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International Journal of Heat and Mass Transfer 46 (2003) 3233–3241
International Journal of HEAT and MASS TRANSFER
www.elsevier.com/locate/ijhmt

Effect of physiology on the temperature distribution of a layered head with external convection

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Received 19 February 2002; received in revised form 30 January 2003

Abstract

To improve the existing thermal models of the human head, we incorporate the effect of the temperature over the metabolic heat generation, the regulatory processes that control the cerebral blood perfusion and their dependence on physiological parameters like, the mean arterial blood pressure, the partial pressure of oxygen, the partial pressure of carbon dioxide, and the cerebral metabolic rate of oxygen consumption.

The introduction of these parameters in a thermal model gives information about how specific conditions, such as brain edema, hypoxia, hypercapnia, or hypotension, affect the temperature distribution within the brain. Our work, on a layered head model, shows that variations of the physiological parameters have profound effect on the temperature gradients within the head.

Keywords: Head models; Physiological parameters; Cerebral blood flow

1. Introduction

This paper explores the effect that cerebral blood perfusion W_b, and the different physiological parameters have over the temperature distribution of a geometrically simplified head model; it also includes the relationship between the tissue temperature and the tissue metabolic rate of oxygen consumption (MRO_2), as well as the metabolic activity q_m.

It is well known that tissue temperature is affected by blood perfusion. In the brain, the cerebral blood flow (CBF) is controlled by regulatory mechanisms, which depend on the mean arterial blood pressure, MABP and on physiological parameters like the partial pressure of oxygen PaO_2, the partial pressure of carbon dioxide, $PaCO_2$, and the cerebral metabolic rate of oxygen consumption ($CMRO_2$), which is also affected by the tissue temperature.

We incorporate these systemic parameters in our thermal model of the head, because they are continuously monitored by clinicians during surgery, or during the treatment of patients with acute brain damage produced by concussions, asphyxiation, stroke or other conditions that result in cerebral ischemia, that is, lack of blood perfusion and/or fall in the cerebral oxygen concentration.

Some other conditions that deviate the physiological parameters (MABP, PaO_2, $PaCO_2$ and $CMRO_2$) from their average values are: severe bleeding, which produces hypoxia and a reduction in the MABP; blood poisoning, which results in hypoxia and hypercapnia (high concentrations of CO_2); formation of edemas, ruptured aneurysms, hydrocephalus, and strokes result from high intracranial pressure or hypertension.

The blood flow in the brain is controlled by the metabolic activity, the physiological parameters and the MABP. Adequate cerebral blood flow is essential to maintain organ function [1]; as a result of this, clinicians continuously monitor these physiological parameters to assess therapy and prevent brain injury after trauma, in sickness or during surgery. On the other hand, it has

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0017-9310/03/$ - see front matter
doi:10.1016/S0017-9310(03)00100-5

Nomenclature

c_i	specific heat of layer i, J/kg °C
$CMRO_2$	cerebral metabolic rate of O_2 consumption
h	heat transfer coefficient, W/m^2 °C
k_i	thermal conductivity of layer i, W/m °C
MABP	mean arterial blood pressure, mmHg
$PaCO_2$	partial pressure of CO_2, mmHg
PaO_2	partial pressure of O_2, mmHg
q_m	tissue metabolic heat generation, W/m^3
q_m^o	tissue metabolic heat generation at normal temperature, W/m^3
r	radial position, m
T	temperature, °C
T_a	mean arterial temperature, °C
T_{air}	surrounding air temperature, °C
W_b	cerebral blood perfusion, ml/min 100 g of tissue
W_o	cerebral blood perfusion at normal temperature, ml/min 100 g of tissue

Greek symbols

ϕ_i	ith physiological parameter
ρ_i	mass density of layer i

been observed in several studies [2,3] that moderate reduction of brain temperature helps decrease tissue damage after ischemia and increases the survival rate after head trauma. However, the direct measurement of brain temperature is highly destructive, and the optimal method to reduce brain temperature is unknown [4].

Therefore, temperature control techniques and non-destructive ways to determine deep tissue temperature are necessary to improve the clinical treatment of head trauma after any kind of ischemic damage. To achieve these goals, it is necessary to develop thermal models that include organ regulatory processes and energy utilization. In this paper, we propose a thermal model of the head that introduces the blood flow regulation mechanisms occurring in the brain and accounts for energy utilization by introducing the effect of temperature over the tissue metabolic activity and the blood flow. We analyze the changes in the temperature distribution of a simplified model of the head as a result of variations in MABP, PaO_2, $PaCO_2$, and $CMRO_2$. We consider the case of air convection at the external surface of the head, as a possible cooling mechanism.

