

A Macroscopic Model for Countercurrent Bioheat Transfer in a Circulatory System

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ABSTRACT

The volume averaging theory of porous media has been applied to obtain a general set of macroscopic governing equations for countercurrent bioheat transfer between terminal arteries and veins in the circulatory system. Capillaries providing a continuous connection between the countercurrent terminal arteries and veins are modeled, introducing the perfusion bleed-off rate. Three distinctive energy equations are derived for the arterial blood phase, venous blood phase, and tissue phase. It has been found that the resulting model, under appropriate conditions, naturally reduces to those introduced by Chato, Bejan, Weinbaum and Jiji, and others for countercurrent heat transfer for the case of closely aligned pairs of vessels. A useful expression for the longitudinal effective thermal conductivity for the tissue has been derived without dropping the perfusion source terms. The expression turns out to be quite similar to Bejan's and Weinbaum and Jiji's expressions. Furthermore, the effect of spatial distribution of perfusion bleed-off rate on total countercurrent heat transfer has been investigated in depth exploiting the present bioheat transfer model.

NOMENCLATURE

<p>A surface area (m^2)</p> <p>A_{int} interface between the fluid and solid (m^2)</p> <p>a_f specific surface area ($1/m$)</p> <p>c_p specific heat at constant pressure (J/kgK)</p> <p>h_f interfacial heat transfer coefficient (W/m^2K)</p> <p>k thermal conductivity (W/mK)</p> <p>K_{ij} permeability tensor (m^2)</p> <p>u_i, u unit vector pointing outward from the fluid side to solid side</p> <p>p pressure (Pa)</p> <p>u_i, u metabolic reaction rate (W/m^3)</p> <p>T temperature (K)</p> <p>u_i, u velocity vector (m/s)</p> <p>V representative elementary volume (m^3)</p> <p>x, y Cartesian coordinates (m)</p>	<p>Greek Symbols</p> <p>α thermal diffusivity (m^2/s)</p> <p>ε porosity (-)</p> <p>ν kinematic viscosity (m^2/s)</p> <p>$\sigma_k, \sigma_\varepsilon$ density (kg/m^2)</p> <p>$\sigma_k, \sigma_\varepsilon$ perfusion bleed-off rate (1/s)</p> <p>Subscripts and Superscripts</p> <p>a artery</p> <p>dis dispersion</p> <p>f fluid</p> <p>s solid</p> <p>v vein</p> <p>Special Symbols</p> <p>$\tilde{\phi}$ deviation from intrinsic average</p> <p>$\langle \phi \rangle$ volume average</p> <p>$\langle \phi \rangle^{f,s,a,v}$ intrinsic average</p>
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1. INTRODUCTION

Bazett and colleagues (1948a,b) conducted a series of experimental studies on countercurrent heat exchange in the circulatory system. They found that the axial temperature gradient in the limb artery of humans, under conditions of very low ambient temperature, is an order of magnitude higher than under normal ambient conditions. From these experimental observations, they proposed the concept of venous shunting to the periphery, namely, that the countercurrent heat transfer takes place in the deep vasculature at the same time the blood is directed to the cutaneous circulation in close proximity to the surroundings. Their experimental finding brought attention to the important role of countercurrent heat exchange in bioheat transfer. Especially when the anatomical configuration of the main supply artery and vein in the limbs is treated, the effect of countercurrent heat transfer between closely spaced arteries and veins in the tissue must be taken into full consideration.

Following the experimental studies conducted by Bazett and colleagues (1948a,b), Scholander and Krog (1957) and Mitchell and Myers (1968) investigated such an effect and successfully demonstrated that the countercurrent heat exchange reduces heat loss from the extremity to the surroundings, which could be quite significant due to a large surface-to-volume ratio. These models, however, were not able to take account of either metabolic reaction or perfusion bleed-off from the artery to vein. Keller and Seiler (1971) established a one-dimensional bioheat transfer model to include the countercurrent heat transfer for the subcutaneous tissue region with arteries, veins, and capillaries. Weinbaum and Jiji (1979, 1985) proposed a model, which is based on some anatomical understanding, considering the countercurrent arteriovenous vessels. Roetzel and Xuan (1998) pointed out that the model may be useful in describing a temperature field in a single organ but would not be convenient to apply to the whole thermoregulation system. The foregoing survey prompts us to establish

a multidimensional model that can be applied to the regions of extremity, where the countercurrent heat transfer between closely spaced arteries and veins in the blood circulatory system occurs. Excellent reviews on these bioheat transfer equations may be found in Chato (1980), Charny (1992), and Khaled and Vafai (2003).

Xuan and Roetzel (1997), Khaled and Vafai (2003), and Khanafer and Vafai (2006) stress the advantages in appealing to the theory of porous media. In this study, we appeal to a general bioheat transfer model based on the volume averaging theory of porous media, recently introduced by Nakayama and Kuwahara (2008). We shall extend this procedure for the case of countercurrent bioheat transfer in a blood circulatory system. The set of macroscopic governing equations consists of continuity and momentum equations for both arterial and venous blood phases and three individual energy equations for the two blood phases and the tissue phase. It will be shown that most shortcomings in existing models are overcome in the present model. Capillaries providing a continuous connection between the countercurrent terminal arteries and veins are modeled introducing the perfusion bleed-off rate, originally introduced in the pioneering article by Pennes (1948). It has been found that the resulting model under certain conditions reduces to existing models for countercurrent heat transfer such as Chato (1980), Bejan (1979), Keller and Seiler (1971), Roetzel and Xuan (1998), and Weinbaum and Jiji (1985) for the case of closely aligned pairs of artery and vein. A general expression has been presented for the longitudinal effective thermal conductivity in the energy equation for the tissue. To examine the present model, we shall apply it to the countercurrent blood vessel configuration examined by Chato. While Chato assumed the constancy of the perfusion bleed-off rate, we shall allow the spatial distribution of the perfusion bleed-off rate and investigate its effect on the total countercurrent heat transfer.

