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Theoretical Evaluation of Contributions of Heat Conduction and Countercurrent Heat Exchange in Selective Brain Cooling in Humans

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Abstract—The purpose of this work is to evaluate the capacity of the heat loss from the carotid artery in the human neck and thus, to provide indirect evidence of the existence of selective brain cooling in humans during hyperthermia. A theoretical model is developed to describe the effects of blood flow rate and vascular geometry on the thermal equilibration in the carotid artery based on the blood flow and the anatomical vascular geometry in the human neck. The potential for cooling of blood in the carotid artery on its way to the brain by heat exchange with the jugular vein and by radial heat conduction loss to the cool neck surface is evaluated. It is shown that the cooling of the arterial blood can be as much as 1.1 °C lower than the body core temperature, which is in agreement with previous experimental measurements of the difference between the tympanic and body core temperatures. The model also evaluates the relative contributions of countercurrent heat exchange and radial heat conduction to selective brain cooling. It is found that these mechanisms are comparable with each other. Results of the present study will help provide a better understanding of the thermoregulation during hyperthermia. *© 2000 Biomedical Engineering Society.*
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Keywords—Selective brain cooling, Human neck, Bioheat transfer, Heat stress

INTRODUCTION

Selective cooling of the brain (SBC) during hyperthermia has been demonstrated to exist in dogs, antelopes, cats, sheep, rabbits, oxen, monkeys, and pigeons.[1] In most animals, panting provides powerful cooling of venous blood only a few millimeters from the brain.[12] The cool venous blood returns to the intracranium from the nasal evaporative surface and from other peripheral tissues of the head and thus cools the warm incoming arterial blood. A countercurrent heat exchange within the carotid *rete* is thought to be responsible for the cooling of the warm arterial blood.

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The human brain temperature cannot be measured directly unless under special circumstances. Thus, the question whether SBC also operates in humans, in which there is no cranial *rete*, has led to an interesting controversy.[3,6,18] The evidence of the existence of this mechanism relies on the experimental observations that the tympanic temperature (T_{ty}) was lower than the trunk (esophageal) temperature (T_{es}) in hyperthermic subjects.[7,11,15,19] Attention has been drawn to vascular arrangements that can serve for selective brain cooling in humans. Cabanac[6] pointed to several sites of intimate thermal contact between the warm arterial and the cold venous blood, e.g., the external jugular vein and external carotid artery, the internal jugular vein and common carotid artery.

Other investigators have argued that the tympanic temperature may be contaminated by the skin temperature and thus, may not be a good index of the brain temperature.[3,16] Theoretical analyses were performed to estimate the effect of countercurrent heat exchange on the extent of cooling in the artery before it reaches the base of the brain. Among these theoretical studies, Wenger[24] estimated the temperature decrease in the artery passing through the cavernous sinus using a simple heat transfer coefficient and concluded that in order to achieve a 1 °C drop in arterial temperature, the temperature difference between the arterial blood and the venous blood in the sinus is at least 119 °C. In another theoretical work by Nielsen,[15] the temperature drop in the artery was found to be less than 0.02 °C per °C difference between the arterial and venous temperatures. Probably the most complicated theoretical model was developed by Nunneley and Nelson,[18] in which the heat transfer rate between the countercurrent artery and vein was predicted using the ''effectiveness number of transfer units'' method for heat exchanger analysis. Under the most optimistic set of assumptions, the carotid artery temperature would be lowered by less than 0.05 °C per °C difference between the arterial and venous temperatures. These theoretical calculations seemed to be contrary to the ex-

perimental observations that the tympanic temperature can be as much as 1.2 °C lower than the body core temperature during outdoor exercise.[15] It is either that the tympanic temperature cannot represent the brain temperature or there are additional heat loss pathways which were not accounted for in these theoretical analyses. Whether the tympanic temperature can reflect changes in cerebral temperature is still controversial.[13,16,17] It seems that more experimental studies are needed to examine the relationship between T_{ty} and the brain temperature.

Examining all these theoretical analyses, one notes that the estimation of the temperature decay in the artery was primarily due to the heat loss to the countercurrent vein. None of the investigations took into account the possible heat loss from the countercurrent vessel pair to the surrounding tissue, such as the heat loss to the neck especially when the neck is exposed to cold, dry moving air. We suspect that the heat loss to the neck surface is of great significance in SBC.

