

ENC-2022-0527

SIMPLIFIED MATHEMATICAL MODEL OF THERMAL RESPONSE IN ABDOMEN AFTER ABDOMINOPLASTY

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Abstract. *The search for beauty and perfection brings with it an increase in the number of plastic surgeries. Thus, there is a need for effective postoperative approaches and immediate observation of possible complications, which can lead to more efficient procedures. Among the plastic surgeries, the aforementioned study selected the abdominoplasty surgery as a reference and a certain area 12 x 12 cm, assuming a size in the infra umbilical region, which will supposedly be in the process of tissue repair and with possible temperature changes on the skin surface. The creation of a simplified 2D mathematical modeling to predict the thermal response of the abdomen is presented here. The model considers the local metabolic activities of a healthy skin before the surgical intervention and after it, considering an obstruction of blood flow at a certain point. The model considers such changes by applying mass and energy conservation equations where the amount of O₂ supplied regulates the local temperature. Therefore, the reduction of oxygen supplied to the tissues causes a drop in the local temperature representing poor circulation. Decreased local temperature can cause tissue death. Determining the period of low metabolism can be crucial for choosing a treatment and avoiding complications, financial expenses and suffering.*

Keywords: mathematical modeling, post-surgical, abdominoplasty, thermal activity, energy flow.

NOMENCLATURE

T_{∞} = Ambient temperature, K
 T_i = Temperature of the control volume “i”, K
 \dot{Q}_{gen} = Heat generated by the cell metabolism, W
 \dot{Q}_{lost} = Heat lost to the environment, W
 m = Mass, kg
 \dot{V} = Volumetric flow rate, L/s
 $[O_2]_i$ = Oxygen concentration of the control volume “i”, %
 R = Speed of the reaction according to Michelis-Menten model, mol/L.s
 α = Obstruction factor (%)

1. INTRODUCTION

Abdominoplasty is one of the most performed procedures in plastic surgery in the world, accounting for 15.9% of plastic surgery cases performed in Brazil, according to the Brazilian Society of Plastic Surgery (Fonseca A, Ishida, 2018). It consists of resection of the entire infraumbilical flap and in some cases combined vertical resection, indicated for people who have excess skin in the abdominal region. (Porchat et al., 2004).

More attentive care in the immediate postoperative period, especially monitoring the inflammatory process inherent to tissue healing, is directly linked to the reduction of complications after abdominoplasty (Di Martino et al., 2010) and (Xia Y. et al., 2019).). Vascular changes with increased blood flow and metabolic activity may be related to abnormalities even when the changes are not yet present in other tests (Brioschi, 2016) . The human body is an efficient heat radiator, regardless of pigmentation or skin color, and it provides a means for a non-invasive method of investigation by thermography (Ring et al., 1968) and (Ring et al., 1977) and (Ring et al., 1977) and (Ring et al., 1978).

A thermal change can be hypothermia, defined as a body temperature below 36.8°C. It is an inevitable pathophysiological consequence of serious injury (Jurkovich et al., 1987). Brandon Bravo, 1999, introduced a simple mathematical model in an attempt to describe the thermal steady state in trauma patients. The mathematical model presented here is defined for an estimated area of 12 cm by 12 cm, assuming a range of abdominal area according to Huger (1979) and seen by Matarasso (1996) as zones that take into account the vascular anatomy of the infraumbilical abdominal wall and the blood supply of this region.

Historically, Pennes, in 1948, was the first to propose a model of heat transfer in human skin through radial temperature distribution. This model gained wide acceptance for presenting data compatible with experimental results (Pennes HH 1948). The present work, based on the Volume Elements Model (Dilay et al., 2015) and (Vargas et al., 2001) , presents a simplified mathematical model to obtain the thermal response of the region where the abdominoplasty took place. The domain of interest is discretized using finite volumes with centered cells, and principles of classical thermodynamics and heat transfer are applied to each cell, resulting in a system of ordinary differential equations for the transient regime, using empirical and analytical correlations to quantify the energy interactions between cells.

