EFFECTOR MECHANISMS IN PREDICTING HEART RATE RESPONSE TO VARIOUS METABOLIC RATES, ENVIRONMENTS AND CLOTHING

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INTRODUCTION

Heart rate response to different metabolic rates and environmental heat stress has been a subject for intensive studies. These studies primarily focused on investigating basic physiological mechanisms of heat exchange and thermoregulation. Some of the studies have also been aimed to describe in mathematical terms the response of heart rate to exercise and heat stress (1-3). The intra-relationship of various factors within the cardiovascular system and their cross dependence under exercise-heat-stress were not thoroughly investigated. The aim of the present study was to express in mathematical terms the relationships between the various components of the physiological effectors under exercise-heat stress.

MATERIALS and METHODS

Subjects Twenty healthy young (20.5±0.8 yrs), fit (VO_{2max}=3.9±0.1; $l \cdot min^{-1}$), acclimated male volunteers, participated in the study. All subjects were medically screened prior to their participation and signed a form of consent.
Measurements and calculations Data were obtained from a total of 36 experimental exposures which consisted of various combinations of metabolic rate, climatic conditions and clothing (table 1). During each exposure that lasted 30 min heart rate (HR), blood pressure (BP), cardiac output (Q), oxygen consumption (VO_2) and skin temperature (T_{sk}) were recorded. Heart rates, using ECG chest electrodes, were continuously radio-telemetered to an oscilloscope tachometer (Life scope 6; Nihon Kohden) were recorded every 5 min. Skin temperature was measured every 5 min by skin thermistors (YSI 409) at 3 locations (chest, arm, leg). Metabolic rate and cardiac output were measured towards the end of the experimental exposure. Expiratory gases were sampled and analyzed every 15 seconds by an automatic metabolic chart (CPX - MGC; Medical Graphic); a mean value of 2 min was used for determining VO_2. Cardiac output was determined by the CO_2 rebreathing techniques. Blood pressure was measured using an automated monitor every 15 min (Paramed 9000).
Models that describe heat balance relations between the various physiological variables underlay the present model. The required evaporation for thermal equilibrium (E_{req}) for indoor exposures and maximal evaporation capacity (E_{max})

Table **1**: The experimental combinations

Condition		Exposure	
Metabolic rate:		Rest	(5.100 W)
		Mild work	(≈300 W)
		Moderate work	(≈450 W)
Climatic condition:			
	indoor	Comfort (20°C, 20% RH)	
		Hot/dry (40°C, **35%** RH)	
		Hot/wet **(35°C,** 75% RH)	
	outdoor	**Solar** radiation (≈900 W/m^2)	
Clothing:		**Shorts only**	(clo=0.35, i_m/clo=0.94)
		Cotton **BDU**	(clo=0.99, i_m/clo=0.75)
		Nylon suit	(clo=0.70, i_m/clo=0.08)

were calculated as suggested by Givoni and Goldman **(4).** For outdoor exposures E_{req} was transformed according to Shapiro et *al.* **(5).**

Total peripheral resistance **(TPR)** was calculated from mean arterial blood pressure (MAP) and cardiac output (Q) as follows: $TPR=80(MAP \cdot Q^{-1})$; $dynes \cdot sec \cdot cm^{-5}$.

Statistical analysis A stepwise regression was applied to determine which of the physiological parameters are significant in characterizing HR response. Statistical calculations were performed using the **SAS 6.04** software. Linear models were fitted by the least square method (REG procedure). Nonlinear models were fitted by the **NLIN** procedure which also makes use of the least square method. All values are presented **as** mean ± **SEM.** p values less than **0.01** were considered significant.