In this paper, a theoretical model is developed to study the thermal equilibration in the countercurrent vessel pair based on the analyses of Wu *et al.*[25] and Weinbaum *et al.*[23] Wu *et al.*[25] presented an analytical solution-based approach for treating any finite number of vessels arbitrarily positioned in a tissue cylinder with surface convection. Later, Weinbaum *et al.*[23] modified this approach to model the thermal interaction between a countercurrent vessel pair with capillary bleed-off and conduction to the surrounding tissue cylinder. While the original solutions in Wu *et al.*[25] and Weinbaum *et al.*[25] were developed to describe countercurrent flow in human limb and skeletal muscle tissue cylinders, respectively, the general approach can be applied directly, as long as the main assumptions made in those previous studies are valid in the present situation. The solution is used to more realistically estimate the temperature decrease along the carotid artery before it reaches the base of the brain. The contributions of both the countercurrent heat exchange and the conduction heat loss to the neck surface, cooled by evaporation and convection to the environment, on the arterial temperature decay are also examined and discussed.

ANATOMIC BACKGROUND

The brain is well supplied with oxygen and nutrients by the blood vessels that form the cerebral arterial circle (Circle of Willis). The blood supply to the brain comes from the right and left common carotid arteries. At the upper level of the larynx, it divides into external and internal carotid arteries. The blood from the face and neck is usually received by the right and left internal jugular veins. They arise as continuation of the sigmoid at the base of the skull. Other sinuses that drain into the internal jugular include the superior sagittal sinus, inferior sagittal sinus, straight sinus, and transverse sinuses. The external jugular veins drain blood from the superficial structures into subclavian veins. In the brain, countercurrent heat exchange could occur between the carotid artery and the adjacent jugular vein on each side of the neck from the aortic arch to the base of the skull, a distance of 250 mm.[18]

The arterial supply to the circle of Willis is approximately 500–750 ml/min.[18] The normal blood flow rate in each common carotid artery of an adult human is thus 250–375 ml/min. Of the total blood supply, about 70% and 30% will be in the internal and external carotid arteries, respectively.[18] A reasonable estimate of the flow rate in the internal and external carotid artery can vary between 75 and 270 ml/min. During moderate muscular exercise, cerebral blood flow may increase approximately by 30% to support the increased neural metabolic activity in motor areas.[6] The measured radius of the carotid arteries in adults varies over the range of 3–6 mm.[26] The jugular vein may have a different size and run parallel to the carotid artery with a center-to-center spacing varying from 6.2 to 8.2 mm.[18]

The main driving force for the heat loss from the carotid artery consists of two parts. One is due to the temperature difference between the artery and the neck surface. During outdoor exercise[15] or face fanning, the skin temperature can be as low as 28 °C, which can be further decreased depending on the air temperature, humidity, wind, and the extent of sweating during hyperthermia. The other is the temperature difference between the countercurrent vessels at their entrance. The jugular vein collects blood flow from the superficial structures and also from the brain itself. The overall venous inlet temperature should be determined by the mixture of the relatively warm blood from the brain and the cold blood from the superficial structures such as the nasal area and the brain surface. Theoretically, it is hard to accurately evaluate the venous inlet temperature without developing a theoretical model which includes heat transfer analysis in both the brain and the neck. In the current model, only the heat transfer in the neck is considered. We consider the experimentally measured canthus temperature at about 18 °C[9] as the lower limit of the venous return temperature.

MATHEMATICAL FORMULATION

The heat exchange between the countercurrent blood vessel pair and the surrounding tissue at the neck level can be described by the schematic diagram shown in Fig. 1, in which an axially uniform vessel and tissue cross section is assumed. The blood supply to the brain is usually carried by the carotid arteries at each side of the neck and the common carotid artery further divides into the internal and external carotid arteries. For simplicity,

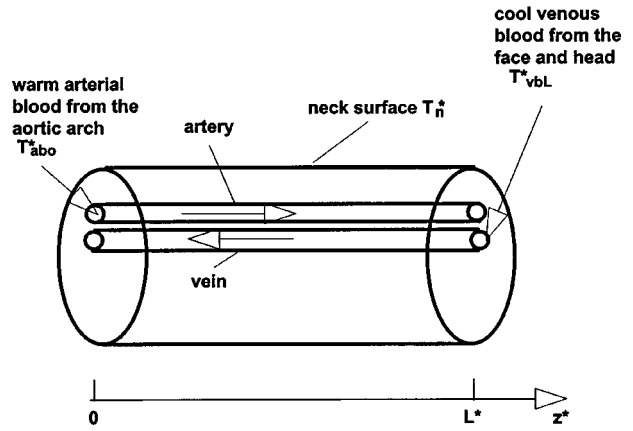

FIGURE 1. Schematic diagram of countercurrent vessels embedded in a cylinder.

only one of the countercurrent artery and vein pairs is considered in the present analysis. Even though the vessels branch from the common artery and the carotid artery, which could result in a longer thermal equilibration length in the countercurrent pair,[23] the countercurrent vessels are treated as of uniform radius along their axial direction as a conservative estimate of the axial temperature decay. The arterial temperature at the aortic arch ($z^*=0$), T^*_{ab0}, represents the body core temperature during moderate exercise. The jugular venous temperature at its entrance ($z^*=L^*$), T^*_{vbL}, is usually lower than the body temperature. The temperature at the outer radius R_t^* of the cylinder (T_n^*) is assumed to be constant along the axial direction. In a real situation, the neck skin temperature may not be uniform. Thus, T_n^* may be viewed as the average skin temperature to account for the overall conduction heat loss.