Such principles are applied initially in an area without blood flow obstruction, as if assuming before a surgical intervention, and the thermal response is analyzed. In this same area, obstructions are then considered, assuming a vascular intercurrence. Obstructions are represented by reduced oxygen supply to cells. The reduction of oxygen supplied to the tissues causes a drop in the local temperature representing poor circulation. The numerical results obtained are the transient response of the temperature of the abdomen and will be used as a tool for the evaluation of complications and postoperative treatment and rehabilitation.

2. MATHEMATICAL MODEL

In order to quantify the temperature variation in the specified area of the abdomen (12cm x 12cm) a simplified 2D mathematical model was developed based on thermal exchanges and energy balance to investigate the effect of an obstruction in vascular flow on the surface temperature of the skin in the pre-established regions.

The model studies the behavior of oxygenation and temperature of a hypothetical tissue in the infra-umbilical region of the abdomen when there is an obstruction of blood flow in a part of this region. Although this area has an irregular geometry, for the computational domain in this study, a square geometry of 12 cm by 12 cm was defined, divided into 9 control volumes, with dimensions of 4 cm by 4 cm, as shown in fig. 1(a). Arrows indicate the direction of blood flow.

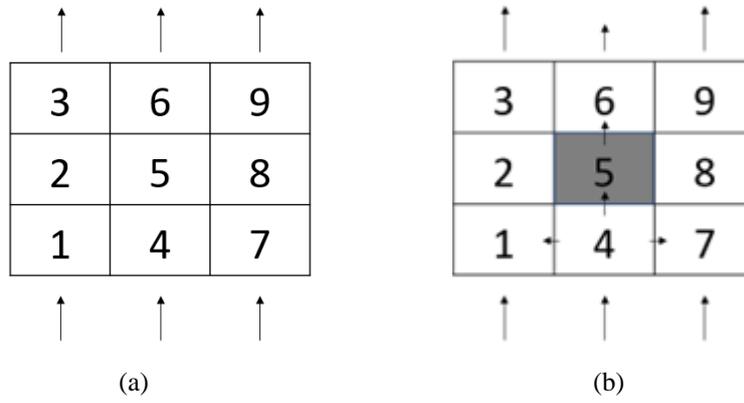


Figure 1. Discretization of abdominal area in 9 control volumes for conditions (a) normal blood flow (no obstruction) and (b) obstruction of blood flow in control volume 5 causing increased blood flow in control volumes 1 and 7 .

This arrangement was necessary to simulate the effect of obstruction, α on control volume 5 and adjacent control volumes. Blood flow in control volume 5 is reduced by a factor α and adjacent control volumes, 1 and 7, have increased blood flow of $(1 - \alpha)/2$, as shown in fig. 1(b).

Mass and energy conservation equations were applied to each control volume represented in fig. 1. Consequently, two equations were formulated for each control volume (VC_i). As there are different behaviors in certain control volumes, the equations were grouped as follows: VC_4 , $VC_{1,7}$, $VC_{5,6}$, $VC_{2,3,8,9}$. VC_4 represents the entry of blood in the second column and is the only one not affected by the obstruction α , because, even if there is an obstruction, it was assumed that the flow would not be dammed, but redistributed equally to the control volumes 1 and 7. $VC_{1,7}$ represents the entry of blood flow in the first and third columns, however, according to α , there will be a contribution from the outflow of blood from the control volume 4. $VC_{5,6}$ represents the volumes negatively affected by the obstruction α . As there is conservation of mass, the flow leaving control volume 5 is the same that enters control volume 6, so the same equations can be used in both. Finally, $VC_{2,3,8,9}$ represents the control volumes that receive the flow from 1, in the first column, and from 7, in the third column.

