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STUDY OF THE TEMPERATURE OF CEREBRAL ARTERIAL BLOOD FLOW, FOR THE CHARACTERIZATION OF THE DEGREE OF STENOSIS: APPLICATION TO STROKE

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ABSTRACT

Based on the biological heat transfer equation of Penne, the internal temperature distribution of the biological tissue was studied, taking into account the evolution of stenosis and hematocrit. The one-dimensional simplifying cylindrical heat equation of the biological living tissues in permanent regime was solved by the finite difference method and analytically, to assess the temperature change under the variation of stenosis, hematocrit, thermal conductivity, kinematic viscosity, generation of metabolic heat and the heat transfer coefficient. The main results show that the temperature increases as the stenosis and hematocrit increase in size; and the secondary results show that the heat transfer coefficient and the thermal conductivity lower the body temperature while metabolic heat generation increases body temperature. This is in accordance with the literature.

KEYWORDS: Temperature, Penne's equation, Stenosis, Hematocrit

INTRODUCTION

With the advancement of clinical medicine in thermal diagnosis of diseases, it is very important to understand thermal phenomena and the behavior of biological body temperature. One approach is to study the distribution of temperature in biological tissue. However, precise thermal analysis of biological tissue is difficult because they include conduction, convection, radiation, internal metabolism, evaporation, phase change and the regulation of the inherent temperature. Not only is the tissue heterogeneous and anisotropic, but the mechanisms also maintaining body temperature, such as blood flow and metabolic heat generation. Recently, the development of research shows that the problem of heat transfer in biological tissues becomes a complex problem. But there are several discussions without conclusion in this area. Thus in (HenneryPennes, 1948) proposed a simple linear mathematical model to describe the thermal interaction between human tissue and perfused blood, and the effect of metabolism. He measured the radial temperature in the forearm by drawing thin thermocouples through the arms of nine elongated subjects. This was based on experimental observation. Using this experimental concept, the model (Mitchell and Myers 1968), the model (Keller and Seiler 1971), the model (Wulff 1974), the model (Chen and Holmes 1980), the model (Weinbaum and Jiji 1985), the model (Khaled and Vafai 2003), (Nakayama and Kuwaha Model 2008) have discussed thermal behavior in various constant parameters with human tissue layers. The

distribution of temperature takes over in the blood and arterial tissues. (Cooper and Trezek 1972) found an analytical solution of heat diffusion equation for brain tissue with negligible effect on blood flow and metabolic heat generation. (D. B. Gurung 2007) studied the abnormal thermoregulation model in the human dermal part. He also studied the temperature distribution in the steady state and in the unstable state in three layers of the dermal part. He also used the numerical method for the finality of the techniques of solution which is the most precise for the analysis of the temperature in the human body. (Hen and Dil 2018) study the effect of the thermal parameters of the dermal part in living cylindrical tissues. For the different models which have been investigated in literature, none of them did not considered the effect of hematocrit and stenosis which strongly influences the flow regime. In the present work, we are going to use the model of (Pennes 1948) use by (Hen and Dil 2018), by coupling with hematocrit and stenosis to better control the variation of the temperature. Furthermore, we shall analyze the partitioning effect of hematocrit and the evolution of atherosclerosis on variation temperature variation. Here, we neglected the axial and angular direction and considered only the stationary state model of radial direction. The numerical (finite difference method) and analytical result obtained are presented graphically and compared to the result of (Zhang et al 2004) and (Hen and Dil 2018) by applying the appropriate values of the physical and physiological parameters. The solution obtained can be used for the measurement of thermal parameters, the reconstruction of the temperature field and thermal diagnosis and in the treatment which maximizes the therapeutic effect while minimizing undesirable side effects. It may also be useful to design medical devices to operate within a special range of heating and cooling temperature rates, such as (Zhang et al 2004), thus for the prediction of cerebrovascular diseases.

II) MATRIELS AND METHODS

II-1) MODELISATION OF TEMPERATURE

Over the past years, many researchers have developed different models with different views. The mathematical model used for the transfer of bio-heat is based on the Penne equation. (Pennes 1948) is preferable for studying the heat transfer between the blood and the tissues which also associates the metabolism effect and the blood perfusion. The modified Pennes equation is written as

$$\rho c \frac{\partial T}{\partial t} = \nabla(k\nabla T) + w_b c_b \rho_b (T_a - T) + q_m \quad (1)$$

where ρ , c , k are the density (kg/m^3), the specific heat ($\text{J}/\text{kg}\cdot^\circ\text{C}$) and the thermal conductivity of tissue, respectively. w_b is the blood perfusion rate per unit volume ($\text{kg}/\text{s}\cdot\text{m}^3$). c_b is the specific blood, q_m is the metabolic heat generation per unit volume (W/m^3), T_a represents the temperature of arterial blood ($^\circ\text{C}$) and T is the tissue temperature ($^\circ\text{C}$). Based on the Pennes equation, the one-dimensional mathematical model was used to describe the heat transfer

from living cylindrical tissues, in the stationary state, is presented below (Kai and Xinxin; Hen and Dil 2018).

$$\frac{1}{r} \frac{d}{dr} \left(r \frac{dT}{dr} \right) + \frac{w_b c_b \rho_b}{k} (T_a - T) + \frac{q_m}{k} = 0 \quad (2)$$

The model is axial symmetric, so the boundary conditions are described as (Kai and Xinxin ;Hen and Dil 2018):

$$\begin{cases} r = 0, \frac{dT}{dr} = 0 \\ r = R, -k \frac{dT}{dr} = h_A (T - T_\infty) \end{cases} \quad (3)$$

Where R is the radius of the concerned tissue, h_A is the coefficient of heat transfer which accounts for the effects of both convection and radiation on the surface of the tissue, T_∞ is the ambient temperature.