The relation between cerebral blood flow and the physiological parameters is introduced in our thermal model based on several clinical observations, such as the fact that changes in the concentration of CO_2 in the brain tissue affect the time required to achieve brain cooling during hypothermic treatment [5,6], or the fact that during drowning in cold water, an individual can survive thanks to the brain metabolic suppression occurring as a consequence of the cooling of arterial blood in the lungs after aspiration of cold water.

The introduction of physiological parameters in a thermal model gives information about how specific conditions, produced by trauma, drugs or sicknesses, affect the temperature distribution within the brain. The consideration of physiological parameters in a thermal model will allow the creation of more accurate thermal models, and can help clinicians to incorporate temperature to the control and diagnostic techniques used in the treatment of head trauma.

To our knowledge, the effect of the regulatory mechanisms and physiological parameters on the cerebral blood perfusion, has not been studied by other authors before [7].

2. Mathematical model

To determine the steady state temperature distribution, the head is modeled as a layered sphere. We use a Pennes like bioheat equation [8] of the form,

$$k_i \frac{d^2 T_i}{dr^2} + \frac{2}{r}\frac{dT_i}{dr} + \rho_b c_b W_b (T_a - T_i) + q_m = 0, \qquad (1)$$

where r extends from $r = 0$ at the center of the head to $r = R$ and the skin surface, $i = 1, 2, \ldots, M$, and M is the number of layers, W_b represents the blood perfusion term, and q_m denotes the metabolic heat generated by the tissue. In this paper we consider the case of air convection at the external skin surface, and calculate the head temperature distribution for different values of the air temperature T_{air}, and the heat transfer coefficient h.

In our calculations, we assume symmetry about the origin and isotropic properties within each layer. Symmetry is assumed after analyzing the numerical results of Van Leeuwen et al. [9], where the Pennes Bioheat equation is used to determine the temperature distribution in the head of an infant; the geometrical model of the head used in [9] is derived from MRI data, and the temperature distribution calculated is symmetric. Based in these observations and the fact that our main goal is to determine the effect of physiology on the temperature distribution, we decided to use a very simplified head model.

On the other hand, the isotropy assumption in inherent to the Pennes formulation [8], but it can be taken with confidence considering the high concentration of

capillaries in the brain tissues, and the fact that it is a common assumption used in thermal modeling of the head [8–13]. Finally, we assume continuity of temperature and heat flux on the layer interfaces located at $r = r_i$, for $i = 1, 2, \ldots, M-1$.

Most available thermal models [9–12] neglect metabolic heat generation or consider it constant, which is a valid assumption for healthy conditions. However, during sickness, trauma or under the effect of drugs like anesthetics, the metabolic activity q_m, the rate or oxygen consumption $CMRO_2$, and the organ blood flow W_b vary considerably, and such variations must be accounted for in order to achieve accurate thermal modeling. Only on two recent papers [12,13], the tissue metabolic heat was considered as a function of the temperature; and to go further in this direction, in this paper, we consider that W_b and q_m are functions of temperature, tissue type and physiological parameters ϕ_i = MABP, $PaCO_2$, PaO_2, and $CMRO_2$.

2.1. Tissue metabolism and physiology

Blood circulation is essential for a tissue, because it allows the transport of substances such as oxygen and glucose, products of the metabolic activity, and hormones. In the brain, there are regulatory mechanisms that help maintain adequate blood flow by dilating or constricting the diameter of the small arteries depending on the tissue ph, the concentrations of oxygen and carbon dioxide, or the mean arterial blood pressure.

Brain autoregulation helps keep the CBF constant despite changes in the MABP, and also maintains the CBF depending on the level of activity within the tissue. Efficacy in cerebral protection during cardiothoracic surgery or after head trauma depends largely upon metabolic suppression and control of cerebral blood flow. For these reasons, clinicians continuously measure the MABP, the concentration of different substances ($PaCO_2$ and PaO_2), and the tissue level of activity ($CMRO_2$).