There are, therefore, eight equations, divided into 4 pairs, that model the behavior of the temperature and oxygenation of the mesh. The first equation of each pair, referring to mass conservation, takes into account the consumption of oxygen by metabolic activities according to the Michaelis-Menten equation. This equation will be referred to later as the ‘‘Oxygenation equation’’. In general, it can be described by the derivative of the oxygen concentration of the control volume i ($[O_2]_i$) in time, which is equal to the ratio between the volumetric flow rate of blood (\dot{V}) and the blood volume (\mathcal{V}), multiplied by the difference in the oxygen concentration from the control volume in question ($[O_2]_i$) and the immediately preceding control volume ($[O_2]_{i-1}$), then the portion corresponding to the oxygen consumed by metabolism in the control volume is subtracted, given by the Michaelis-Menten equation (R), which corresponds to the ratio of the product of the maximum rate of the enzymatic reaction (V_{max}) by the oxygen concentration of the control volume in question ($[O_2]_i$) by the sum of the Michaelis-Menten constant (K) and the oxygen concentration of the control volume in question ($[O_2]_i$). The volumetric flow rate of blood (\dot{V}) can be obtained by the ratio between the mass flow rate of blood (\dot{m}) and the density of blood (ρ_{blood}), assumed to be equal to water’s density.

The second equation of the pair models temperature from the energy balance, assuming that there is, in addition to heat exchange from blood flow, heat generated by metabolism and heat lost to the environment in the form of convection. This equation will be referred to later as the ‘‘Temperature Equation’’. In general, it can be described by the product between the mass flow rate of blood (\dot{m}), its specific heat (c_p) and the difference between the inlet temperature of the control volume in question (T_{i-1}) and the temperature of the control volume in question, which coincides with the outlet temperature of this same control volume (T_i), to which the heat generated by metabolism is added (Q_{gen}) and the heat lost to the environment is subtracted (Q_{lost}). This entire portion is then multiplied by the inverse of the product of the blood mass (m) by its specific heat (c_p) and this is equated with the derivative of the temperature of the control volume in question (T_i) in time. Q_{gen} is obtained by multiplying the Michaelis-Menten equation (R) by the blood volume (\mathcal{V}) and by the proportionality constant (Q_0), while Q_{lost} is given by the product of the difference between the temperature of the control volume in question (T_i) and the ambient temperature (T_∞) by the convection coefficient (h) and the surface area of the control volume (A).

$$\frac{d[O_2]_i}{dt} = \frac{\dot{V}}{\mathcal{V}} \{ [O_2]_{i-1} - [O_2]_i \} - R_i, \text{ with } R_i = \frac{V_{max} [O_2]_i}{K + [O_2]_i} \text{ and } \dot{V} = \frac{\dot{m}}{\rho_{blood}} \quad (\text{Oxygenation Eq.})$$

$$\frac{dT_i}{dt} = \frac{1}{mc_p} \left[\dot{m} c_p (T_{i-1} - T_i) + Q_{gen,i} - Q_{lost,i} \right], \text{ with } Q_{gen,i} = \left(\frac{V_{max} [O_2]_i \mathcal{V}}{K + [O_2]_i} \right) Q_0 \text{ and } Q_{lost,i} = hA(T_i - T_\infty) \quad (\text{Temperature Eq.})$$

Purely applying the oxygenation and temperature equations to VC₄ obtain equations (1) and (2), since there is no need to compensate for any relationship with the obstruction α .

$$\frac{d[O_2]_4}{dt} = \frac{\dot{V}}{V} \{ [O_2]_0 - [O_2]_4 \} - R_4 \quad (1)$$

$$\frac{dT_4}{dt} = \frac{1}{mc_p} \left[\dot{m}c_p(T_0 - T_4) + Q_{gen,4} - Q_{lost,4} \right] \quad (2)$$

For VC_{1,7}, one must consider the portion of flow from control volume 4 as a function of obstruction α . Resulting in equations (3) and (4), in which $i = 1$ or 7 .