II-2) STENOSIS MODELING

Stenoses are characterized by a lesion of lipid infiltration, called an athermous plaque which develops on the internal surface of the arterial vessel (Figure II- 1) and which blocks blood circulation and hematocrit (Figure II-2).

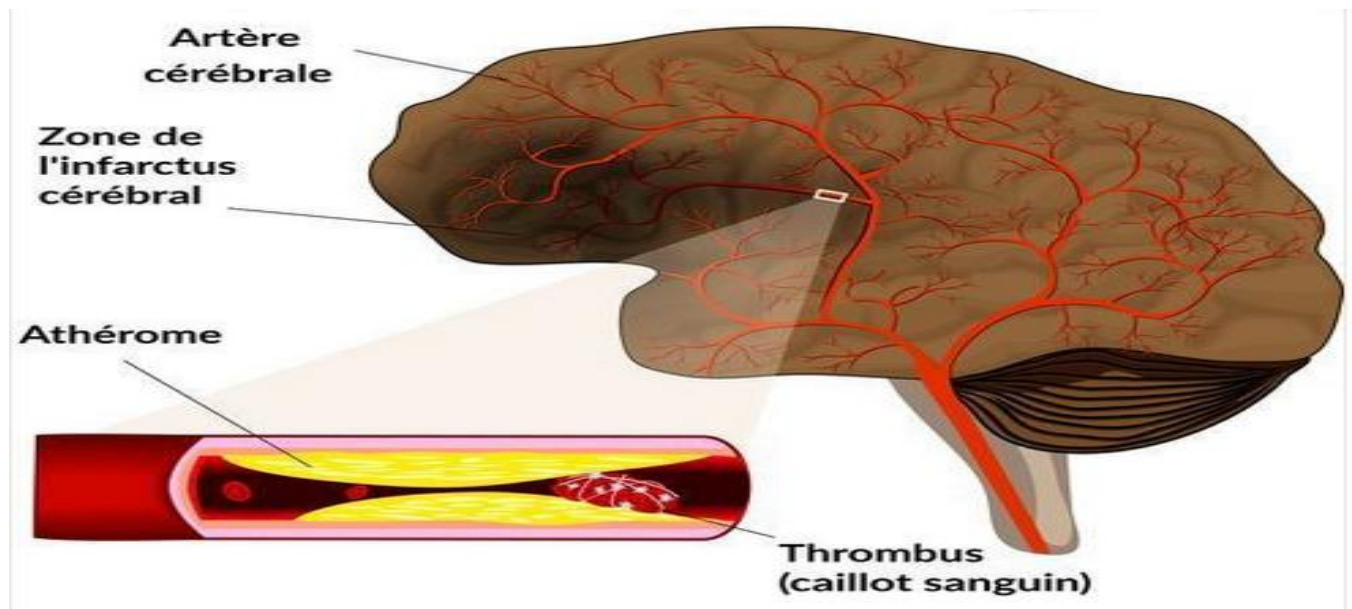


Figure (II-1) https://www.doctissimo.fr/html/sante/encyclopedie/sa_1583_ischemie_cereb.htm

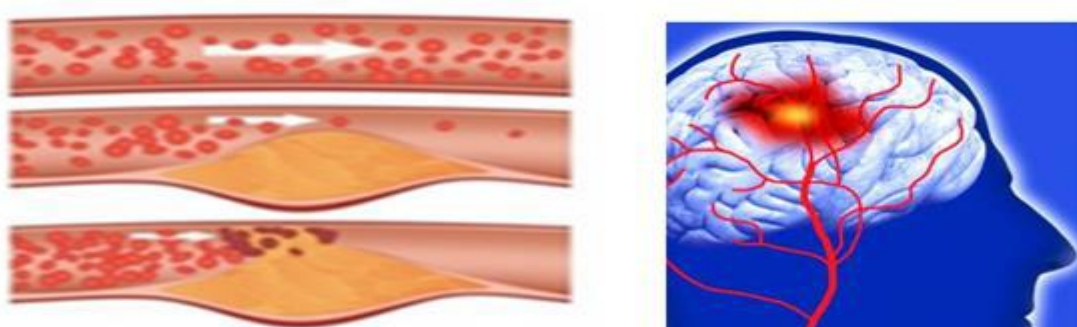


Figure (II-2) :Circulation of hematocrit in different stage of stenoses (Tsafack et al 2019)