In humans, the cerebral metabolic rate of oxygen consumption depends on the core or deep tissue temperature. Experimental results [14], show that $CMRO_2$ increases exponentially with temperature and that changes in the metabolic rate of oxygen consumption affect the CBF in a linear fashion. The exponential temperature dependence of $CMRO_2$ is valid for tissue temperatures lower than the protein break down temperature (42 °C), and larger than 10 °C. The $CMRO_2$ can be expressed as follows

$$CMRO_2 = (CMRO_2)_o Q_{10}^{\left(\frac{T-T_o}{10}\right)}, \tag{2}$$

where $(CMRO_2)_o$ is the value at the normal average tissue temperature T_o, and Q_{10} is a parameter known as the Q_{10} coefficient or the van't Hoff temperature coefficient; its value depends on the species and varies with the age. F or humans, $Q_{10} \approx e$, where e is the Euler number. Q_{10} represents the ratio between the $CMRO_2$ at a normal average temperature (T_o) and a temperature $T = T + T_o$. Eq. (2) is referred as the Q_{10} law, and it was derived from Arrhenius rate law and the fact that $CMRO_2$ is the result of chemical reactions [15] catalyzed by enzymes.

The metabolic heat released by a tissue q_m is proportional to its oxygen consumption ($CMRO_2$), and it is expressed as

$$q_m(T) = q_m^o \exp(\Delta T/10), \tag{3}$$

where q_m^o is the tissue metabolic heat generated at normal core temperature, and its values for different tissue types are reported in the literature [13]; ΔT represents the temperature difference between the tissue temperature T and the normal average tissue temperature T_o, i.e. $\Delta T = T - T_o$.

The average tissue temperature T_o, is assumed equal to the arterial or core temperature T_a. The arterial temperature in homeotherms remains constant to insure optimal organ function; in humans, T_a is maintained between 37 and 37.5 °C. In some mammals, the arterial temperature varies up to 0.5 °C during periods of sleep, and while eating [16,17]. In the presence of drugs like anesthetics, the thermoregulatory response is suppressed and T_a falls. There are models, derived from experimental measurements, that reproduce the time variation of the arterial temperature in dogs during cold water drowning [5,6]; however, in all the steady state studies available to determine temperature distribution of the head, and most of the transient studies [10] the arterial temperature is maintained constant throughout the simulation. Based on the studies cited before, in this paper we consider T_a constant and with a numerical value of 37 °C.

Because of the relation between the physiological parameters and the blood flow, and the importance of the blood flow in the temperature of a perfused tissue, we incorporate the regulatory mechanisms in our thermal model of the head. We introduce the autoregulation mechanism in the thermal model by defining the cerebral tissue perfusion W_b as a function of the temperature, tissue type and the systemic parameters (MABP, PaO_2, $PaCO_2$, and $CMRO_2$).

The relations between the cerebral blood perfusion W_b and the physiological parameters are obtained from a cerebral dynamics model [18], and are incorporated as follows

$$W_b(\phi, T, r) = W_o(1 + \Delta CBF_\phi), \tag{4}$$

where W_o is the average blood perfusion in the cerebral tissue at normal core temperature; ΔCBF_ϕ represents the percentile change in the cerebral blood flow due to

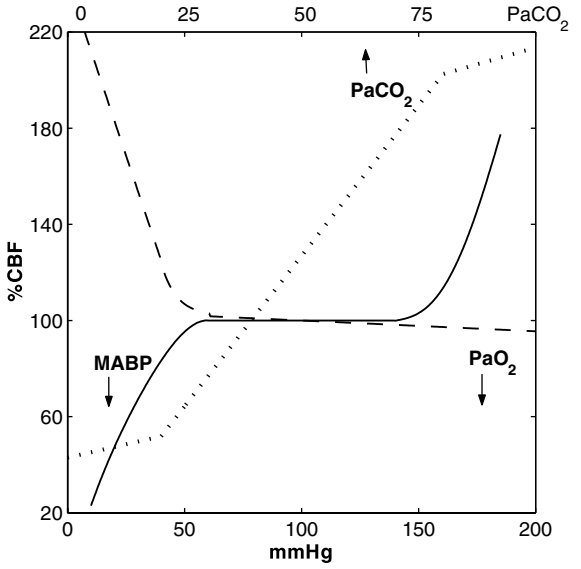

Fig. 1. Variations of the cerebral blood flow (CBF) with the mean arterial blood pressure (MABP), the partial pressure of carbon dioxide ($PaCO_2$), and the partial pressure of oxygen (PaO_2). The normal average values for these parameters are: MABP = 100 mmHg, $PaO_2 = 100$ mmHg, and $PaCO_2 = 40$ mmHg.

variations in the physiological parameter ϕ_i, which is defined as

$$\Delta \mathrm{CBF}_{\phi} = \sum_{\phi_i} \left(\frac{\%\mathrm{CBF}_{\phi_i} - 100}{100} \right), \tag{5}$$

for $\phi_i = \mathrm{MABP}$, PaO_2, $PaCO_2$, and $CMRO_2$). The expressions for the functions $\%\mathrm{CBF}_{\phi_i}$ are documented in the literature [18], and plotted in Fig. 1. The cerebral metabolic rate of oxygen consumption $CMRO_2$ depends exponentially with the temperature, and affects the CBF linearly as noted before.