2. VOLUME AVERAGING PROCEDURE

We shall treat the whole anatomical structure as a fluid-saturated porous medium, through which the blood infiltrates, and try to apply the principle of heat and fluid flow in a fluid-saturated porous medium to derive a set of the volume-averaged governing equations for the bioheat transfer and blood flow. A schematic view of the tissue layer close to the skin surface is shown in Fig. 1, in which the arteries and veins are paired such that the countercurrent heat transfer takes place. Thus we assign individual dependent variables, such as temperature, to the arterial blood, venous blood, and tissue, which leads us to propose a three-energy equation model.

In an anatomical view, three compartments are identified in the biological tissues, namely, blood vessels, cells, and interstitium, as illustrated in Fig. 2.

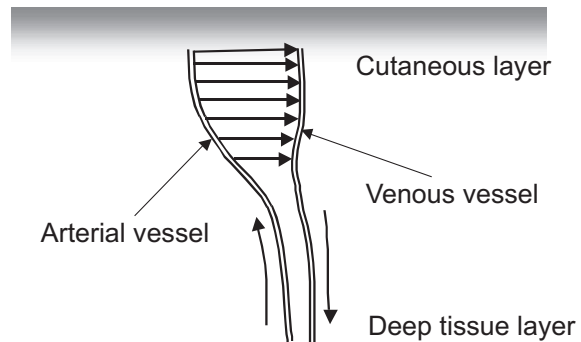


Figure 1. Schematic view of countercurrent heat exchange near the skin surface

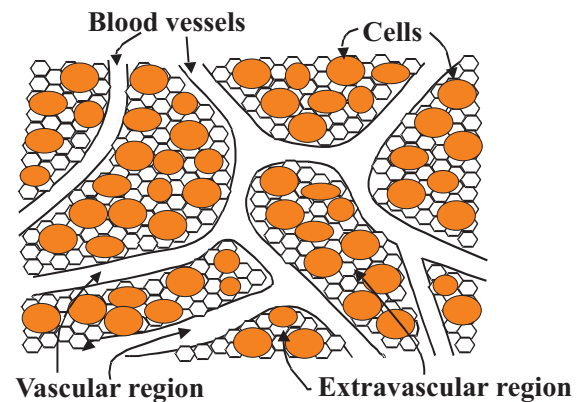


Figure 2. Schematic view of biological tissue

The interstitial space can be further divided into the extracellular matrix and the interstitial fluid. However, for sake of simplicity, we divide the biological tissue into two distinctive regions, namely, the vascular region and the extravascular region (i.e., cells and the interstitium), and treat the whole anatomical structure as a fluid-saturated porous medium, through which the blood infiltrates. The extravascular region is regarded as a solid matrix (although the extravascular fluid is present) and will be simply referred to as the “tissue” region to differentiate it from the “blood” region.

In what follows, we shall introduce the principle of heat and fluid flow in a fluid-saturated porous medium to derive a set of the volume-averaged governing equations for the bioheat transfer and blood flow. A general bioheat transfer model based on the volume averaging theory of porous media, recently introduced by Nakayama and Kuwahara (2008), may readily be extended for the case of countercurrent bioheat transfer in a blood circulatory system. The blood perfusion heat source term will be identified as an extra surface integral term resulting from changing the sequence of integration and derivation, as we obtain the macroscopic energy equation by integrating the microscopic convection term within a local control volume.

Let us consider a local control volume V in a fluid-saturated porous medium, as shown in Fig. 3, whose

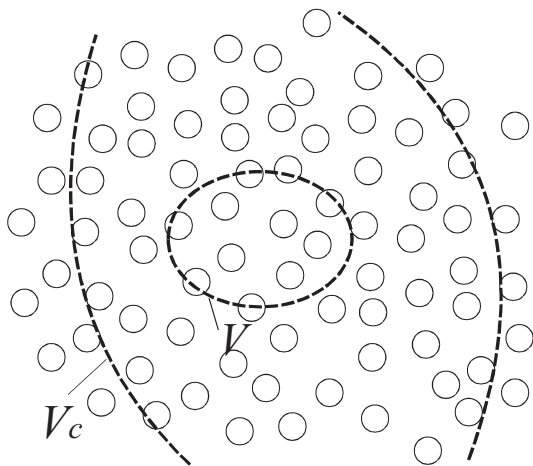


Figure 3. Control volume in a porous medium

length scale $V^{1/3}$ is much smaller than the macroscopic characteristic length $V_c^{1/3}$, but, at the same time, much greater than the microscopic characteristic length (see, e.g., Cheng, 1978; Nakayama, 1995). Under this condition, the volume average of a certain variable φ is defined as

$$\langle \varphi \rangle \equiv \frac{1}{V} \int_{V_f} \varphi dV \quad (1)$$

Another average, namely, intrinsic average, is given by

$$\langle \varphi \rangle^f \equiv \frac{1}{V_f} \int_{V_f} \varphi dV \quad (2)$$

where V_f is the volume space that the fluid (blood) occupies. Obviously, two averages are related as $\langle \varphi \rangle = \varepsilon \langle \varphi \rangle^f$, where $\varepsilon \equiv V_f/V$ is the local porosity, namely, the volume fraction of the vascular space, which is generally less than 0.1.