II-3) CHARACTERIZATION OF DIFFERENT VALUES OF REYNOLD

To characterize the Reynolds difference value at the different stages of stenosis, we concentrated our mine in the work of (Maurizio 2018), which during its simulation calculated the Reynolds number (Re). The Reynolds number is a dimensionless quantity which is useful for determining whether the flow is laminar or turbulent: if it is greater than a critical value, the flow is turbulent, if it is less, the flow is laminar. In general, the Reynolds number is defined by the flowing formulation

$$Re = \frac{\rho_b \vartheta d}{\mu_b} \quad (4)$$

Where ρ_b is the blood density, d is the diameter of the vessel, ϑ is the mean blood velocity throughout the vessel and it can be expressed as the ratio between the mean incoming flow (Q) and the inlet area (A):

$$\vartheta = \frac{Q}{A} = \frac{4Q}{\pi d^2} \quad (5)$$

μ_b is the blood viscosity that is express using a simplified formulation of the Einstein relationship and is given by the following formula

$$\mu_b = \mu_p (1 + 2.5H_t) \quad (6)$$

where μ_p represents plasma viscosity

For low value of shear rate, blood behave like pseudoplastic fluids and this behavior occurs for $H_t > 12\%$. This is due to the presence of fibrinogen which determine the aggregation of the red blood cells into “rouleau”, generally mode up by fewer tens of erythrocytes, increasing

blood viscosity. Since these parameters were known, Reynolds number were calculated at rest condition both for the non-stenotic model and in correspondence of the 3 stenoses diameter, to see if these three degrees of stenosis may promote a laminar flow or cause a turbulent flow. The value obtained by (Maurizio 2018) are reported in the following table (1)

Table (1): Different stages of artery conditions with correspondent of Reynolds values determine by (Maurizio 2018) at rest condition $Q_{rest} = 60ml/min$

Different stage of artery condition	Non-stenotic model	30% stenosis	50% stenosis	75% stenosis
Re	334.04	482.27	675.15	1350.33

II-4) FINAL MODEL FORMULATION

In this section, we will couple the bio-heat model with the stenosis model. Introducing equation 6 into equation 4, we will have

$$Re = \frac{\rho_b \vartheta d}{\mu_p(1+2.5H_t)} \quad (7)$$

Using equation (7), we have the new blood density

$$\rho_b = \frac{\mu_p(1+2.5H_t)Re}{\vartheta d} \quad (8)$$

Now let us introduce equation (8) into equation (2) to have a final expression of our model

$$\frac{1}{r} \frac{d}{dr} \left(r \frac{dT}{dr} \right) + \frac{w_b c_b \mu_p (1 + 2.5H_t) Re}{k \vartheta d} (T_a - T) + \frac{q_m}{k} = 0 \quad (9)$$

II-5) RESOLUTION BASED ON FINITE DIFFERENCE METHOD

From equation (9), we have

$$\frac{d^2 T}{dr^2} + \frac{1}{r} \left(\frac{dT}{dr} \right) + \frac{w_b c_b \mu_p (1 + 2.5H_t) Re}{k \vartheta d} (T_a - T) + \frac{q_m}{k} = 0 \quad (10)$$

Using the Finite Difference Method (FDM)

$$\frac{\partial T}{\partial r} = \frac{T_{i+1} - T_{i-1}}{2h} \quad (11)$$

$$\frac{\partial^2 T}{\partial r^2} = \frac{T_{i+1} - 2T_i + T_{i-1}}{h^2} \quad (12)$$

with $h = \Delta r$

Now let us introduce equation (11) and (12) into equation (10), with have

$$\left(1 - \frac{1}{2i}\right) T_{i-1} - \left(2 + \frac{w_b c_b \mu_p (1 + 2.5H_t) \text{Re}}{k \vartheta d} h^2\right) T_i + \left(1 + \frac{1}{2i}\right) T_{i+1} = F \quad (13)$$

where $F = - \frac{h^2}{k} \left(q_m + \frac{w_b c_b \mu_p (1 + 2.5H_t) \text{Re}}{k \vartheta d} T_a\right)$

II-6) DISCRETISATION OF BOUNDARY CONDITION

from equation (3)

$$\text{for } i = 0 : 2T_1 - \left(2 + \frac{w_b c_b \mu_p (1 + 2.5H_t) \text{Re}}{k \vartheta d} h^2\right) T_0 = F \quad (14)$$

for $i = 1, 2, \dots, R-1$

$$\left(1 - \frac{1}{2i}\right) T_{i-1} - \left(2 + \frac{w_b c_b \mu_p (1 + 2.5H_t) \text{Re}}{k \vartheta d} h^2\right) T_i + \left(1 + \frac{1}{2i}\right) T_{i+1} = F \quad (15)$$

and for $i = R$

$$2T_{R-1} - DT_R + E = F \quad (16)$$

with

$$D = \left[2 + \frac{w_b c_b \mu_p (1 + 2.5H_t) \text{Re}}{k \vartheta d} h^2\right] - \frac{2hh_a}{k} \left(1 + \frac{1}{2R}\right)$$