The exponential model for $q(T)$ was known by physiologists since 1944 [14]; however, it has been considered only recently by Zhu and Diao [13] in a thermal model of the head. The present work includes not only the temperature dependent metabolic heat described in Eq. (3), but it introduces for the first time the effect that some physiological parameters have on the organ blood flow and the tissue temperature distribution by using intracraneal dynamics models.

3. Results

The temperature dependence of the metabolic heat and blood perfusion expressed in Eqs. (3) and (4), gives a nonlinear, second-order differential equation which is solved numerically using the Gauss–Seidel iterative method.

In this paper, we calculate the radial temperature distribution for a 3-layer sphere formed by skin, skull and brain tissue [12,13], subject to a convective boundary condition at the skin surface (T_{air}, h). In our calculations, the metabolic heat generation q_{m} and the tissue blood perfusion W_{b} for the brain tissue region are given by Eqs. (3) and (4), respectively. Because the blood flow in the skin and skull layers is considerable smaller than the blood flow in the brain tissue, and we are not sure that the exponential relations for q_{m} and W_{b} are valid in these layers, we assume that in the skin and skull layers, q_{m} and W_{b} are constant, and their values are given in Table 1.

To analyze the effect of variations in the heat transfer coefficient h, we keep the surrounding air temperature T_{air} constant, and calculate the radial temperature distribution for $h = 25$, 50, 75, and 100 W/m^2 °C, as well as the case of $h \to \infty$, which corresponds to fixed temperature at the external skin surface [7] (see Fig. 2). For these calculations, the normal average values of the physiological parameters were considered (MABP = 100 mmHg, $PaCO_2 = 40$ mmHg, and $PaO_2 = 100$ mmHg).

To study how variations in the surrounding air temperature T_{air}, affect the radial temperature distribution within the head, we keep h constant, and set T_{air} equal to 5, 10, and 20 °C, as shown in Fig. 3, and as before, we use the normal average values of the physiological parameters.

The values of h and T_{air} chosen for these calculations are based on other studies involving head cooling by external convection [11], and in the fact that the room temperature in an operating or emergency room is below 20 °C. From [11], the value of $h = 4$ W/m^2 °C represents convection to still air, and using the Whitaker relation for the average heat transfer coefficient for flow across a single sphere [19], the air velocity corresponding to $h = 25$ W/m^2 °C is of approximately 25 m/s.

In Figs. 2 and 3, as in the case of fixed surface temperature [7], the temperature distribution in the deep

Table 1
Physical and physiological parameters for the 3-layer head model

	Brain	Skull	Skin
k_i (W/m °C)	0.50	1.16	0.34
ρ_i (kg/m^3)	1050	1500	1000
c_i (J/kg °C)	3700	2300	4000
W_{o} (ml/100 gr of tissue/min)	50.02	0.1	2.0
q_{o} (W/m^3)	10437	368.3	363.4
Thickness (mm)	85	4	4

For the blood, $c_{\mathrm{b}} = 3800$ J/Kg °C, and $\rho_{\mathrm{b}} = 1050$ Kg/m^3. These values were taken from Ref. [13].