The basic geometry, symbols, and coordinate system of the cross-sectional plane are sketched in Fig. 2. The thermal properties of the blood and the tissue are assumed to be the same and homogenous within the neck. A steady-state temperature field is assumed in both the blood vessels and the surrounding tissue. This model assumes equal size and volumetric flow rate in the artery and vein. This assumption is reasonable since previous analysis[23] showed that the effect of unequal vessel size is small and produces less than 5% variation in the temperature decay. For simplicity, the artery and the vein are located symmetrically about the x axis. We assume that the flow in the vessels is laminar with a parabolic velocity profile with mean velocity u_a^* since the Reynolds number is small (<2300). The Peclet number for the flow in the blood vessels is very large ($\mathrm{Pe}>2000$) so that axial conduction in the blood vessel and end effects can be neglected.[27] Axial conduction in the tissue is mainly due to the temperature difference from the aorta arch to the base of the brain. It is unlikely that a large

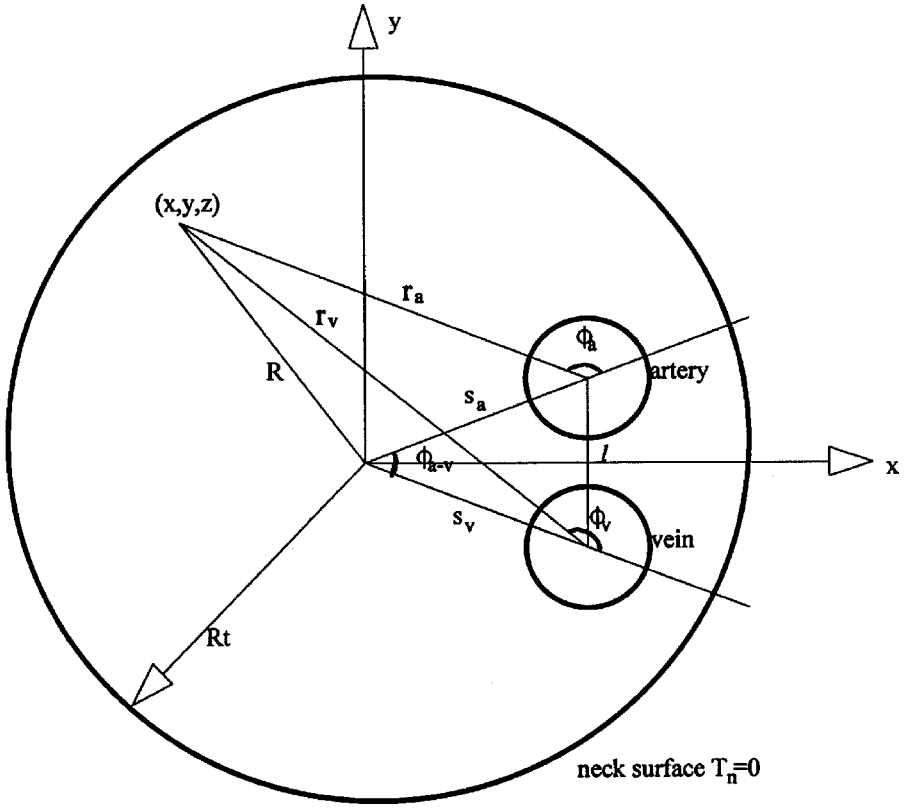

FIGURE 2. Geometry of the cross-sectional plane and coordinate system.

temperature gradient in this direction exists considering the long characteristic length L^* (250 mm) and small temperature drop (<2 °C). On the other hand, a large temperature gradient exists in the radial direction since the temperature difference can be as much as 10 °C on the length scale of 60 mm. The axial conduction term in the governing equation of the tissue is of $O(\Delta T_a^*/L^{*2})R_t^{*2}/(T_a^*-T_n^*)$ times the radial conduction term. Since both R_t^{*2}/L^{*2} and $\Delta T_a^*/(T_a^*-T_n^*)$ are of $O(10^{-1})$, the axial conduction term is of lower order in the governing equation. Thus, the error associated with neglecting the axial conduction in the tissue will be insignificant.