$$E = \frac{2hh_a T_\infty}{k} \left(1 + \frac{1}{2R}\right)$$

From equation (14), (15) and (16) we can find the following system of linear equations represented in matrix form:

$$AX = B \quad (17)$$

where

$$\begin{bmatrix} a & 2 & 0 & \dots & 0 \\ b_i & a & c_i & \dots & 0 \\ 0 & b_i & a & c_i & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 2 & -D \end{bmatrix}; \quad X = \begin{bmatrix} T_0 \\ T_1 \\ T_2 \\ \vdots \\ T_R \end{bmatrix} \text{ and } B = \begin{bmatrix} F \\ F \\ F \\ \vdots \\ F - E \end{bmatrix}$$

$$a = -\left(2 + \frac{w_b c_b \mu_p (1 + 2.5 H_t) \text{Re}}{k \vartheta d} h^2\right); \quad b_i = \left(1 - \frac{1}{2i}\right) \text{ and } c_i = \left(1 + \frac{1}{2i}\right) \text{ where } i \text{ is the number of line.}$$

The matrix (17) gives the nodal values in FDM which make it possible to calculate the temperature distribution profile.

II-7) ANALYTICAL RESOLUTION

To solve analytically, we first carry out the dimensioning of equation (9) and its boundary condition by introducing the characteristic quantities (Ozisik et al 1983 and Wang et al 1992)

$$r^* = \frac{r}{R}; \quad T^* = \frac{T - T_\infty}{T_a - T_\infty} \quad (16)$$

Then, replace equation (16) in equation (9):

$$\frac{1}{r^*} \frac{d}{dr^*} \left(r^* \frac{dT^*}{dr^*} \right) + \frac{w_b c_b \mu_p (1 + 2.5 H_t) \text{Re}}{k \vartheta d} R^2 (1 - T^*) + \frac{q_m R^2}{k(T_a - T_\infty)} = 0 \quad (17)$$

Here the dimensionless parameters and variables will be defined as

$$w_b^* = \frac{w_b c_b \mu_p (1 + 2.5 H_t) \text{Re}}{k \vartheta d} R^2; \quad q_m^* = \frac{q_m R^2}{k(T_a - T_\infty)}; \quad h_A^* = \frac{h_A R}{k} \quad (18)$$

Thus equations (9) and (3) can be rewritten in the form

$$\frac{1}{r^*} \frac{d}{dr^*} \left(r^* \frac{dT^*}{dr^*} \right) - w_b^* T^* - w_b^* T^* + (w_b^* + q_m^*) = 0 \quad (19)$$

$$r^* = 0, \frac{dT^*}{dr^*} = 0 \text{ and } r^* = 1, \frac{dT^*}{dr^*} = -h_A^* T^* \quad (20)$$

Also, in order to normalize the equation, we assume

$$A = w_b^* + q_m^*, \quad B = w_b^*, \quad \varphi = A - B T^* \quad (21)$$

By thus substituting equation (21) in equation (19), we obtain:

$$\frac{d^2\varphi}{dr^{*2}} + \frac{1}{r^*} \frac{d\varphi}{dr^*} - B\varphi = 0 \quad (22)$$

It is clear that equation (22) is a modified zero order Bessel differential equation, the general solution of which can be expressed as follows:

$$R(z) = c_1 I_\nu(z) + c_2 k_\nu(z) \quad (23)$$

Where I_ν and k_ν are respectively the modified Bessel function of the second type. In order to determine, if the analytical solution can be expressed by the Bessel function, equation (22) was compared to the generalized Bessel equation as follows:

$$\frac{d^2R}{dx^2} + \left[\frac{1-2m}{x} - 2\alpha \right] \frac{dR}{dx} + \left[p^2 a^2 x^2 p^{-2} + \alpha^2 + \frac{\alpha(2m-1)}{x} + \frac{m^2 - p^2 v^2}{x^2} \right] R = 0 \quad (24)$$

The corresponding solution of equation (24) is:

$$R = x^m e^{\alpha x} [c_1 J_\nu(ax^p) + c_2 Y_\nu(ax^p)] \quad (25)$$

Where J_ν and Y_ν are respectively the modified Bessel functions of the first type, c_1 and c_2 are arbitrary constants which can be obtained as a function of the given boundary conditions. The result of the comparison between equation (22) and equation (24) is shown below:

$$\alpha = 0, m=0, v = 0, p=1, a^2 = -\beta$$

Thus, the solution of equation (22) can be expressed as:

$$\varphi = c_1 I_0(\sqrt{B}r^*) + c_2 K_0(\sqrt{B}r^*) \quad (26)$$

By replacing (26) with (21), the solution of (19) can be written as

$$T^* = \frac{w_b^* + q_m^*}{w_b^*} - \left[\frac{c_1}{w_b^*} I_0(\sqrt{w_b^*} r^*) + \frac{c_2}{w_b^*} K_0(\sqrt{w_b^*} r^*) \right] \quad (27)$$

The next step is to determine the values of two arbitrary constants c_1 and c_2 .