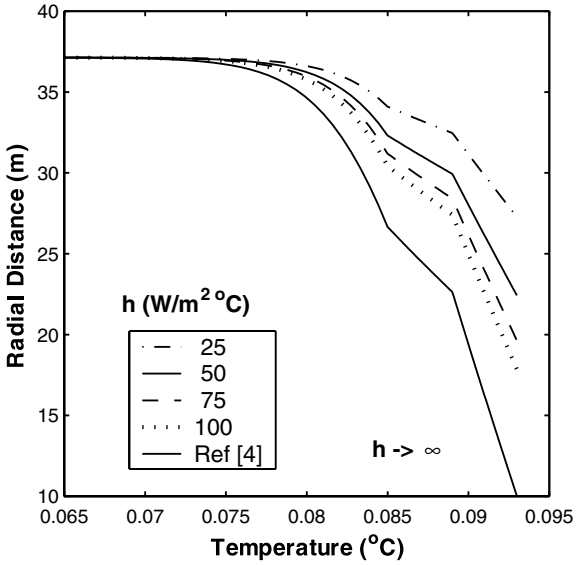

Fig. 2. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Arterial temperature $T_a = 37$ °C, external air temperature $T_{air} = 10$ °C, and heat transfer coefficient $h = 25$, 50, 75 and 100 W/m^2 °C. The last solid curve, represents the limit of $h \to \infty$, and agrees with the results presented in [7] for constant surface temperature.

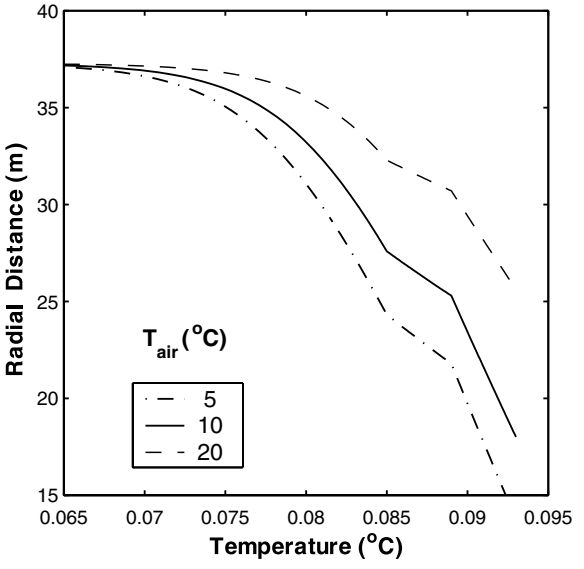

Fig. 3. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Heat transfer coefficient $h = 75$ W/m^2 °C, arterial temperature $T_a = 37$ °C, and external air temperature $T_{air} = 5$, 10 and 20 °C.

structures of the head (gray and white matter) varies within a distance d (*penetration depth*) from the interface between the brain and skull. The temperature distribution in the deeper structures of the head, tends to a limiting value numerically close to the arterial temperature T_a. This limiting temperature is reached as a result of the large volume of warm arterial blood continuously perfusing the brain tissue. The large blood perfusion value in the brain tissue, makes very difficult to alter the deep brain temperature, and reduces the effect of the external boundary conditions of either convection cooling or the contact cooling when $h \to \infty$.

The penetration depth d increases in magnitude with h, until it reaches a maximum constant value that depends on the surrounding air temperature. For $T_{air} = 10$ °C, the penetration depth varies from 4.6 cm when $h = 4$ W/m^2 °C to 6.2 cm for $h \to \infty$. On the other hand, d decreases linearly as the external air temperature raises, and the slope associated depends on the value of h. In Figs. 2 and 3, we note that the temperature at the skin surface is a function of T_{air} and h. We observe that the temperature at the external skin surface decreases exponentially with the heat transfer coefficient, and varies proportionally to the air temperature T_{air}. We will show latter that the temperature at the skin surface also changes with the physiological parameters, but this change is small.

We are interested in the variations between the normal temperature distribution, and the temperature distributions obtained when the physiological parameters depart from their normal average values. To analyze these variations, we vary one of the physiological parameters at a time, keeping the others in their average normal values (MABP = 100 mmHg, $PaO_2 = 100$ mmHg, $PaCO_2 = 40$ mmHg), and we monitor the penetration depth d, the external skin temperature, and the maximum temperature difference between the normal average temperature distribution and the temperature distribution produced by values of the physiological parameter different from the normal average.

In Figs. 4–6, we vary the physiological parameters and the heat transfer coefficient h. We consider the cases of $h = 25$, 75 W/m^2 °C, and the particular case of $h \to \infty$, which reduces the temperature distribution obtained for the case of fixed external surface temperature $T_o = T_{air}$ [7]. In these plots we observe that, regardless of the numerical value of the physiological parameters, as the heat transfer coefficient increases, the penetration depth increases, and reaches its maximum when for $h \to \infty$.