The nondimensional parameters and variables illustrated in Fig. 2 are defined as follows:

$$r_a=\frac{r_a^*}{a_a^*},\quad r_v=\frac{r_v^*}{a_a^*},\quad s_a=\frac{s_v^*}{a_a^*},\quad s_v=\frac{s_v^*}{a_a^*},\quad z=\frac{z^*}{a_a^*},$$

$$R=\frac{R^*}{a_a^*},\quad R_t=\frac{R_t^*}{a_a^*},$$

$$L=\frac{L^*}{a_a^*},\quad \mathrm{Pe}=\frac{2\rho_f C_f a_a^* u_a^*}{k_f}=\frac{2a_a^* u_a^*}{\alpha},$$

$$T_{a,v,t,n}=\frac{T_{a,v,t,n}^*-T_n^*}{T_{a0}^*-T_n^*}, \tag{1}$$

where ρ is density, C is specific heat, k is the thermal conductivity, and α is the thermal diffusivity. The subscripts *a,v* refer to artery and vein, asterisks denote dimensional variables, a_a^* is the dimensional radius of the artery and vein, and s^* is vessel eccentricity. The vessel center-to-center spacing is denoted by l^*, and R_t^* is the neck radius. All length variables are scaled by the artery radius a_a^*.

Based on the above assumptions and definitions, the dimensionless energy equations for the artery, vein, and tissue can be written as

$$\frac{1}{r_a}\frac{\partial}{\partial r_a}\left(r_a\frac{\partial T_a}{\partial r_a}\right)+\frac{1}{r_a^2}\frac{\partial^2 T_a}{\partial \phi_a^2}=\mathrm{Pe}(1-r_a^2)\frac{dT_{ab}}{dz},$$

$$r_a\leqslant 1,\quad 0\leqslant z\leqslant L, \tag{2}$$

$$\frac{1}{r_v}\frac{\partial}{\partial r_v}\left(r_v\frac{\partial T_v}{\partial r_v}\right)+\frac{1}{r_v^2}\frac{\partial^2 T_v}{\partial \phi_v^2}=-\mathrm{Pe}(1-r_v^2)\frac{dT_{vb}}{dz},$$

$$r_v\leqslant 1,\quad 0\leqslant z\leqslant L, \tag{3}$$

$$\frac{1}{R}\frac{\partial}{\partial R}\left(R\frac{\partial T_t}{\partial R}\right)+\frac{1}{R^2}\frac{\partial^2 T_t}{\partial \phi^2}=0,\quad R\leqslant R_t,\quad r_{a,v}>1, \tag{4}$$

where the temperature gradients $\partial T_{a,v}/\partial z$ in the convective term of the vessel energy equations are approximated by the axial gradients of the vessel bulk temperatures, $dT_{a,v}/dz$, as previously justified in Wu *et al.*[25]

The continuity of temperature and heat flux on the vessel surfaces and the convection boundary condition on the tissue cylinder surface require that

$$T_{a,v}=T_t,\quad \text{for } r_{a,v}=1,$$

$$\frac{\partial T_{a,v}}{\partial r_{a,v}}=\frac{\partial T_t}{\partial r_{a,v}},\quad \text{for } r_{a,v}=1, \tag{5}$$

$$T_t=T_n,\quad \text{for } R=R_t. \tag{6}$$

In Eqs. (2) and (3), T_{ab} and T_{vb} are the artery and vein bulk temperatures, respectively, defined as

$$T_{\mathrm{ab}}(z)=\frac{2}{\pi}\int_{-\pi}^{\pi}\int_0^1 T_a(r_a,\phi_a,z)(1-r_a^2)r_a dr_a d\phi_a, \tag{7}$$

$$T_{\mathrm{vb}}(z)=\frac{2}{\pi}\int_{-\pi}^{\pi}\int_0^1 T_v(r_v,\phi_v,z)(1-r_v^2)r_v dr_v d\phi_v. \tag{8}$$

SOLUTION FOR COUNTERCURRENT FLOW

The solution to this boundary value problem can be found in detail in Weinbaum *et al.*[23] Specifically, one can separate the variables and solve the boundary value problem in the cross-sectional plane independent of the axial direction. Using this approach, the axial interaction between vessels is reduced to a coupled system of ordinary differential equations for the axial bulk temperature variation in each vessel, which are

$$T_{\mathrm{ab}}=A_{11}\frac{dT_{\mathrm{ab}}}{dz}+A_{12}\frac{dT_{\mathrm{vb}}}{dz}, \tag{9}$$

$$T_{\mathrm{vb}}=A_{21}\frac{dT_{\mathrm{ab}}}{dz}+A_{22}\frac{dT_{\mathrm{vb}}}{dz}. \tag{10}$$

The coefficients in Eqs. (9) and (10) are given by

$$A_{11}=-A_{22}=-\frac{\mathrm{Pe}}{4}\left\{\ln\left[R_t\left(1-\frac{s_a^2}{R_t^2}\right)\right]+\frac{11}{24}\right\}, \tag{11}$$

$$A_{12}=-A_{21}=\frac{\mathrm{Pe}}{4}\ln\left[\frac{R_t}{l}\sqrt{1-\frac{2s_a^2\cos\phi_{a-v}}{R_t^2}+\frac{s_a^4}{R_t^4}}\right]. \tag{12}$$

TABLE 1. Key parameters.