Following Cheng (1978), Vafai and Tien (1981), Nakayama (1995), Quintard and Whitaker (1993), and many others, we decompose a variable into its intrinsic average and the spatial deviation from it:

$$\varphi = \langle \varphi \rangle^f + \tilde{\varphi} \quad (3)$$

All dependent variables in the microscopic governing equations for the arterial blood, venous blood, and tissue phases are decomposed in this manner, and then these governing equations are integrated over the local control volume. After some manipulations, following Nakayama and Kuwahara (2008), we obtain the volume-averaged set of the governing equations, which can be written assigning the subscripts a , v , and s to arterial blood vessels (arteries and arterioles), venous blood vessels (veins and venules), and tissue, as follows:

For the Arterial Blood Phase:

$$\frac{\partial \varepsilon_a \langle u_j \rangle^a}{\partial x_j} + \omega'_a = 0 \quad (4)$$

$$-\frac{1}{\rho} \frac{\partial \langle p \rangle^a}{\partial x_i} - \frac{\nu}{K_{a_{ij}}} \varepsilon_a \langle u_j \rangle^a - \omega'_a u_{i_{\text{int}}} = 0 \quad (5)$$

$$\begin{aligned} & \varepsilon_a \rho_f c_{p_f} \frac{\partial \langle T \rangle^a}{\partial t} + \rho_f c_{p_f} \frac{\partial}{\partial x_j} \varepsilon_a \langle u_j \rangle^a \langle T \rangle^a \\ &= \frac{\partial}{\partial x_j} \left(\varepsilon_a k_a \frac{\partial \langle T \rangle^a}{\partial x_j} + \varepsilon_a k_{\text{dis}_{a_j k}} \frac{\partial \langle T \rangle^a}{\partial x_k} \right) \\ & - a_a h_a (\langle T \rangle^a - \langle T \rangle^s) - \rho_f c_{p_f} \omega'_a \langle T \rangle^a \end{aligned} \quad (6)$$

For the Venous Blood Phase:

$$\frac{\partial \varepsilon_v \langle u_j \rangle^v}{\partial x_j} + \omega'_v = 0 \quad (7)$$

$$-\frac{1}{\rho} \frac{\partial \langle p \rangle^v}{\partial x_i} - \frac{\nu}{K_{v_{ij}}} \varepsilon_v \langle u_j \rangle^v - \omega'_v u_{i_{\text{int}}} = 0 \quad (8)$$

$$\begin{aligned} & \varepsilon_v \rho_f c_{p_f} \frac{\partial \langle T \rangle^v}{\partial t} + \rho_f c_{p_f} \frac{\partial}{\partial x_j} \varepsilon_v \langle u_j \rangle^v \langle T \rangle^v \\ &= \frac{\partial}{\partial x_j} \left(\varepsilon_v k_v \frac{\partial \langle T \rangle^v}{\partial x_j} + \varepsilon_v k_{\text{dis}_{v_j k}} \frac{\partial \langle T \rangle^v}{\partial x_k} \right) \\ & - a_v h_v (\langle T \rangle^v - \langle T \rangle^s) - \rho_f c_{p_f} \omega'_v \langle T \rangle^v \end{aligned} \quad (9)$$

For the Solid Tissue Phase:

$$\begin{aligned} & (1 - \varepsilon) \rho_s c_s \frac{\partial \langle T \rangle^s}{\partial t} = \frac{\partial}{\partial x_j} \left((1 - \varepsilon) k_s \frac{\partial \langle T \rangle^s}{\partial x_j} \right) \\ & + a_a h_a (\langle T \rangle^a - \langle T \rangle^s) + \rho_f c_{p_f} \omega'_a \langle T \rangle^a + a_v h_v \\ & \times (\langle T \rangle^v - \langle T \rangle^s) + \rho_f c_{p_f} \omega'_v \langle T \rangle^v + (1 - \varepsilon) S_m \end{aligned} \quad (10)$$

where ε_a and ε_v are the volume fractions of the arterial blood and that of the venous blood, respectively, such that $\varepsilon = \varepsilon_a + \varepsilon_v$. The terms associated with the surface integral are modeled as

$$\begin{aligned} & \frac{1}{V_f} \int_{A_{\text{int}}} \left(-\frac{p}{\rho_f} + \nu_f \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) \right) n_j dA \\ &= -\frac{\nu_f}{K_{ij}} \varepsilon \langle u_j \rangle^f \end{aligned} \quad (11)$$

which is simply Darcy's law, and

$$\int_{A_{\text{int}}} \rho_f u_j n_j dA/V = \rho_f \omega' \quad (12)$$

is the mass flow rate per a unit volume through the interface A_{int} , modeled in terms of the perfusion bleed-off rate ω' (1/s). The perfusion bleed-off rate ω' describes the volume rate of the fluid per a unit volume, bleeding off to the solid matrix through the interfacial vascular wall. Thus the momentum bleed-off rate is modeled as

$$\int_{A_{\text{int}}} \rho_f u_i u_j n_j dA/V = \rho_f \omega' u_{i_{\text{int}}} \quad (13a)$$

where $u_{i_{\text{int}}}$ is the velocity vector averaged over the interface. Likewise, the enthalpy bleed-off rate is modeled as

$$\int_{A_{\text{int}}} \rho_f c_{p_f} u_j T n_j dA/V = \rho_f c_{p_f} \omega' \langle T \rangle^f \quad (13b)$$

For the interfacial heat transfer, Newton's cooling law is adopted as

$$\frac{1}{V} \int_{A_{\text{int}}} k_f \frac{\partial T}{\partial x_j} n_j dA = a_f h_f (\langle T \rangle^s - \langle T \rangle^f) \quad (14)$$

where a_f and h_f are the specific surface area and interfacial heat transfer coefficient, respectively. Furthermore, $k_{\text{dis}_{j k}}$ is the thermal dispersion conductivity tensor, as introduced by Nakayama et al. (2006).