In Fig. 4, we show how variations of the MABP affect the temperature distribution. Changes in the MABP are produced by drugs or conditions like brain edema, brain swelling, vessel collapse, the presence of aneurysms or other conditions that result in stroke. We consider values of the MABP that correspond to mild hypotension (MABP = 45 mmHg), normotension (MABP = 100 mmHg), and mild hypertension

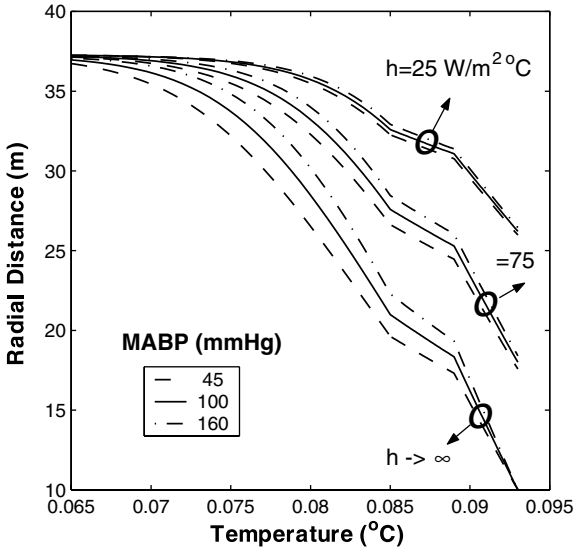

Fig. 4. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Arterial temperature $T_a = 37$ °C, external air temperature $T_{air} = 10$ °C, $PaCO_2 = 40$ mmHg, $PaO_2 = 100$ mmHg, mean arterial blood pressure MABP = 45, 100 and 160 mmHg, and heat transfer coefficient $h = 25$ and 75 W/m^2 °C. The last set of curves, corresponds to the limit of $h \to \infty$, and agrees with the results presented in [7] for constant surface temperature $T_o = 10$ °C.

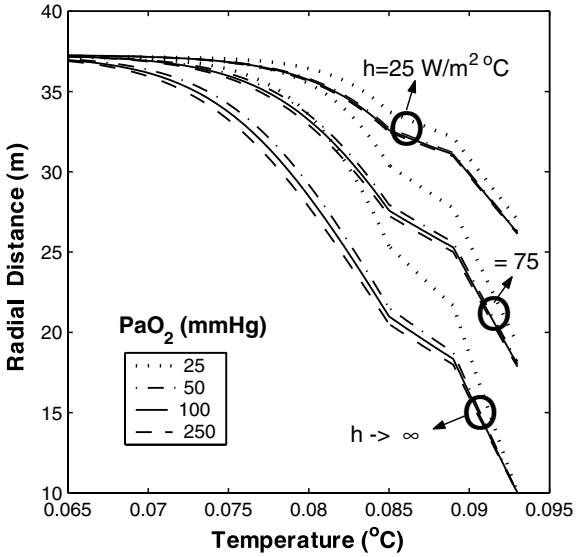

Fig. 5. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Arterial temperature $T_a = 37$ °C, external air temperature $T_{air} = 10$ °C, $PaCO_2 = 40$ mmHg, MABP = 100 mmHg, partial pressure of oxygen $PaO_2 = 25$, 50, 100 and 250 mmHg, and heat transfer coefficient $h = 25$ and 75 W/m^2 °C. The last set of curves, corresponds to the limit of $h \to \infty$, and agrees with the results presented in [7] for constant surface temperature $T_o = 10$ °C.

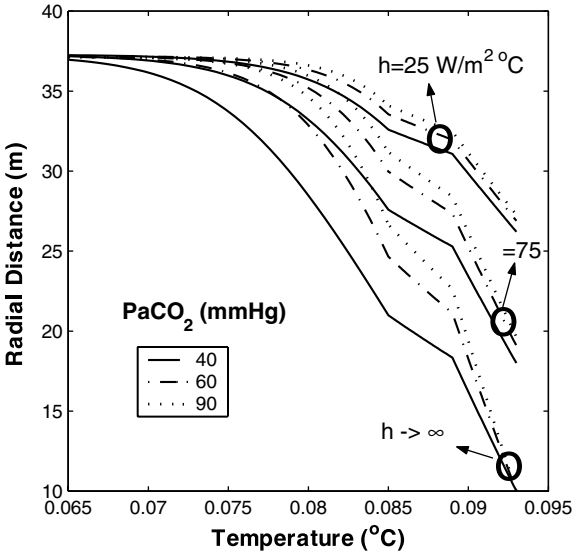

Fig. 6. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Arterial temperature $T_a = 37$ °C, external air temperature $T_{air} = 10$ °C, $PaO_2 = 100$ mmHg, MABP = 100 mmHg, partial pressure of carbon dioxide $PaCO_2 = 40$, 60 and 90 mmHg, and heat transfer coefficient $h = 25$ and 75 W/m^2 °C. The last set of curves, corresponds to the limit of $h \to \infty$, and agrees with the results presented in [7] for constant surface temperature $T_o = 10$ °C.