According to the characteristics of the Bessel equation, when $z = 0$, we have

$$I_1(0) = 0 \text{ and } K_1(0) \rightarrow \infty$$

Taking into account the boundary conditions (20), simple derivations lead to:

$$c_2 = 0, \frac{dT^*}{dr^*} = -\frac{c_1}{w_b^*} I_1(\sqrt{w_b^*} r^*) \quad (28)$$

so we have :

$$T^*(r^*) = \left(1 + \frac{q_m^*}{w_b^*}\right) \left[1 - \frac{I_0(\sqrt{w_b^*} r^*)}{I_0(\sqrt{w_b^*}) + \frac{\sqrt{w_b^*}}{h_A^*} I_1(\sqrt{w_b^*})}\right] \quad (29)$$

Finally, the analytical solution of T is:

$$T(r^*) = T_\infty + (T_a - T_\infty) \left(1 + \frac{q_m^*}{w_b^*}\right) \left[1 - \frac{I_0(\sqrt{w_b^*} r^*)}{I_0(\sqrt{w_b^*}) + \frac{\sqrt{w_b^*}}{h_A^*} I_1(\sqrt{w_b^*})}\right] \quad (30)$$

II-8) DETERMINATIONS OF PARAMETERS

Table 2: Values of parameters

Symbol	Value	Unit	Description	Reference
w_b	0.00003	Kg/s.m ³	Blood perfusion	
c_b	3850	J/Kg.°C	Blood specific heat	Hen and Dil 2018
K	0.48	W/m. °C	Tissue thermal conductivity	Hen and Dil 2018
h_A	30.023	W/m. °C	Heat transfer coefficient	Hen and Dil 2018
q_m	1085	W/m ³	Heat generation per unit volume	Hen and Dil 2018
T_a	37	°C	Artery temperature	Hen and Dil 2018

R	0.0285	m	radius	Hen and Dil 2018
H_t	45%	-	Hematocrite	Hen and Dil 2018
ϑ	0.0167		mean blood velocity	
μ_p	0.165		plasma viscosity	

III) RESULTS AND DISCUSSIONS

III-1) VALIDATION OF RESULTS

The results of the new model were validated by the reproduction of the results of the literature established for a simple pipe (without stenosis and Hematocrit). Figure (1) shows a decrease in temperature when the radial direction becomes important. This is due to the fact that when the body temperature is above the set value, the hypothalamus causes the phenomenon of sweating, evaporation, which causes a lowering of the skin temperature. At the same time, the skin arterioles dilate in order to promote heat exchanges with the outside.

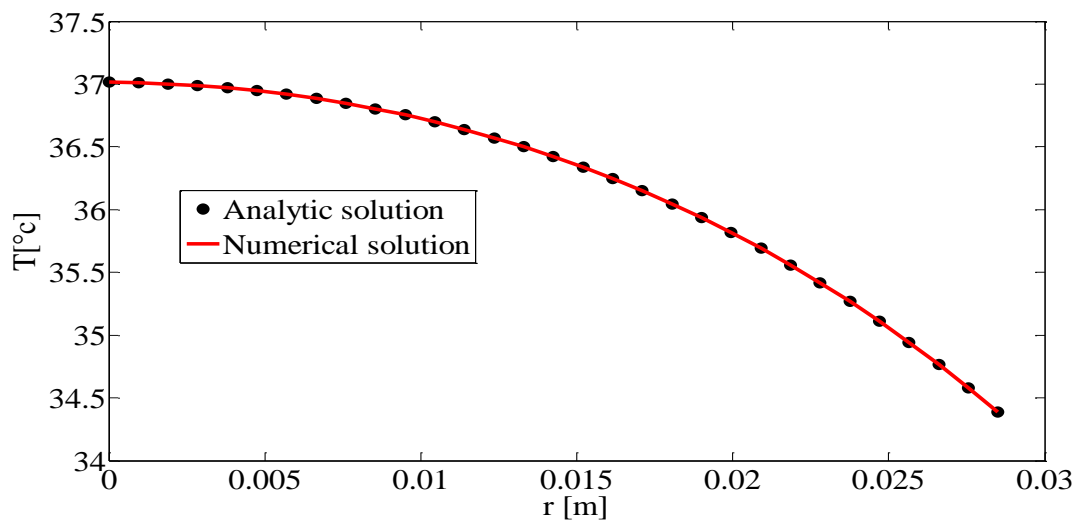


Figure (1): Temperature as a function of the radial direction

II-2) INFLUENCE OF STENOSIS ON TEMPERATURE

Figure (2) describes the evolution of the temperature with the increasing of the stenosis size. It shows that, as the obstacle increases, the temperature increases too. This increase in temperature is due to the friction of the fluid particles. This implies that, the stress becomes important at the cross of the stenosis section and it will therefore be important to control the temperature to avoid the evolution of stenosis. In the previous work of (Tsafack et al 2019) it was observed that, when the stenosis increased radially, the amplitude of the oscillations decreased as the inertial blood flow decreased rapidly due to blockage of the lumen. This suggests that the loss of singular inertial blood flow occurs when there is disturbance of the normal flow.