For the microcirculation of peripheral tissue in which capillaries provide a continuous connection between the terminal artery and vein (i.e., arterial-venous anastomoses), as shown in Fig. 1, we may readily set $\omega'_a = -\omega'_v$ such that the present energy Eq. (10) for the solid tissue phase reduces to

$$\begin{aligned} & (1 - \varepsilon) \rho_s c_s \frac{\partial \langle T \rangle^s}{\partial t} = \frac{\partial}{\partial x_j} \left((1 - \varepsilon) k_s \frac{\partial \langle T \rangle^s}{\partial x_j} \right) \\ & + a_a h_a (\langle T \rangle^a - \langle T \rangle^s) + a_v h_v (\langle T \rangle^v - \langle T \rangle^s) \\ & + \rho_f c_{p_f} \omega'_a (\langle T \rangle^a - \langle T \rangle^v) + (1 - \varepsilon) S_m \end{aligned} \quad (15)$$

3. COMPARISON OF PRESENT AND EXISTING BIOHEAT TRANSFER MODELS

Most existing bioheat transfer models for countercurrent bioheat transfer already reside in the present model based on the theory of porous media. Let us revisit some of the existing models and try to generate them from the present general model.

3.1. Keller and Seiler Model

Keller and Seiler (1971) noted that the axial temperature gradient in the limb is much higher than the transverse one and considered an energy balance within a control volume for the idealized one-dimensional steady case, as illustrated in Fig. 4, for which they proposed

$$\begin{aligned}
 (1 - \varepsilon) k_s \frac{d^2 \langle T \rangle^s}{dx^2} + a_a h_a (\langle T \rangle^a - \langle T \rangle^s) \\
 + a_v h_v (\langle T \rangle^v - \langle T \rangle^s) + \rho_f c_{p_f} \omega' (\langle T \rangle^a - \langle T \rangle^s) \\
 + (1 - \varepsilon) S_m = 0
 \end{aligned} \tag{16}$$

which is almost identical to what we would get for the one-dimensional case from our multidimensional expression (15), except that the temperature difference in the perfusion term somewhat differs from ours. Keller and Seiler obtained solutions assuming that the arterial blood enters the peripheral region at the isothermal core temperature and that the venous

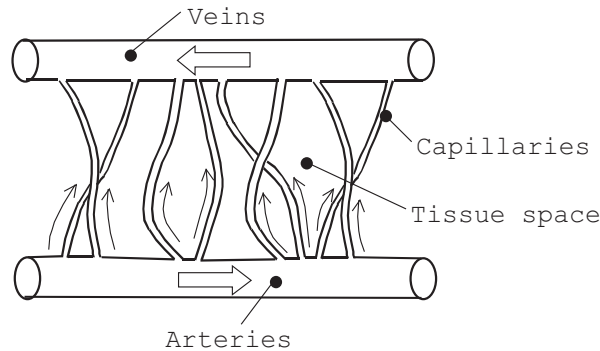


Figure 4. One-dimensional model for countercurrent heat exchange

blood is completely equilibrated with the tissue at the cutaneous layer.

3.2. Chato Model

Chato’s (1980) countercurrent heat transfer model differs from Keller and Seiler (1971) in its neglect of heat transfer between the blood and tissue. In this way, he was able to concentrate on the two temperatures, instead of three, as in Keller and Seiler. Chato assumes the flow rate decreases linearly, which corresponds with the case of a constant perfusion bleed-off rate. His one-dimensional model can easily be generated from our general expressions (6) and (9) along with (4) and (7), dropping the transient and conduction terms, as

$$\begin{aligned}
 \rho_f c_{p_f} \frac{d}{dx} \varepsilon_a \langle u \rangle^a \langle T \rangle^a = -a_f h_f (\langle T \rangle^a - \langle T \rangle^v) \\
 - \rho_f c_{p_f} \omega'_a \langle T \rangle^a
 \end{aligned} \tag{17}$$

$$\begin{aligned}
 \rho_f c_{p_f} \frac{d}{dx} \varepsilon_v \langle u \rangle^v \langle T \rangle^v = -a_f h_f (\langle T \rangle^v - \langle T \rangle^a) \\
 + \rho_f c_{p_f} \omega'_a \langle T \rangle^a
 \end{aligned} \tag{18}$$

where the interfacial heat transfer coefficients are assumed to be the same. The continuity Eqs. (4) and (7) readily provide

$$\varepsilon_a \langle u \rangle^a = u_0 - \omega'_a x \tag{19}$$

$$\varepsilon_v \langle u \rangle^v = -u_0 + \omega'_a x \tag{20}$$

Note that u_0 is the apparent velocity at $x = 0$ and that the right-hand side terms in the two Eqs. (17) and (18) cancel out each other, as they should for this “perfect” heat exchange system. Chato obtained arterial and venous temperature profiles along the length of the vessels and demonstrated that the effect of perfusion bleed-off is to increase the heat transfer between the vessels as compared with the case of constant mass flow rate (i.e., $\omega'_a = 0$).