(MABP = 160 mmHg), and maintain the rest of the physiological parameters in their average normal values.

Varying the MABP, the difference of temperature at the skin surface changes up to 1%, this difference reduces as the heat transfer coefficient increases. In the low pressure case, the blood flow decreases approximately 10%, producing a temperature drop that increases in magnitude with h. This is evidence that the external cooling has a stronger effect when the cerebral blood flow is reduced. When the MABP = 160 mmHg, the CBF increases 13%, and the temperature in the bone layer and the outer part of the brain tissue increases. The maximum temperature difference between the normal arterial pressure case, and the hypotensive and hypertensive cases occurs at the bone/brain interface, and it is about the same magnitude in both cases because the change in the CFB is similar in both cases, and increases with the convection coefficient (h). The penetration depth (d) varies inversely with the MABP, and remains constant for mean arterial blood pressures between 50 and 140 mmHg. This variation is the result of the increase in blood flow for values of MABP lower than 50 mmHg, and the reduction in blood flow for MABP > 140 mmHg, as seen in Fig. 1.

In Figs. 5 and 6, we show the variation of the radial temperature distribution for different values of PaO_2 and $PaCO_2$, keeping the other physiological parameters in their average values and using the exponential temperature dependence for the $CMRO_2$ (Eq. (2)) and the metabolic heat q_m (Eq. (3)). Variations of PaO_2 and $PaCO_2$ are clinically achieved by respiration of air with different concentrations of either O_2 or CO_2, or pathologically produced by asphyxiation or CO_2 poisoning, respectively.

In Fig. 5 we show the radial temperature distribution for $PaO_2 = 25$, 50, 100 and 250 mmHg. Values of the partial pressure of oxygen of less than 100 mmHg represent an hypoxic condition, and produce a CBF increase. For $PaO_2 = 25$ and 50 mmHg, the CBF increases 70% and 7%, respectively; while $PaO_2 = 250$ mmHg reduces the CBF only 2% of its average value, producing a condition called hyperoxia.

Varying PaO_2 in the range 25–250 mmHg, the temperature difference reaches its maxima at the bone/brain interface, and produces a maximum local temperature change of about 1, 1 and 5.4 °C, for $h = 25$, 75 W/m^2 °C, and $h \to \infty$, respectively. When $PaO_2 = 25$ mmHg, the penetration depth d is reduced between 20% and 23%, for $h = 25$ W/m^2 °C and $h \to \infty$, respectively. We observe, for values of h between 4 W/m^2 °C and $h \to \infty$, that the penetration depth increases with the oxygen concentration until it reaches a maximum value; also, as noted before the penetration depth increases with the heat transfer coefficient h. In Fig. 5, we observe that for all values of T_{air} and h considered, the external skin temperature changes less than 4% with respect to the skin temperature value at average PaO_2. The increment in the penetration depth with the oxygen saturation (PaO_2) is the result of the reduction in the blood flow as the oxygen concentration approaches to normal values (Fig. 1). As PaO_2 increases, then volume of warm blood entering the brain tissue decreases, and the external boundary condition has more effect on the tissue.

In Fig. 6, we show the radial temperature distribution for normal concentrations of CO_2 ($PaCO_2 = 40$ mmHg) and compare it with the case of mild and severe hypercapnia, corresponding to $PaCO_2$ values of 60 and 90 mmHg. $PaCO_2$ is a strong vasodilator of the cerebral vasculature; which means that, as the CO_2 concentration increases, the radius of the arterioles will grow and as a result the volume of blood entering the tissue will increase (Fig. 1b). Hypocapnia ($PaCO_2 < 40$ mmHg), on the other hand, reduces CBF and improves the cerebral autoregulatory capacity [6], that is helps the brain vessels to reduce its diameter and regain elasticity after hypertension or conditions that affect the amount of blood entering the brain, like a migraine headache.