Thermal properties	Geometric properties	Blood flow and temperature
$\rho_b=\rho_t=1000$ kg/m^3	$R_t^*=60$ mm	$Q=120-360$ ml/min (Nunneley and Nelson)[d]
$C_b=3600$ J/kg K	$a_a^*=a_v^*=2.5$ mm (1.5–3 mm) (Nunneley and Nelson)[d]	$u_a^*=u_v^*=0.102-0.306$ m/s
$k_b=k_t=0.56$ J/mK (Chato)[a]	$s_d^*=s_v^*=30$ mm	Pe$=3274-9822$
$\alpha=1.56\times10^{-7}$ m^2/s	$l^*=6.2-8.2$ mm	$T_{ab0}^*=39$ °C
$1.18-1.8\times10^{-7}$ m^2/s by Chato[a] Valvano *et al.*,[b] Wang *et al.*[c]	$L^*=250$ mm[d] (Nunneley and Nelson)[d]	$T_{vbL}^*=19-29$ °C
		$T_n^*=19-37$ °C

[a]Reference 8.
[b]Reference 21.
[c]Reference 22.
[d]Reference 18.

The solution of Eqs. (9) and (10) requires specification of two boundary conditions in the axial direction. For countercurrent flow, these boundary conditions are the prescribed bulk temperatures at the inlets of the vessels. The arterial inlet temperature $T_{ab}(0)$ is always equal to 1, as shown in its dimensionless form, while the venous inlet temperature $T_{vb}(L)$ is usually less than 1. The temperature difference at their entrance is one of the driving forces for the axial temperature gradient. These boundary conditions are given by

$$z=0, \quad T_{ab}=1, \tag{13}$$

$$z=L, \quad T_{vb}=T_{vbL}<1. \tag{14}$$

The general solution of Eqs. (9) and (10) is an eigenfunction of the form of $e^{\beta z/\mathrm{Pe}}$, in which the eigenvalues β can be negative or positive. T_{ab} and T_{vb} are arbitrary combinations of all possible eigenfunctions. Equations (9) and (10) involve only two coefficients and can be easily solved. An expression for the axial temperature distribution in the artery can be obtained.

RESULTS

From this analysis, the axial temperature decay in the artery is a function of the blood flow rate and the vascular geometry of the countercurrent vessel pair and surrounding tissue. The thermal properties of the tissue and blood[2,8,21] and the geometrical parameters describing the locations of the vessels[18] taken from the literature are summarized in Table 1. The range of the blood flow rate is also listed in Table 1.[8,18,21,22]

We would like to examine the effects of several factors on the arterial temperature at its outlet ($z^*=L^*$), such as vessel eccentricity s and vessel center-to-center spacing l. For a blood flow rate of $Q=240$ ml/min, Fig. 3 shows how s influences the axial temperature variation in the vessels. Heat loss from the artery is found to almost double as the vessel pair moves closer to the neck surface. However, unless the vessel pair is close to the neck surface ($s_{a,v}^*>50$ mm), increasing s^* from 5 to 50 mm increases the axial temperature drop by less than 20%. The dependence of axial temperature distribution on l is illustrated in Fig. 4 for the two limiting conditions when the neck temperature $T_n^*=19$ and 37 °C. Note that l affects the axial temperature decay by altering the countercurrent thermal resistance between the blood vessels. Large l implies that it is difficult for the artery to lose heat to its countercurrent vein. Change in l^* from 6.2 to 8.2 mm produces only an 8% variation in the axial temperature decay when T_n^* is 19 °C, while it produces a 25% variation when $T_n^*=37$ °C. This trend is expected since increasing T_n^* enhances the relative importance of countercurrent heat exchange. The influence of l on the artery temperature drop is thus more obvious with higher neck temperature.

The variation in the vascular geometry causes only minor change in the axial temperature decay. The most important factors that can dramatically influence the axial temperature decay are T_{vbL}^*, T_n^*, and Q in the

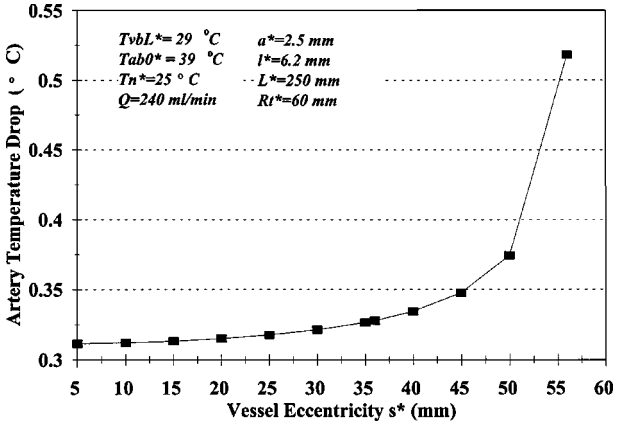

FIGURE 3. Effect of vessel eccentricity on the axial temperature decay in the artery.