In the same vein (Gou et al 1999) shows for a high temperature, more than 38 °C, the patient is victim of cardiovascular and cerebrovascular disease. This is in agreement with our result, since figure (2) shows that, at a higher temperature of 38 °C the stenosis is already more than 50% of reduction in the initial wall.

Furthermore, in the experience of (kin et al 1996), he observed a rat with a body temperature above 40 °C, plunging into a brain violation in a few hours. this is in agreement with our result, since figure (2) shows that, when the temperature is higher than 40 °C, the degree of stenosis is higher than 75% of reduction of the arterial wall, therefore this is normal that, this rat is exposes to the brain offense.

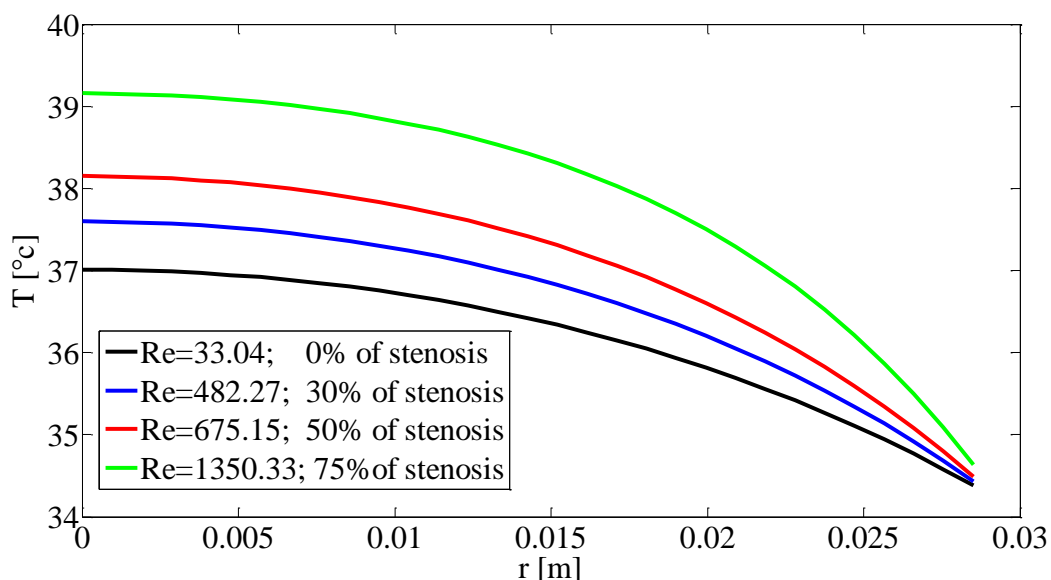


Figure (2): Influence of stenosis on temperature

III-3) TEMPERATURE DECREASE STUDY

III-3-1) INFLUENCE OF THE HEAT TRANSFER COEFFICIENT

Figure (3) shows that increasing the heat transfer coefficient decreases body temperature. This is because the temperature of the atmospheric pressure cools the body surface.

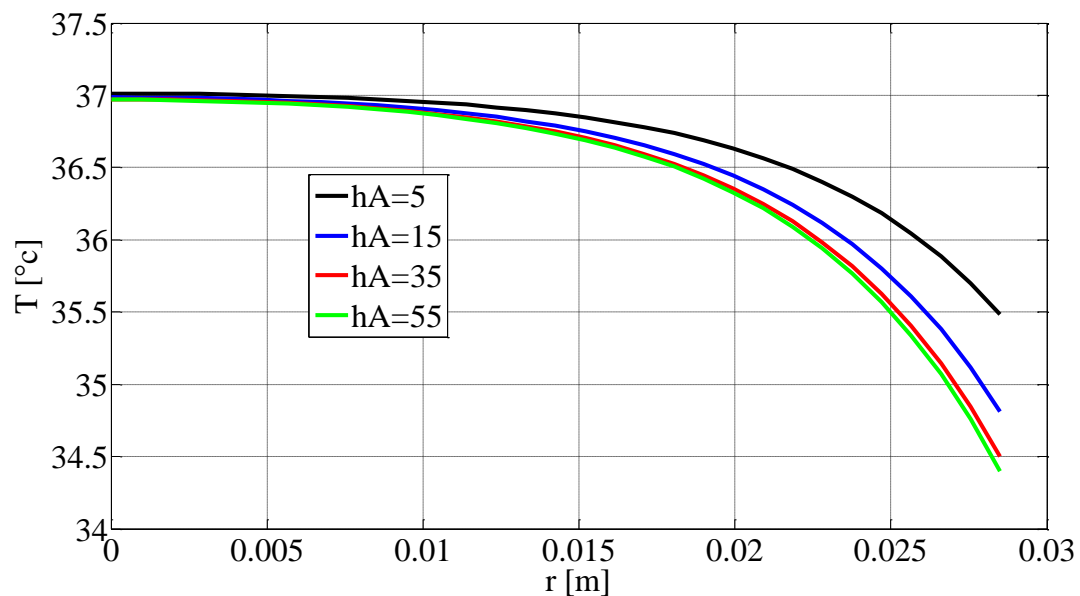


Figure (3): Influence of the heat transfer coefficient on temperature

III-3-2) INFLUENCE OF THERMAL CONDUCTIVITY (K)

The result of figure (4) shows that the thermal conductivity has the effect of lowering the temperature. This decrease in temperature is because the atmospheric air touches the body at a lower temperature. According to Fourier law the temperature moves from the hottest medium to the least hot medium.

