## Incorporating Variable Vascular Heat Exchangers into Models Of Human Thermoregulation

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## Abstract

Models of human thermoregulatory function generally assume that heat transfer across the skin surface is uniform. However, only glabrous skin regions contain unique vascular structures that enable a large volume of blood to flow immediately below the skin surface. These are the body's radiators. We are constructing a novel model of thermoregulatory function that incorporates heat transfer across the glabrous skin regions as separate from heat transfer across the general skin surface.

## Modeling of human temperature regulation

Many mathematical models of thermoregulation in humans have been developed over the last 50 years. The first models were developed in the 1960's and became the accepted standard for human thermoregulatory function modeling. These early models were based on cylindrical representations of the body (e.g., three cylinders representing the head, trunk, and extremities, or six cylinders representing the head, trunk, two arms, and two legs). Key assumptions of these early models were that conduction within the cylindrical elements was purely radial, that heat transfer across the skin was uniform, and that different body segments are homogeneous with respect to temperature. While the models have grown in complexity and diversity over the years, the assumption that heat transfer across the skin surface is uniform has persisted (for examples see Wissler, 2008, Nelson 2009, Yokota et al 2008). Unfortunately, when these original generated the primary mammalian models were thermoregulatory effector mechanism - the body's radiators - had yet to be appreciated. In fact hands and feet were excluded from the models.

A majority of the internally generated heat is dissipated from the body across the skin surface (less the portion of the internally produced heat that is dissipated through respiration). Heat is moved around the body by the circulating blood. Heat is produced as a byproduct of cellular metabolism. The greater the metabolic effort of a cell, the greater is the heat production.

The tissues of body can be sorted according to their resting metabolic activity. The thermal core is comprised of the metabolically active visceral organs (kidneys, heart, lungs, brain, and splanchnic organs). These core organs comprise less that 10% of the total body mass but account for 70-75% of the total resting heat production. Peripheral tissues are comprised of those tissues with very low resting metabolic activity (the skin, skeletal muscles, bone, and connective tissues). During rest the peripheral tissues are metabolically quiescent and, although comprising more than 90% of the total body mass, account for less than 30% of the resting metabolic heat production.

Generally, blood flow to the tissues is determined by metabolic demand. In a human resting in a thermoneutral environment 65-70% of the total cardiac output goes to the core organs and 30-35% goes to the peripheral tissues with less than 3% flowing to the skin (excluding thermoregulatory blood flow). Capillary blood flow to all skin regions is uniform and relatively stable.

There are two conditions in which the distribution of cardiac output changes: exercise and heat stress. During exercise the metabolic demand of the active muscles results in increased blood flow to those tissues. During heat stress large volumes of blood are delivered to the skin to facilitate heat transfer. Both conditions result in an increase in cardiac output (up to 14 L/min with heat stress alone and up to 20 L/min during exercise). During heat stress the metabolic demands of the core organs and peripheral tissues do not change and, thus, the blood flow serving the

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metabolic demands of the tissues does not change. In fact, blood flows to all tissues measured (including the hairy skin regions) decrease slightly during heat stress. During exercise only the active muscles receive an increase in blood flow (Rowell, 1974).

Circulating blood delivers heat to the body surface for dissipation to the external environment. However, regional distribution of blood flow to the skin is not uniform. The surface of the body is characterized by two types of skin; glabrous skin and non-glabrous skin. The glabrous skin covers about 5% of the body surface (palms of the hands, soles of the feet, and portions of the face and ears) and is characterized by an absence of hair follicles and a dense network of subcutaneous vascular structures. The subcutaneous vascular structures consist of arteriovenous anastamoses (AVAs) and glomerular structures of tortuous thin vessels (retia venosa) (Sangiorgi et al 2004, Manelli et al 2005). These densely packed structures serve no metabolic function and, although they are 2-5X larger in diameter than capillaries, they are as densely packed as the capillary beds. The non-glabrous skin covers the remaining 95% of the body surface and is characterized by the presence of hair follicles and an absence of the AVAs and retia venosa.

Blood flow patterns though the two different skin types are dramatically different. Blood flow through the nonglabrous skin regions is of relatively low volume and high stability. Conversely, blood flow through the glabrous skin regions is extremely variable and can be of high volume.

During exercise and/or heat stress, the substantial increases in skin blood flow are confined to the glabrous skin regions of the body surface (Yamazaki 2002). Only the glabrous skin regions of the body surface contain the AVAs and retia venosa that can accommodate large increases in blood flow to facilitate heat loss. Thus, increased capacity for heat dissipation occurs primarily across the non-hairy surfaces of the body. Blood flow through the AVAs and retia venosa is tightly regulated and determined primarily by thermoregulatory demands.

The subcutaneous structures underlying the glabrous skin regions are arranged in parallel. When open, these vascular structures provide a low resistance to flow pathway between the arterial output and venous return side of the heart. This enables the thermal effect at the skin surface to be rapidly transported back to the central circulation. Contrary to conventional wisdom, changes in vasomotor tone (i.e., vasodilation and vasoconstriction) refer to changes in blood flow through the glabrous skin regions, not the entire body surface.