RESULTS

The only physiological variables which have been found significant in determining HR were TPR, VO_{2max}, VO_2 and **Q.** The following relationship between these variables and HR could be expressed **as** follows:

$$HR = -65.5 + 99(VO_2/VO_{2max}) + 0.00009TPR^2 + 12.94Q - 0.29Q^2 \quad ;bpm$$

This model correlated well with the observations (R=0.72 p<0.0001), which distributed metrically without a trend around the line of identity as demonstrate in figure la. The validity of the model was confirmed by applying a separate set of database from a different study containing over **180** measurements **(6),** the co elation between measured and predicted values was **R=0.73** p<0.0001 (figure lb)

A set of odels based on bioclimatic variables could be applied to VO_2, TPR, and **Q** for the present model, **as** follows:

	R	p<
$VO_2 = VO_{2NT} + H_{r+c}(0.00055H_{r+c} + 125.3/E_{max})$	0.88	0.001
$TPR = 1490.6 - 0.795VO_2 - 1.074VO_{2max} + 0.00051(VO_{2max})^2$	0.68	0.001
$Q = 6.79 + 0.019VO_2 + 0.0217VO_{2max} - 0.00001(VO_{2max})^2 - H_{r+c}/E_{max}(3.44 + 0.0028VO_{2max})$	0.79	0.001

VO_{2NT} - VO_2 at normothermia (range: 300-500 **watt).**
H_{r+c} - *dry* heat exchange **(4).**

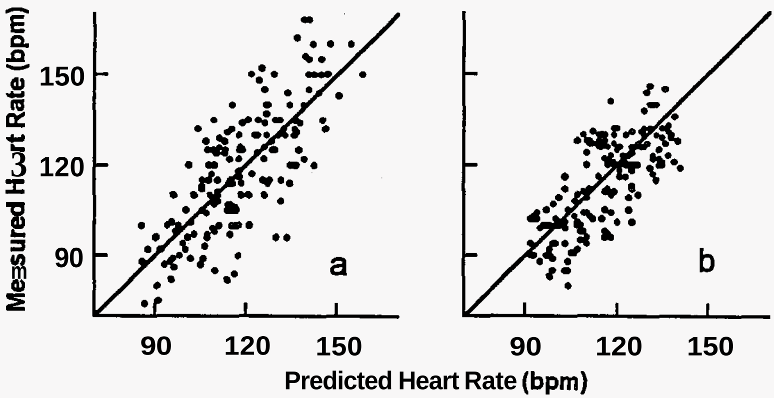

Figure 1. Predicted vs. measured heart rate response [a], and validation of the suggested model [b]; database compiled from Sagiv et al (6).

CONCLUSIONS

A previous mathematical model to predict HR used independent variables: climate and metabolic rate (3), while the present model is based on a cybernetic approach consisting of physiological parameters that are related to the cardiovascular system (VO_{2max}, TPR, Q,). Exercise and climate have a profound effect in determining the system's function (figure 2). This enables to express the physiological parameters (VO_2, TPR, Q) by biophysical parameters concerning heat load and heat dissipation (E_{req}, E_{max}). The implementation of E_{req}, and E into those parameters express the contribution of environmental condition on heat absorption and heat dissipation.

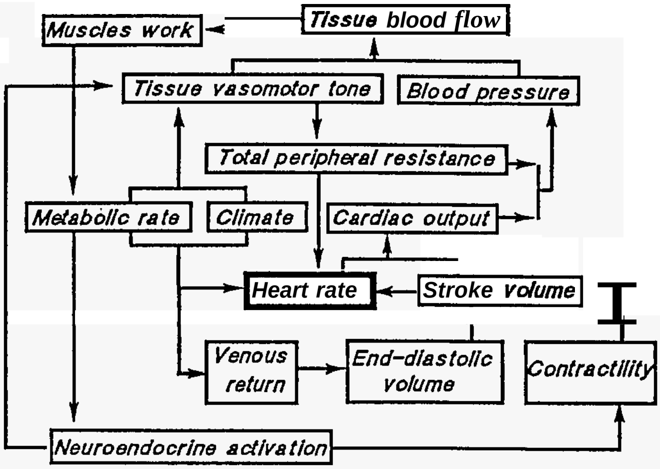

Figure 2. Effector mechanism in the cardiovascular system dynamics (modified from Rothe **(7)).**

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