3.3. Roetzel and Xuan Model

Roetzel and Xuan (1998) used the theory of porous media to simulate a transient response of the limb to external stimulus, in which the effect of the counter-current heat exchange on the temperature response is expected to be significant. Their energy equation for the tissue in our notation runs as

$$\begin{aligned} (1 - \varepsilon) \rho_s c_s \frac{\partial \langle T \rangle^s}{\partial t} &= \frac{\partial}{\partial x_j} \left((1 - \varepsilon) k_s \frac{\partial \langle T \rangle^s}{\partial x_j} \right) \\ &+ a_a h_a (\langle T \rangle^a - \langle T \rangle^s) + a_v h_v (\langle T \rangle^v - \langle T \rangle^s) \\ &+ (1 - \varepsilon) S_m \end{aligned} \quad (21)$$

Comparison of the foregoing equation against our expression (15) for the tissue reveals that the perfusion term $\rho_f c_{pf} \omega'_a (\langle T \rangle^a - \langle T \rangle^v)$ is missing. Obviously, they did not retain the term describing the transcappillary fluid exchange via arterial-venous anastomoses, namely, $\int_{A_{\text{int}}} \rho_f c_{pf} u_j T n_j dA/V = \rho_f c_{pf} \omega' \langle T \rangle^f$. If they did, they would have obtained our expression (15), which may be rearranged in their form as

$$\begin{aligned} (1 - \varepsilon) \rho_s c_s \frac{\partial \langle T \rangle^s}{\partial t} &= \frac{\partial}{\partial x_j} \left((1 - \varepsilon) k_s \frac{\partial \langle T \rangle^s}{\partial x_j} \right) \\ &+ (a_a h_a + \rho_f c_{pf} \omega'_a) (\langle T \rangle^a - \langle T \rangle^s) + (a_v h_v \\ &- \rho_f c_{pf} \omega'_a) (\langle T \rangle^v - \langle T \rangle^s) + (1 - \varepsilon) S_m \end{aligned} \quad (22)$$

In their model, the convection-perfusion parameters, namely, $(a_f h_f \pm \rho_f c_{pf} \omega')$, are replaced by the interfacial convective heat transfer coefficients, $a_f h_f$. This difference should not be overlooked since the perfusion heat sources could be quite significant for the bioheat transfer in the extremities, as Chato (1980) demonstrated using his model.

3.4. Weinbaum-Jiji Model and Bejan Model

Weinbaum and Jiji (1979) considered bioheat transfer between a paired countercurrent terminal artery and vein. They took account of the vascular structure in which vessel number density, velocity, and diameter vary significantly from the deep tissue layer

toward the skin layer. Later, Weinbaum and Jiji (1985) proposed a simplified model in which an effective thermal conductivity tensor is introduced as a function of the local blood velocity. They claimed that the perfusion heat source vanishes within the capillary bed and derived a single equation to describe the steady state tissue temperature variations, which, when the vessels are in parallel to the temperature gradient, reduces to

$$\begin{aligned} \frac{d}{dx} \left(\left((1 - \varepsilon) k_s + \frac{\pi \varepsilon_a (\rho_f c_{pf} \langle u \rangle^a R)^2}{2\sigma (1 - \varepsilon) k_s} \right) \frac{d \langle T \rangle^s}{dx} \right) \\ + (1 - \varepsilon) S_m = 0 \end{aligned} \quad (23)$$

where σ is a geometrical factor of the vessel structure, whereas R is the local radius of the vessel. It is seen that the longitudinal effective thermal conductivity due to countercurrent flow is proportional to the square of blood mass flow rate. It is also interesting to note that the concept of the longitudinal effective thermal conductivity in countercurrent heat transfer was already explicit in Bejan (1979; A. Bejan, pers. comm., 2007), in which he presented a novel method for thermal insulation system optimization. Bejan (1979) seems to be the first to point out the relationship associated with the square of the mass flow rate and the longitudinal effective thermal conductivity by convection. His expression is a simple one:

$$Q = - \frac{(\dot{m}_f c_{pf})^2}{UP} \frac{d \langle T \rangle^s}{dx} \quad (24)$$

where Q , \dot{m}_f , U , and P are the heat flow from the warm end to the cold end, the mass flow rate of the hot (or cold) fluid, the overall heat transfer coefficient, and the wetted perimeter, respectively. The group $(\dot{m}_f c_{pf})^2 / (UP)$ plays the same role as Ak_{eff} in the one-dimensional insulation system. On noting that $\dot{m}_f = A \rho_f \varepsilon_a \langle u \rangle^a$ and $P = A a_f$, Bejan's Eq. (24) may be translated in the present bioheat transfer problem as

$$\frac{d}{dx} \left(\left((1 - \varepsilon) k_s + \frac{(\rho_f c_{pf} \varepsilon_a \langle u \rangle^a)^2}{a_f U} \right) \frac{d \langle T \rangle^s}{dx} \right) + (1 - \varepsilon) S_m = 0 \tag{25}$$

In these countercurrent heat transfer models, namely, Bejan's and Weinbaum and Jiji's, the perfusion heat sources are ignored. Thus, in what follows, we shall attempt to reduce the present set of governing equations to a single equation for the tissue temperature variations, without neglecting these perfusion heat source terms.