During severe hypercapnia ($PaCO_2 = 90$ mmHg), the CBF is increased over 200%. The maximum temperature difference increases with $PaCO_2$ and h. The penetration depth d reduces as $PaCO_2$ increases, and for the values of h and T_{air} considered in the calculation, the penetration depth is reduced approximately 23% with respect to the temperature value obtained for the average CO_2 concentration. The reduction of d as the CO_2 tension increases, is occurs as a result of the increment in the volume of warm blood entering the brain tissue. At the external skin surface, the temperature changes up to 5.33% with respect to the value reached when $PaCO_2 =$ 40 mmHg.

In Figs. 4 and 5 we observed that when the physiological parameters depart from their normal average value, the temperature at the skin surface varies a few degrees, and the maximum temperature difference also varies. We also concluded that the penetration depth d is affected by variations in the physiological parameters, changes in the air temperature and by the heat transfer coefficient. We noticed that the effect of the the external boundary condition over the deep tissue temperature increases when the blood flow is reduced by

From Figs. 4 and 5, we observe that the penetration depth d is small despite severe the variations of the different physiological parameters. This is due to the high blood perfusion of the brain tissue, and the fact that this blood enters the tissue at a high temperature (T_a). Therefore, as one can see in Fig. 7, if the temperature of the deep tissue is to be altered, the arterial blood temperature must be changed. Variation of the

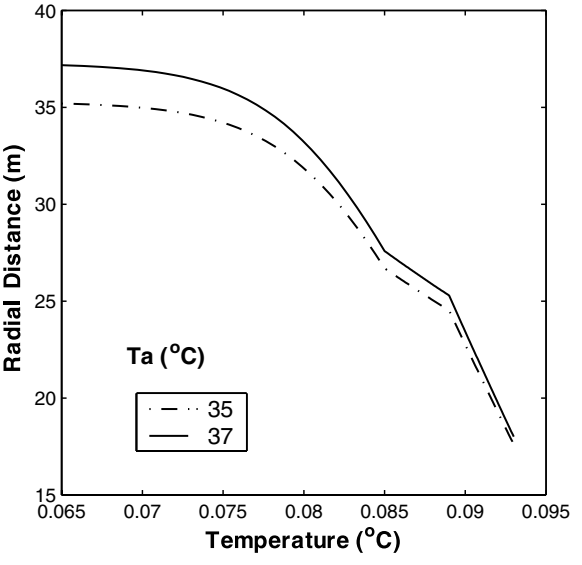

Fig. 7. Temperature distribution for a 3-layer sphere (skin/skull/brain) subject to external convection at the skin surface. Heat transfer coefficient $h = 75$ W/m^2 °C, external air temperature $T_{air} = 10$ °C, normal values of the physiological parameters, and $T_a = 35$, and 37 °C.

arterial blood temperature is usually done through extracorporal perfusion. As seen experimentally [6], using extracorporal perfusion to cool the arterial blood and the help of the alterations in blood flow by the different physiological parameters can help to achieve cooling of the deep brain tissue.

4. Summary and conclusions

This work provides an extension to current thermal models, it introduces organ specific regulatory effects and energy utilization by considering parameters that affect tissue metabolism and organ blood flow. The metabolic heat generation is assumed temperature dependent, and the blood flow is considered a function of temperature, and other physiological parameters.

We conclude that any change in the cerebral blood volume, produced as a result of the regulatory mechanisms (i.e. variations in the physiological parameters), will affect the head temperature distribution. We observe that varying the different physiological parameters so that the CBF is increased, the penetration depth d is reduced and viceversa.

Cooling brain tissue is important in the treatment of head trauma, stroke, or after birth asphyxia, but we observed that no matter how drastic the external boundary conditions are ($h \to \infty$, $T_{air} \to 0$) achieving deep cooling is impossible due to the high blood flow in the brain tissue. A way to overcome this problem, is to reduce blood flow, but lack of blood perfusion causes ischemia and tissue death. Therefore, the ability to control the deep tissue temperature will strongly depend on the temperature of the arterial blood entering the brain, which can be altered by extracorporal perfusion. Once T_a has been lowered, variations in the physiological parameters can help to reduce or increase temperature gradients in the brain, as noted experimentally [6].

The results of this model can help anesthesiologists and pediatricians interested in cerebral protection by temperature reduction, to develop new experiments; it can also be used to create teaching simulators, to help medical students understand the effect of physiological parameters and external cooling conditions on the brain temperature distribution.

Acknowledgements

This work is partially supported by the National Science Foundation grant no: CTS 0222504. The authors would like to thank Dr. Matt Bayazitoglu for his comments and guidance throughout this work.

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