heat production, blood flow and sweat production, and that at least for control of shivering, distributed controller gains different from the pattern of distribution of muscle tissue are required.

Supported by Deutsche Forschungsgemeinschaft (We 919/2)

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