When the blood flow is strong enough to neglect the macroscopic diffusion, the energy Eqs. (6) and (9) for arterial and venous blood flows for the one-dimensional steady state reduce to

$$\rho_f c_{pf} \frac{d}{dx} \varepsilon_a \langle u \rangle^a \langle T \rangle^a = -a_f h_f (\langle T \rangle^a - \langle T \rangle^s) - \rho_f c_{pf} \omega'_a \langle T \rangle^a \tag{26}$$

$$\rho_f c_{pf} \frac{d}{dx} \varepsilon_v \langle u \rangle^v \langle T \rangle^v = -a_f h_f (\langle T \rangle^v - \langle T \rangle^s) + \rho_f c_{pf} \omega'_a \langle T \rangle^v \tag{27}$$

where the interfacial heat transfer coefficients are assumed to be the same as in the case of Chato. However, the foregoing equations are different from Chato's Eqs. (17) and (18), since we do take account of the heat transfer between the bloods and tissue. On noting the continuity relationship $\varepsilon_a \langle u \rangle^a = -\varepsilon_v \langle u \rangle^v$, as given by the continuity Eqs. (4) and (7), with $\omega'_a = -\omega'_v$, we subtract Eq. (27) from (26) to obtain

$$\rho_f c_{pf} \frac{d}{dx} \varepsilon_a \langle u \rangle^a (\langle T \rangle^a + \langle T \rangle^v) = -a_f h_f \times (\langle T \rangle^a - \langle T \rangle^v) - \rho_f c_{pf} \omega'_a (\langle T \rangle^a + \langle T \rangle^v) \tag{28a}$$

or

$$\rho_f c_{pf} \varepsilon_a \langle u \rangle^a \frac{d}{dx} (\langle T \rangle^a + \langle T \rangle^v) = -a_f h_f \times (\langle T \rangle^a - \langle T \rangle^v) \tag{28b}$$

as we note the continuity relationship, namely, $d(\varepsilon_a \langle u \rangle^a)/dx = -\omega'_a$. Weinbaum and Jiji (1985)

proposed that the mean tissue temperature around an artery-vein pair can be approximated as

$$\langle T \rangle^s = \frac{\langle T \rangle^a + \langle T \rangle^v}{2} \tag{29}$$

Following their approximation, we obtain

$$\langle T \rangle^a - \langle T \rangle^v = -2 \frac{\rho_f c_{pf} \varepsilon_a \langle u \rangle^a}{a_f h_f} \frac{d \langle T \rangle^s}{dx} \tag{30}$$

from Eq. (28b). Using Eqs. (26) and (27), we may replace both interfacial and perfusion heat source terms in the energy Eq. (15) for the tissue by the blood convection terms as

$$\begin{aligned} & \frac{d}{dx} \left((1 - \varepsilon) k_s \frac{d \langle T \rangle^s}{dx} \right) + a_a h_a (\langle T \rangle^a - \langle T \rangle^s) \\ & + a_v h_v (\langle T \rangle^v - \langle T \rangle^s) + \rho_f c_{pf} \omega'_a (\langle T \rangle^a - \langle T \rangle^v) \\ & + (1 - \varepsilon) S_m = \frac{d}{dx} \left((1 - \varepsilon) k_s \frac{d \langle T \rangle^s}{dx} \right) - \rho_f c_{pf} \frac{d}{dx} \\ & \times (\varepsilon_a \langle u \rangle^a \langle T \rangle^a + \varepsilon_v \langle u \rangle^v \langle T \rangle^v) + (1 - \varepsilon) S_m = 0 \end{aligned} \tag{31}$$

As we note the continuity relationship $\varepsilon_a \langle u \rangle^a = -\varepsilon_v \langle u \rangle^v$ and use Eq. (30) for the last expression in Eq. (31), we finally have

$$\begin{aligned} & \frac{d}{dx} \left(\left((1 - \varepsilon) k_s + 2 \frac{(\rho_f c_{pf} \varepsilon_a \langle u \rangle^a)^2}{a_f h_f} \right) \frac{d \langle T \rangle^s}{dx} \right) \\ & + (1 - \varepsilon) S_m = 0 \end{aligned} \tag{32}$$

which we find almost identical to Bejan's Eq. (25), as we note the overall heat transfer coefficient corresponds to

$$U = \frac{1}{\frac{1}{h_a} + \frac{1}{h_v}} = \frac{h_f}{2} \tag{33}$$

It is most interesting to find that the foregoing relationship for the longitudinal effective thermal conductivity holds for all cases, with or without perfusion bleed-off sources, as long as the local values are used to evaluate the effective thermal conductivity by convection.

4. EFFECT OF SPATIAL DISTRIBUTION OF PERFUSION BLEED-OFF RATE ON TOTAL COUNTERCURRENT HEAT TRANSFER

As an example for illustration, we shall consider Chato's (1980) one-dimensional problem of counter-current heat transfer, as schematically shown in Fig. 4. Chato assumed the constancy of the perfusion bleed-off rate ω'_a , namely, a linear decrease in the arterial flow rate, and that all of the bleed-off fluid that leaves the artery reenters the vein at the same location. We shall relax his assumption, allowing the spatial variation of ω'_a so as to investigate its effect on the total countercurrent heat transfer. Let us assume that the perfusion bleed-off rate ω'_a follows

$$\omega'_a = (1 + n) \bar{\omega}'_a \left(\frac{x}{L}\right)^n \quad (34)$$

along the blood vessel of length L , where $\bar{\omega}'_a$ is the average perfusion rate such that the total amount of perfusion is given by $\bar{\omega}'_a L$, irrespective of the value of n . The exponent n may take any value equal to zero (i.e., Chato's case) or greater than zero such that we can compare the results against Chato's and elucidate the effect of blood pressure on the bioheat transfer for fixed total amount of perfusion. As we substitute the foregoing equation into the continuity Eqs. (4) and (7), we readily obtain

$$\varepsilon_a \langle u \rangle^a = u_0 - \bar{\omega}'_a L \left(\frac{x}{L}\right)^{1+n} \quad (35)$$

$$\varepsilon_v \langle u \rangle^v = -u_0 + \bar{\omega}'_a L \left(\frac{x}{L}\right)^{1+n} \quad (36)$$

where u_0 is the apparent blood velocity at $x=0$. As illustrated in Fig. 5, the exponent n controls the distribution of the perfusion rate. For a large exponent n , the perfusion bleed-off takes place rather suddenly toward the end of the vessel, indicating poor blood circulation.