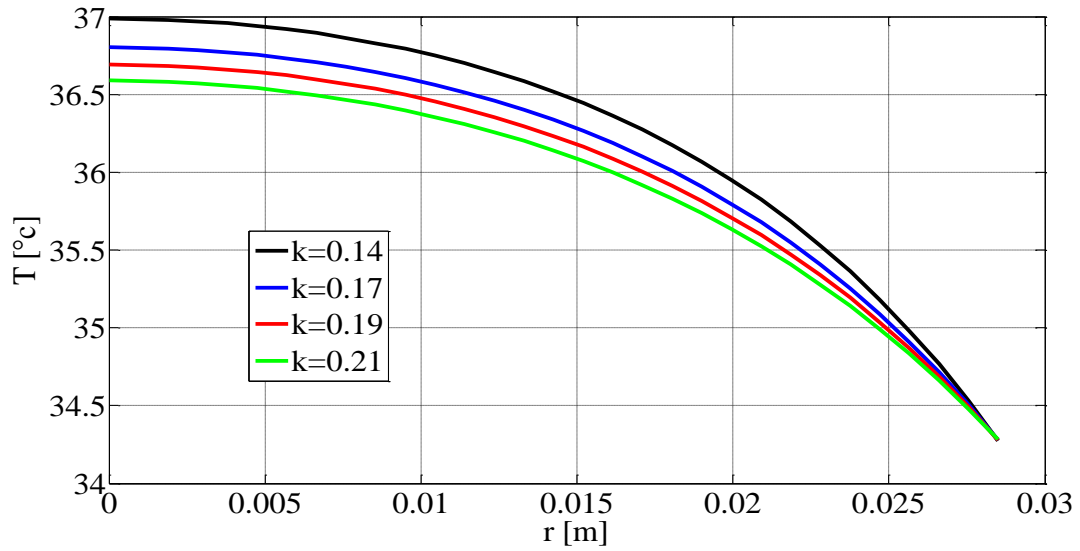


Figure (4): Influence of thermal conductivity (K) on the temperature

III-4) STUDY OF AN INCREASE OF TEMPERATURE

III-4-1) INFLUENCE OF HEMATOCRITIS (Ht)

The result of figure (5) shows that, the increase of hematocrit increases the body temperature; this is due to the fact that, the hematocrit increases the density of the blood, and that provokes an increase in resistance of the blood flow (Shung et al 1992 and Tsafack et al 2019).

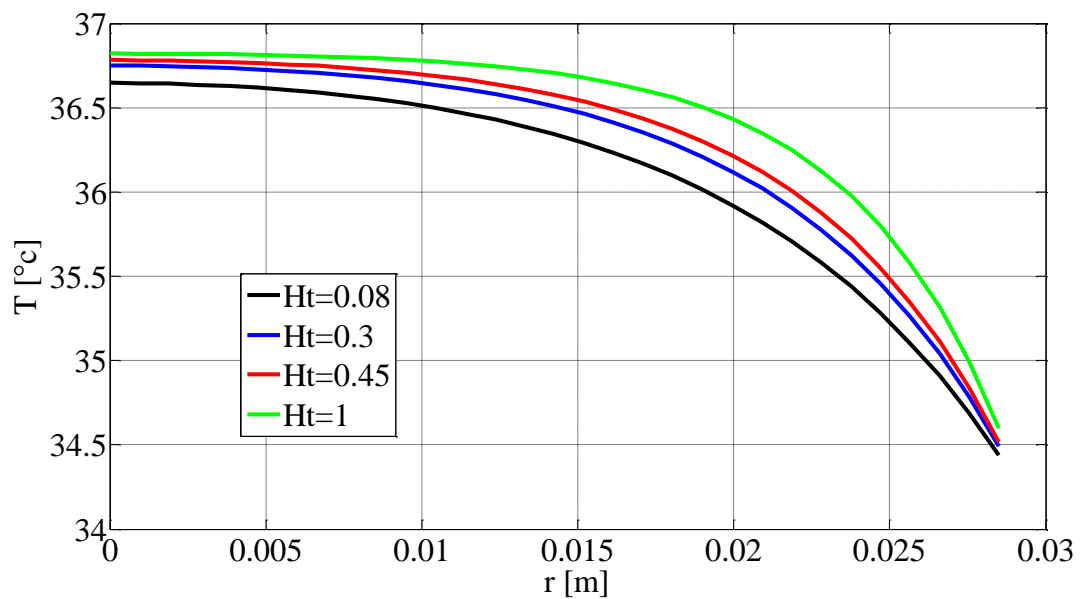


Figure (5): Influence of hematocrits (ht) on the temperature

III-4-2) INFLUENCE OF THE GENERATION OF METABOLIC CHARGER (q_m)

Figure (6) shows that, increasing (q_m) increases the temperature. This is due to the fact that the metabolic generates heat, which causes the body temperature to rise.