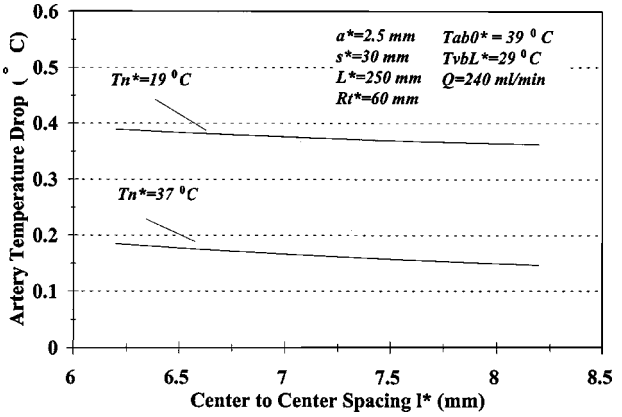

FIGURE 4. Effect of vessel center-to-center spacing on the axial temperature decay in the artery.

countercurrent vessel pair. Figure 5 examines the artery temperature drop over an axial distance L^* as a function of the neck surface temperature T_n^* for different blood flow rates. There are two principal sources for the axial temperature decay. The axial temperature decay is determined by the combined effects of countercurrent heat exchange with the vein and heat conduction to the neck surface. One notes that the artery temperature drop is almost inversely proportional to the blood flow rate Q. A small blood flow rate causes large temperature decay in the axial direction, as expected. Figures 5(a) and 5(b) show the axial temperature decay when the venous inlet temperature T_{vbL}^* is 19 and 29 °C, respectively. For the circumstance of $T_n^* = 36$ °C, close to the artery temperature, increasing the inlet temperature difference between the artery and the vein, $T_{ab0}^* - T_{vbL}^*$, by a factor of 2 almost doubles the axial temperature drop. While at $T_n^* = 19$ °C, when the conduction heat loss to the neck surface becomes more significant, this results in less than a 35% increase in the temperature drop. The theory predicts that the combined effects of both mechanisms can produce as much as 1.1 °C decrease in the axial artery temperature. The total heat loss from the carotid arterial blood is also calculated for different situations, as shown by Table 2. The temperature decays in a single vessel for different blood flow rates are from Fig. 5(b). Therefore, Table 2 gives the upper limit of the heat loss from the carotid arteries. It may be as much as 32.3 W if the total blood supply to the brain is 480 ml/min.

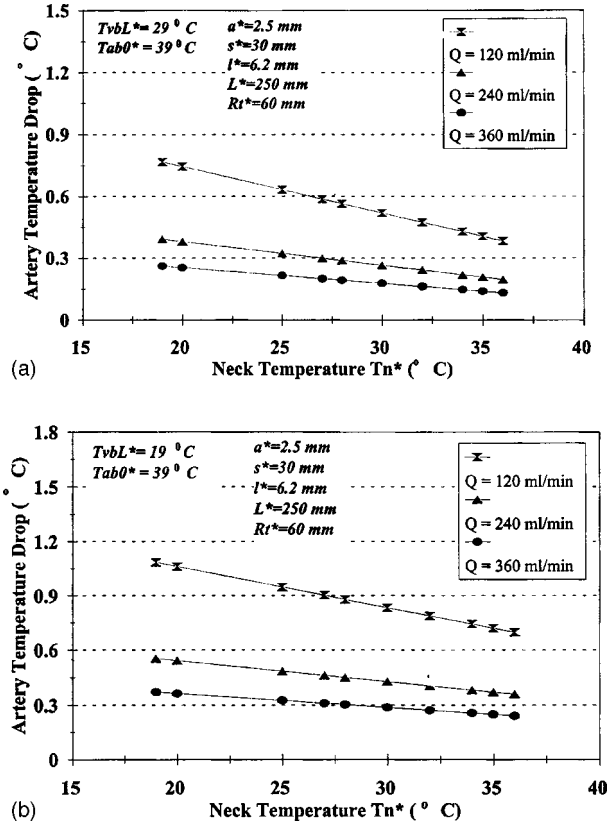

FIGURE 5. Artery temperature drop as a function of neck surface temperature for different blood flow rates.