$$\frac{d[O_2]_i}{dt} = \frac{\dot{V}}{V} \left\{ [O_2]_0 - [O_2]_i \left[1 + \frac{\alpha}{2} \right] + \frac{\alpha}{2} [O_2]_4 \right\} - R_i \quad (3)$$

$$\frac{dT_i}{dt} = \frac{1}{mc_p} \left\{ \frac{\dot{m}c_p}{2} [2(T_0 - T_i) + \alpha T_4 - \alpha T_i] + Q_{gen,i} - Q_{lost,i} \right\} \quad (4)$$

If for VC_{1,7} the addition of terms that compensate for the increase in flow from control volume 4 occurs, for VC_{5,6} this portion must be discounted, since the increase in obstruction will decrease the inflow of blood in control volume 5 and consequently in the control volume 6. Thus, equations (5) and (6) were obtained, with $i = 5$ or 6 .

$$\frac{d[O_2]_i}{dt} = \frac{\dot{V}}{V} (1 - \alpha) ([O_2]_{i-1} - [O_2]_i) - R_i \quad (5)$$

$$\frac{dT_i}{dt} = \frac{1}{mc_p} \left\{ (1 - \alpha) \dot{m}c_p (T_{i-1} - T_i) + Q_{gen,i} - Q_{lost,i} \right\} \quad (6)$$

For the last control volumes VC_{2,3,8,9}, the output of control volumes 1 and 7 is considered, which is the combination of the flow coming from 1 or 7 plus half of the flow that was prevented from going to control volume 5, due to the obstruction. Resulting in equations (7) and (8), with $i = 2, 3, 8$ or 9 .

$$\frac{d[O_2]_i}{dt} = \frac{\dot{V}}{V} \left[\left(1 + \frac{\alpha}{2} \right) ([O_2]_{i-1} - [O_2]_i) \right] - R_i \quad (7)$$

$$\frac{dT_i}{dt} = \frac{1}{mc_p} \left\{ \dot{m}c_p \left[(T_{i-1} - T_i) + \frac{\alpha}{2} (T_{i-1} - T_i) \right] + Q_{gen,i} - Q_{lost,i} \right\} \quad (8)$$

Equations (1) (8) form a system of differential equations that were numerically integrated using the 4th order Runge-Kutta method. Then, the "Matlab" software was used to solve and plot the graphs that will be discussed in the results. Below is the table containing the variables which had their values fixed.

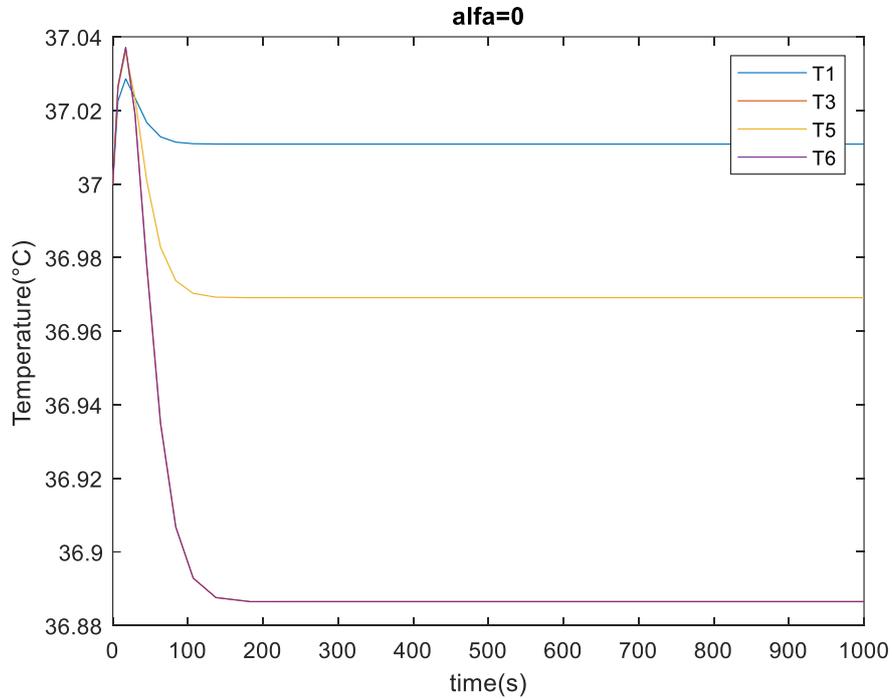
Table 1 - List of variables with their respective fixed values and units.