Our technical objective was to design a mathematical model of human thermoregulatory function that included the glabrous skin region AVAs and retia venosa as a variable and controlled heat loss component. Previous models assumed that different body segments are homogeneous with respect to temperature and that heat was exchanged with the environment in a uniform manner across all surfaces of the body. While heat loss across the general body surface is passive and uncontrolled, heat loss through the non-hairy skin regions is tightly regulated by controlling blood flow through the AVAs and retia venosa. We are building these variable heat exchangers into a model of human thermoregulation to generate a dynamic computer model of heat distribution within the body and heat exchange between the body and surrounding environment as a function of uncontrolled heat transfer across the general body surface and regulated heat transfer accommodates different metabolic loads, external thermal loads, insulation, and evaporative water loss.

We have constructed the model in QUCS, an open source circuit simulation package similar to SPICE. This program has a facility called "Equation Defined Device" that allows you to add new circuit elements. We added an element that models pumped flow heat transfer. With that additional element, the program has all the features necessary to model almost any dynamic heat transfer system.

For simplicity the body is divided into two compartments, the core and the periphery. Each element has an associated heat capacity, conduction paths, and pumped flow paths to scenario specific and time varying boundary conditions. The flow rates are controlled by algorithms implemented as circuit elements in the model. Time varying exercise load is an input to the model. Because the model is implemented as a circuit diagram changes in parameters, and topology, can be very quickly implemented without coding. An initial version of the model is operational, and has produced physiologically plausible results in agreement with a small number of critical experiments. The model is being refined incrementally by comparison with additional experimental results.

The current version of the model has flow loops for the core, the periphery, the non-glabrous skin, the glabrous skin, and the heart and lungs (see Figure 1 below). Boundary conditions can simulate exercise load, ambient and AVA temperatures, evaporative cooling, and radiative cooling or heating.

Boundary conditions are controlled by time varying elements that can include arbitrary table driven profiles. Flows and flow switching are controlled by circuit elements in the control block.

All parameters in the model are observable. A typical configuration will have 45 measured parameters.

This model can accommodate different metabolic loads, external thermal loads, insulation, and evaporative water loss. All these parameters can be time varying.

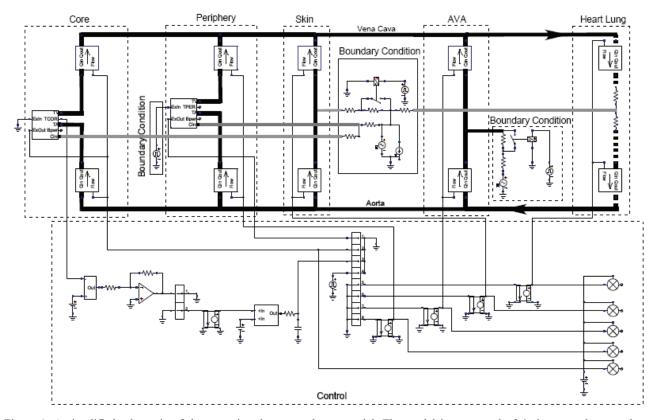


Figure 1. A simplified schematic of the emerging thermoregulatory model. The model is composed of 6 elements: the core thermal compartment (core), the peripheral thermal compartment (periphery), the non-glabrous skin (skin), the glabrous skin (AVA), the heart and pulmonary loop (heart lung), and a control system (control). The thick black lines indicate routs of circulation and, thus, circulatory system mediated heat transfer. The thick gray lines indicate paths of heat transfer to the environment. The control system drives the pump (heart) and blood flow rates to the various elements based on the demands of the system as programmed into the simulation. Boundary condition blocks: left block - exercise input, middle and right blocks - environment.

This model is still a work in progress. Once the model is fully operational and has been validated against the empirical data generated in the laboratory, we will conduct simulations of various experimental conditions, such as: 1) modesty clothed subject resting in a thermoneutral environment with a thermal load or sink applied to one or more of the non-hairy skin surfaces, 2) heavily insulated subject performing fixed load treadmill in a hot environment, and 3) subjects immersed in a temperaturecontrolled circulating water bath.

## References

Wissler, E. H. 2008. A quantitative assessment of skin blood flow in humans. *Eur J Appl Physiol*. 104:145-157.

Nelson D. A., Charbonnel S., Curran A. R., Marttila E. A., Fiala D., Mason P. A., and Ziriax J.M. 2009. A high-resolution voxel model for predicting local tissue temperatures in humans subjected to warm and hot environments. *J Biomech Eng.* 131(4):041003.

Yokota M., Berglund L., Cheuvront S., Santee W., Latzka W., Montain S., Kolka M., and Moran D. 2008. Thermoregulatory model to predict physiological status from ambient environment and heart rate. *Comput Biol Med.* 38(11-12):1187-93. Rowell, L. B. 1974, Human cardiovascular adjustments to exercise and thermal stress. *Physiol Rev.* 54(1):75-159.

Sangiorgi, S., Manelli, A., Congiu, T., Bini, A., Pilato, G., Reguzzoni, M., and Raspanti, M., 2004, Microvascularization of the human digit as studied by corrosion casting. *J Anat.* 204(2):123-131.

Manelli, A., Sangiorgi, S., Ronga, M., Reguzzoni, M., Bini, A., and Raspanti, M. 2005. Plexiform vascular structures in the human digital dermal layer: a SEM--corrosion casting morphological study. *Eur J Morphol.* 42(4-5):173-177.

Yamazaki, F., and Sone, R. 2006, Different vascular responses in glabrous and nonglabrous skin with increasing core temperature during exercise. *Eur J Appl Physiol.* 97(5):582-590.