To further examine the contributions of both conduction heat loss and countercurrent heat exchange to the axial temperature distribution in the artery, Fig. 6 shows the artery temperature drop as a function of neck radius. The extreme condition of very large neck radius represents approximately the situation of the radial conduction heat loss from the artery to the neck surface being negligible, where the arterial temperature decay is approximately 0.23 °C. The lines for $T_n^* = 19$ and 30 °C approach each other at very large R_t^*, as if there were no conduction heat loss to the neck surface. Note that the vessels are located 30 mm from the center of the neck cylinder for any cylinder size. When the neck radius is 60 mm, the temperature decay due to the combined mechanisms is 0.27 and 0.39 °C for $T_n^* = 30$ and 19 °C, respectively. As expected, the contribution of conduction

TABLE 2. Total heat loss from the carotid arterial blood.

Blood flow rate in a single vessel	No. of countercurrent pairs n	Total blood flow to the brain	Heat loss from a single vessel pair $= Q\rho C\Delta T_{ab}$	Total heat loss $= nQ\rho C\Delta T_{ab}$
$Q = 120$ ml/min	4	480 ml/min	8.1 W	32.3 W
$Q = 240$ ml/min	2	480 ml/min	8.0 W	16.0 W

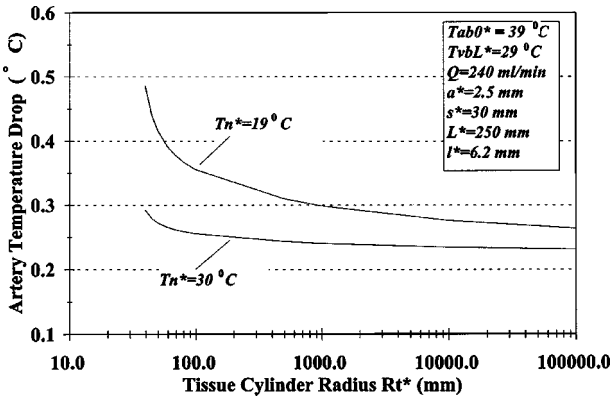

FIGURE 6. Contributions of countercurrent heat exchange and heat conduction to the arterial temperature decay for different neck surface temperatures.

heat transfer becomes more important when there exists a large temperature difference between the neck surface and the artery inlet temperature. When the neck radius is 60 mm, the conduction heat transfer contributes only approximately 15% [(0.27–0.23 °C)/0.27 °C] to the arterial cooling when $T_n^* = 30\,^\circ\mathrm{C}$, while it accounts for more than 41% [(0.39–0.23 °C)/0.39 °C] of the axial temperature decay when $T_n^* = 19\,^\circ\mathrm{C}$.

Surface cooling of the brain and neck surface has been proposed as a therapeutic option for achieving protection from cerebral ischemia in brain injury patients.[10] During the hypothermia therapy, it is critical to closely monitor the brain temperature. Usually the brain temperature for patients at risk for ischemic brain injury is not directly monitored due to the concern of inducing additional tissue damage by the temperature probes. It was assumed that brain temperature parallels body core temperature. If the radial conduction heat loss is dominant when the neck surface is cold, as shown by this study, a significant disparity between the brain temperature and the body core temperature may exist. The results shown in Fig. 5 can be extrapolated to the situation when the neck surface is close to 0 °C (surface is cooled by ice pack). It is interesting to note that the upper limit of the temperature drop in the carotid artery (brain temperature) can be as much as 2 °C. Thus, this deviation has to be taken into consideration during the hypothermia therapy.

DISCUSSION

The theoretical approach presented in this paper provides a simple, but reasonable, model to estimate the potential for cooling of the carotid arterial blood on its way to the brain by heat exchange with its countercurrent jugular vein and by heat conduction loss, as well as the contributions of each mechanism to the arterial cooling. It is the first time the radial conduction is included in the theoretical analysis in SBC. The geometrical factors, such as the vessel center to center spacing and the vessel pair location within the neck, are also examined to elucidate their effects on the thermal equilibration in the artery.

The present calculations have assumed an axially uniform vessel cross section in solving the axial temperature distribution and the surrounding tissue is treated as a conductive media. Blood vessel bifurcation and capillary bleed-off from the main carotid vessel pair will affect the thermal equilibration of the arterial blood. According to the calculation in Weinbaum *et al.*,[23] capillary bleed-off tends to decrease the thermal equilibration length in the artery so that more heat loss will occur if this effect is considered. On the other hand, treating the surrounding tissue as a conductive media may overestimate the heat loss from the carotid blood. If the local blood perfusion in the surrounding tissue is described by a volumetric heat source (blood temperature is higher than tissue temperature), the tissue region near the carotid vessel pair where a smaller blood–tissue temperature difference occurs, may not be affected significantly. However, more rigorous theoretical analysis is necessary where those three factors are taken into account to estimate the overall effect.