On substituting these velocity distributions into the momentum Eqs. (5) and (8), we obtain

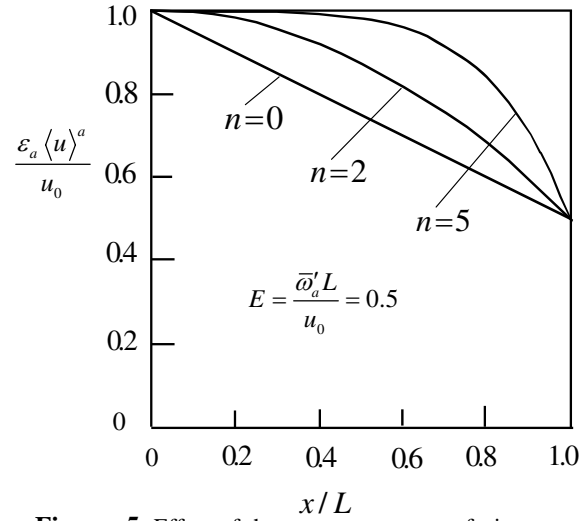


Figure 5. Effect of the exponent n on perfusion rate

$$\begin{aligned} \langle p \rangle|_{x=0} - \langle p \rangle|_{x=L} &= (\varepsilon_a \langle p \rangle^a + \varepsilon_v \langle p \rangle^v)|_L^0 \\ &= \mu \left(\frac{\varepsilon_a}{K_a} - \frac{\varepsilon_v}{K_v} \right) u_0 L \left(1 - \frac{E}{2+n} \right) \end{aligned} \quad (37)$$

where

$$E = \frac{\bar{\omega}'_a L}{u_0} \quad (38)$$

is the dimensionless perfusion bleed-off rate, while μ is the viscosity. Thus the pressure difference within the body may never be large since $\varepsilon_a/K_a \cong \varepsilon_v/K_v$. Equation (37), however, indicates that the blood pressure difference increases for either small perfusion bleed-off rate E or large exponent n , which may result from aging.

Following Chato (1980), we note that the axial conduction terms in the blood energy equations are negligibly small as compared with the convection and perfusion terms. Then, the energy Eqs. (17) and (18), along with the foregoing velocity distributions, reduce to

$$\frac{d \langle T \rangle^a}{d(x/L)} = - \frac{N}{1 - E \left(\frac{x}{L}\right)^{1+n}} (\langle T \rangle^a - \langle T \rangle^v) \quad (39)$$

$$\frac{d\langle T \rangle^v}{d(x/L)} = \frac{N+(1+n)E\left(\frac{x}{L}\right)^n}{1-E\left(\frac{x}{L}\right)^{1+n}} (\langle T \rangle^v - \langle T \rangle^a) \quad (40)$$

where

$$N = \frac{a_f h_f L}{\rho_f c_{pf} u_0} \quad (41)$$

is the number of heat transfer units. The boundary conditions are given by

$$x/L = 0 : \quad \langle T \rangle^a = \langle T \rangle_0^a \quad (42)$$

$$x/L = 1 : \quad \langle T \rangle^v = \langle T \rangle_L^v \quad (43)$$

A series of numerical integrations were carried out for various sets of three important dimensionless parameters, namely, the dimensionless perfusion rate E , the number of heat transfer units N , and the exponent n . Thus the temperature profiles along the vessel axes are obtained for the case of $n = 0$ and presented in Figs. 6a,b for a physiological range of E and N values. The results appear to be in perfect agreement with the exact expressions reported by Chato (1980). The difference between the present curves for $n = 0$ and those based on Chato's solution is indiscernible in the figure. Naturally, a better blood circulation (i.e., larger E) results in warming the venous blood efficiently. The figures show that its

efficiency as a heat exchanging system increases with N .

The temperature profiles along the vessel axes for the case of $n = 5$ are presented in Figs. 7a,b. It is interesting to note that the arterial blood temperature for the case of nonzero n always stays higher than that for the case of $E = 0$ (i.e., without perfusion), even at the end of the vessel. Following Chato, we shall evaluate the total heat transfer from the artery to vein in terms of

$$q_{a-v} = \rho c_{pf} u_0 (\langle T \rangle_0^v - \langle T \rangle_L^v) \quad (44)$$

or its dimensionless form, namely,

$$\frac{q_{a-v}}{\rho c_{pf} u_0 (\langle T \rangle_0^a - \langle T \rangle_L^v)} = \frac{\langle T \rangle_0^v - \langle T \rangle_L^v}{\langle T \rangle_0^a - \langle T \rangle_L^v} \quad (45)$$

The total heat transfer from the artery to vein is plotted against the exponent n for various sets of N and E values in Fig. 8. The total heat transfer decreases with increasing n (i.e., worsening the blood circulation), while it increases with E (i.e., increasing the perfusion rate).