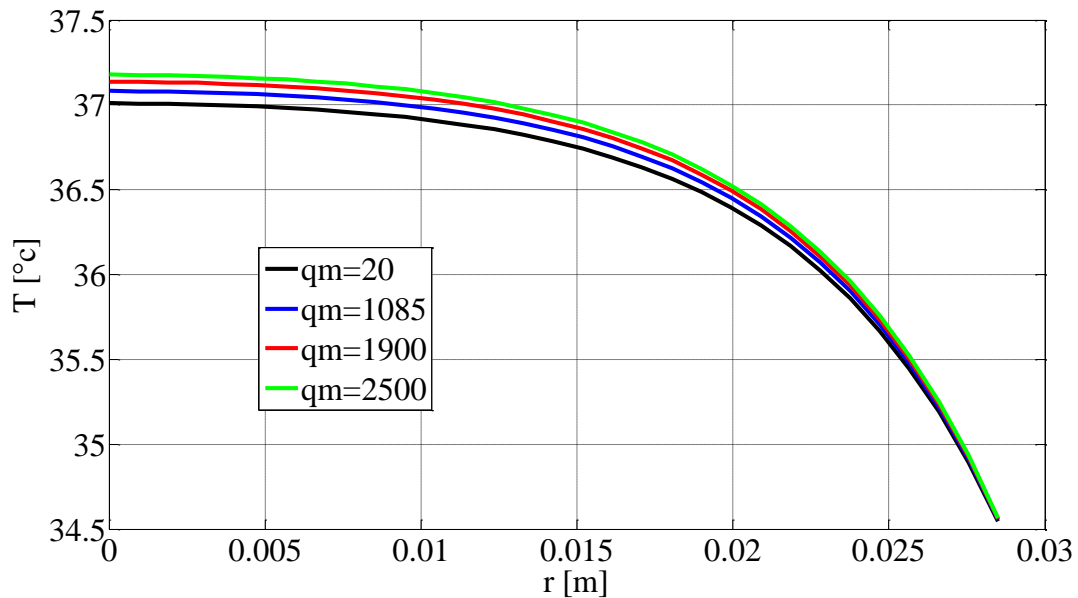


Figure (6): Influence of the generation of metabolic charger (q_m) on temperature

II-4-3) INFLUENCE OF ARTERIAL TEMPERATURE

Figure (7) shows that increasing arterial temperature increases body temperature. Furthermore we observe that the arterial temperature is equal to the body temperature ($T_a = 36.5; 37; 37.5; 38$ is equal to body temperature $T = 36.5; 37; 37.5; 38$ respectively)

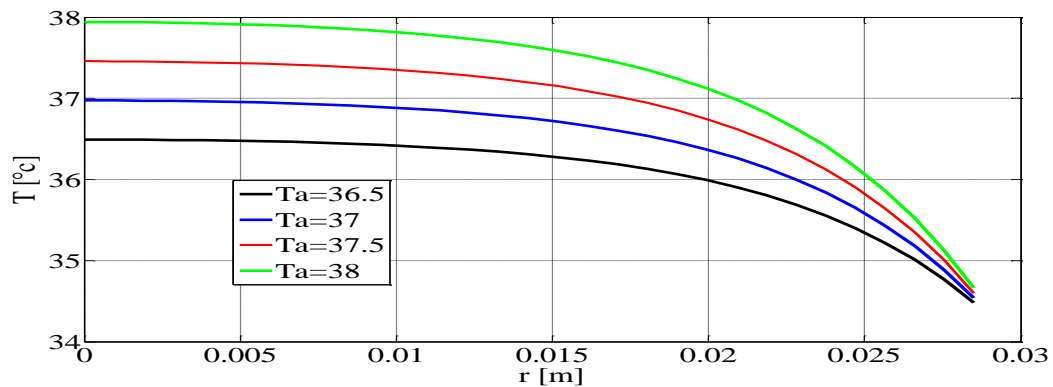


Figure (7): Influence of arterial temperature on the skin temperature

CONCLUSION

In this article we have studied the distribution of the temperature of biological tissue, taking into account stenosis and hematocrit. The heat transfer model of penne in one-dimensional biological heat transfer in steady state has been solved by the numerical and analytical method, to obtain the temperature changes with variation in the size of the stenosis and hematocrit. The results of this contribution brought a new technic to use temperature to control the evolution of stenosis; this is to improve the prediction of cardiovascular and cerebral diseases. The results provided by this article promote the knowledge to control the evolution size of stenosis by taking just a temperature of the body. These provide a good knowledge of the thermal behavior of biological tissue, which is precious for the measurement of thermal parameters, the reconstruction of a temperature field and the diagnosis and treatment of cardiovascular and cerebral disease.

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