Symbol	Description	Value	Unit
\dot{m}	Blood mass flow	0.001	kg/s
\dot{V}	Blood volume	0.016	L
ρ_{blood}	Blood density	1	kg/L
c_p	Blood specific heat	4.18	J/kgK
T_0	Control volume inlet temperature	310.15	K
Q_0	Proportionality constant	60	J/mol
h	Convection coefficient	1306.25	W/ m ² K
A	Control volume surface area	0.0016	m ²
V_{max}	Max oxygen consumption rate	0.5	mol/s
K	Michaelis-Menten constant	18	Dimensionless
$[O_2]_0$	Initial oxygen concentration	100	%

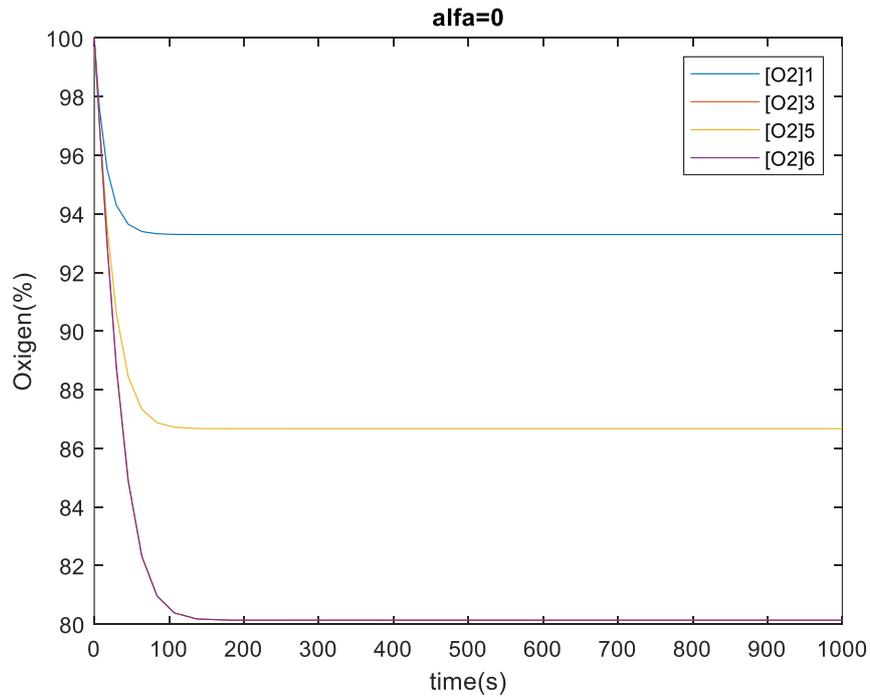
3. RESULTS

Graphs 1 to 8 show the behavior of temperature and oxygenation of control volumes as a function of time for certain obstructions α . Due to the existing symmetry in the proposed mathematical modeling, the other control volumes were not represented because they present identical behavior.

For $\alpha = 0$, before the procedure, represented by graphs 1 and 2, there is no significant temperature difference between the control volumes and there is a decrease in oxygenation as there is oxygen consumption in each previous control volume, with equal values in control volumes 3 and 6.