5. CONCLUSIONS

The present model accounts for not only the tissue temperature field, but all velocity and temperature fields for the arterial blood, venous blood, and tissue

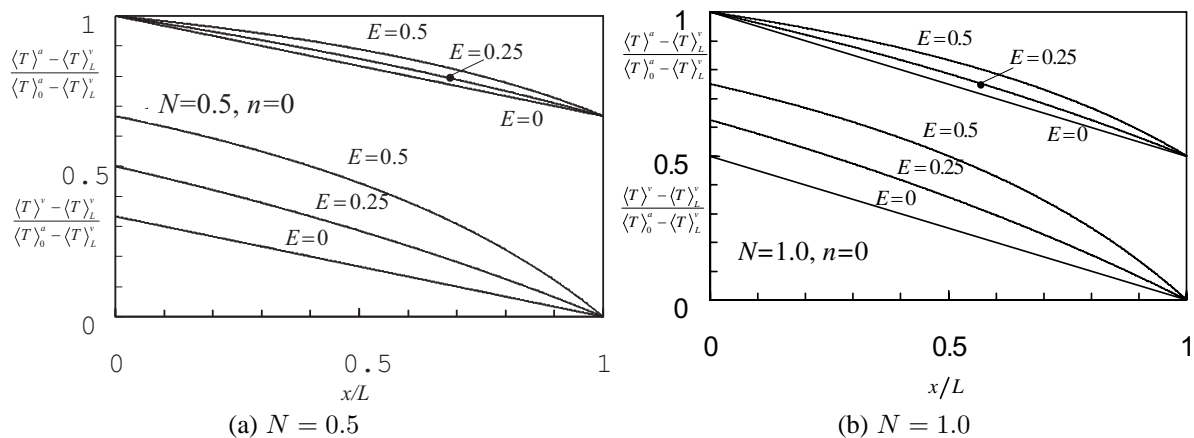


Figure 6. Arterial and venous blood temperature profiles along the vessel axes for the case of $n = 0$

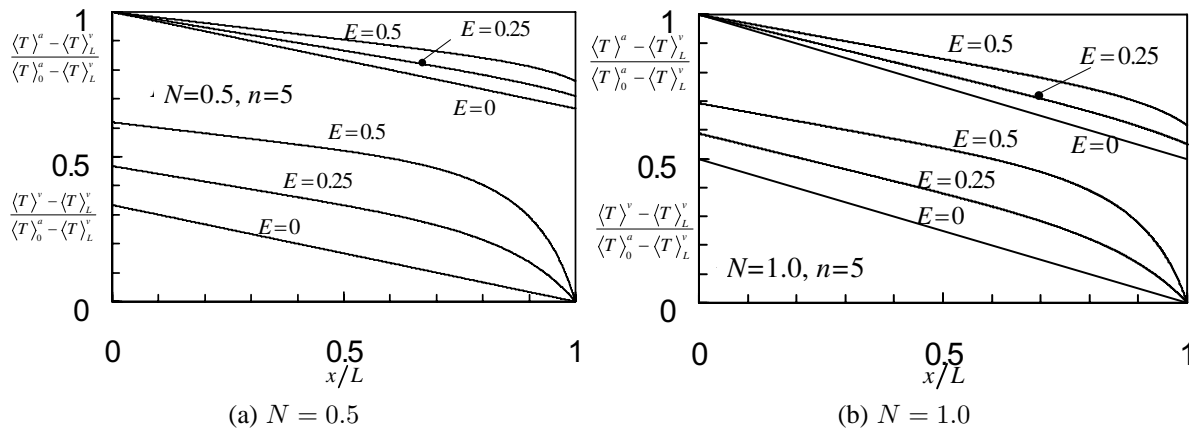


Figure 7. Arterial and venous blood temperature profiles along the vessel axes for the case of $n = 5$

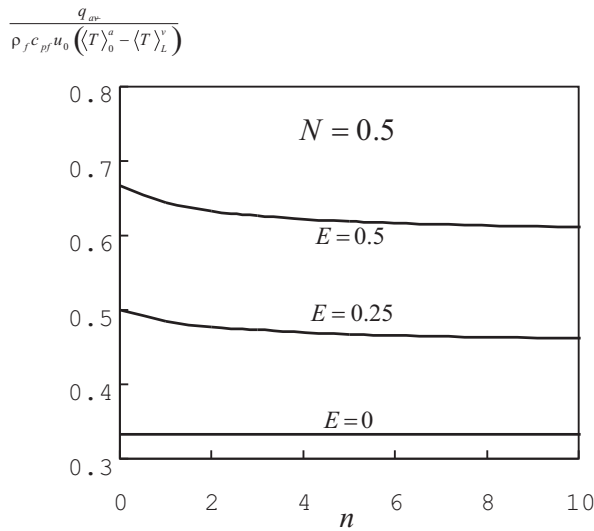


Figure 8. Effect of the exponent n on the total heat transfer from artery to vein

in the countercurrent blood circulatory system, and all these governing equations are coupled with one another in terms of blood perfusion bleed-off rates and interfacial heat transfer coefficients. The model based on the theory of anisotropic porous media provides a closed set of macroscopic governing equations for both velocity and temperature fields in intra- and extravascular phases, for the first time.

Existing models such as Chato (1980), Bejan (1979), Keller and Seiler (1971), and Weinbaum and Jiji (1985) were obtained by writing the present model for the idealized one-dimensional case. A useful ex-

pression for the longitudinal effective thermal conductivity for the tissue was derived without dropping the perfusion source terms. The effects of spatial distribution of perfusion bleed-off rate on total countercurrent heat transfer were investigated applying the present bioheat transfer model to Chato's one-dimensional problem. The present three-energy equation model in a multidimensional and anisotropic form is quite general and can be applied to the regions where the countercurrent heat transfer in the blood circulatory system plays an important role in the peripheral heat transfer from the extremity to the surroundings.

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