In addition to countercurrent heat exchange and conduction to the neck surface, the interaction among neighboring countercurrent vessel pairs may also play a significant role on selective brain cooling. With minor modification, the solution approach is readily extended to situations in which the number of blood vessels is greater than 2. Indeed, this is the case in the human brain, where the common arteries at each side bifurcate to form the internal and external carotid arteries. In addition, the countercurrent heat exchange between the vertebral artery and venous plexus may participate in selective brain cooling as suggested by Zenker and Kubik.[26] Axial variation of the cross-sectional area could also be taken into consideration by solving Eqs. (9) and (10) where the coefficients A_{11}–A_{22} are also functions of axial distance.

During hyperthermia caused by high environmental temperature or by muscular exercise and increased neuronal activities, the body core temperature can rise to as high as 42 °C.[6] However, the human brain is heat sensitive and does not tolerate high temperatures. We suggest it is the body's natural reaction to prevent to a certain extent the overheating, during hyperthermia, of the extremely heat-susceptible cerebral tissue. It is true that temperature depression in the artery is limited by its high flow rate and low surface area per unit length. However, to achieve a 1 °C temperature decrease in the artery blood supplied to the brain during hyperthermia, the artery needs not reach thermal equilibration with the surrounding tissue or the countercurrent vein. Less than a

10% heat loss occurring in the arterial blood before it reaches the base of the brain is enough to allow the brain to be 1 °C cooler than the body core temperature if $T^*_{ab0} - T^*_{vbL}$ and/or $T^*_{ab0} - T^*_n$ are larger than 10 °C.

The conduction contribution is found to be comparable to the countercurrent heat exchange and can account for more than 41% of the total heat loss provided that the neck skin temperature is low. It will become dominant if the neck surface is very cold. Previous experimental observations[20] have shown a 0.2–0.5 °C fall in carotid temperature when ice packs were placed on the homolateral side of the face. McCafferey *et al.*[14] and Cabanac *et al.*[5] found the cooling or heating of small skin areas of the face, forehead, or neck altered tympanic temperature. Those experimental observations were interpreted as evidence that the tympanic temperature was contaminated by the cool skin surface. It is understandable that placing a cool pack on the skin surface can alter the local tissue temperature field. However, if radial heat conduction to the neck surface is playing an important role in the axial temperature decay, placing a cool pack on the skin surface may be interpreted as an enhancement in the radial heat conduction and therefore, to cause more heat loss in the artery blood supply.

The question is whether it is possible to achieve the large local temperature difference, which depends greatly on a number of conditions including air temperature, wind speed, local humidity, sweating on the skin surface, and heat transfer efficiency in the upper airway. The skin surface could lose heat through convection and evaporation when exposed to cold and dry air. If the environmental temperature is high, evaporative heat loss becomes the main factor if the air is hot and the skin is sweating. It is well accepted that the physiological reactions to various influences such as hyperthermia are probably controlled by the central nervous system (CNS), though, how the CNS controls these is not quiet clear. It is suggested that the body core and skin temperatures appear as independent inputs to the CNS, and that the integrating process has some kind of weighting of signals from thermal-sensitive sites distributed within the body and skin. The control of sweating in humans has been previously studied in terms of skin and core temperature by Brengelmann *et al.*[4] Their measurements showed a linear relationship between the body core temperature T_{es} and the rate of sweating at the skin.

In this paper, a theoretical model has been developed to describe the effect of blood flow rate and vascular geometry on the thermal equilibration in the carotid artery. The theoretical approach is used to estimate the potential for cooling of the carotid arterial blood on its way to the brain by heat exchange with its countercurrent jugular vein and by radial heat conduction loss to the cool neck surface. The model is able to evaluate the relative importance of both countercurrent heat exchange and radial heat conduction in selective brain cooling. Results of the present study help provide a better understanding of the thermoregulation during hyperthermia. The model can be used to guide the design of further experimental investigations in SBC.

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NOMENCLATURE

a^*_a	blood vessel radius
C_p	specific heat
k	thermal conductivity
l	vessel center to center spacing, Fig. 2
L	axial length of the blood vessels
Pe	Peclet number of blood flow, Eq. (1)
Q	Blood flow rate
r	radial coordinate of vessels, Fig. 2
R	radial coordinate of cylinder, Fig. 2
s	vessel eccentricity, Fig. 2
T	temperature
T^*_{ab0}	artery bulk temperature at $z^*=0$
T^*_{vbL}	vein bulk temperature at $z^*=L^*$
T^*_n	neck surface temperature
x,y	Cartesian coordinates, Fig. 2
u^*_a	mean velocity of the blood flow
z	axial coordinate

Greek Symbols

α	thermal diffusivity
β	eigenvalue
ρ	density
ϕ_{a-v}	angle between the artery and the vein

Subscripts

a	artery
b	bulk
n	neck
t	tissue
v	vein

Superscripts

*	dimensional parameters

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