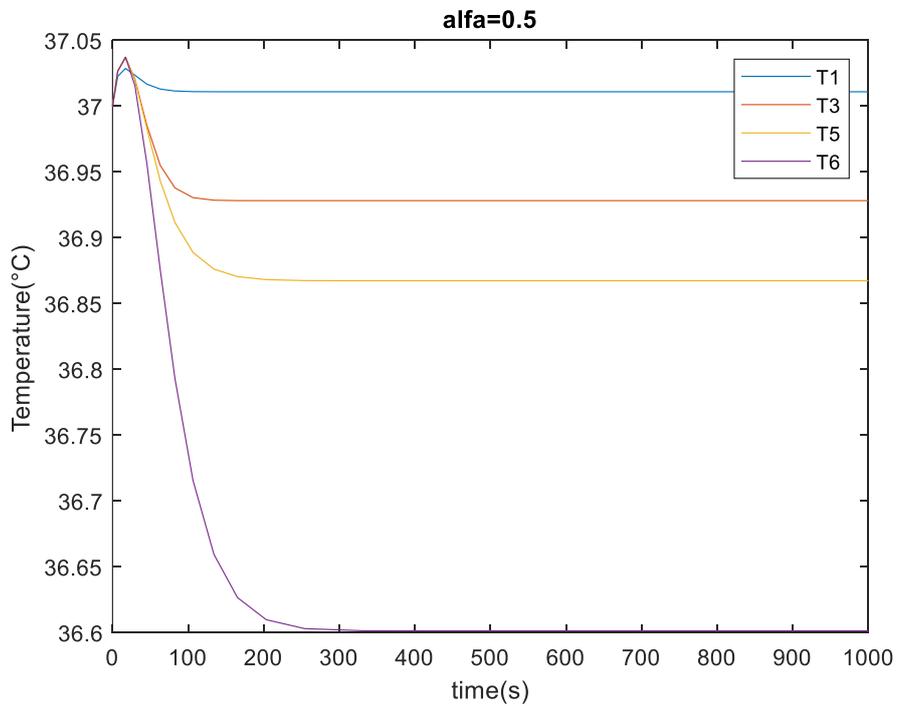


Graph 1 – Temperature versus time with normal blood flow (no obstruction).

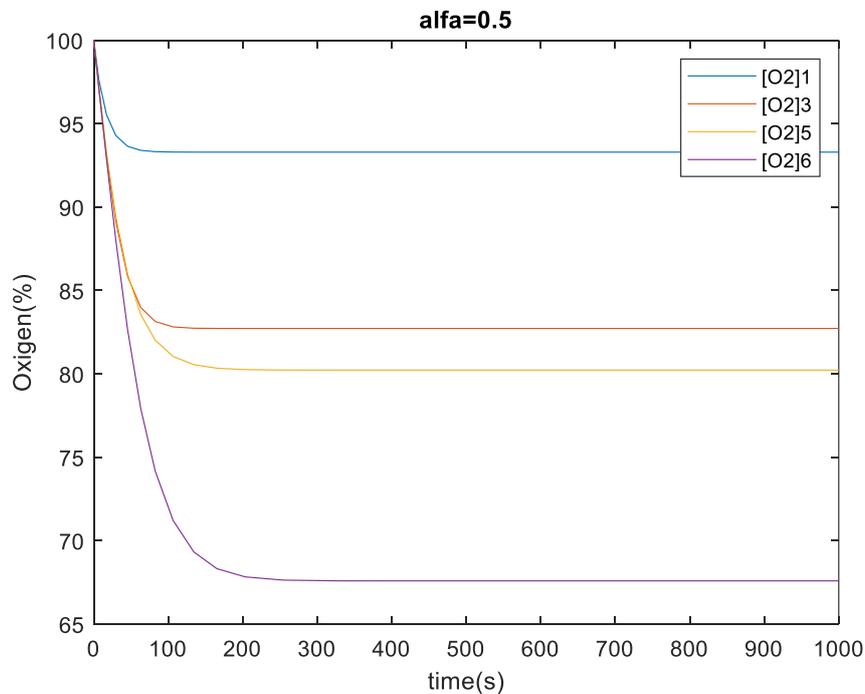


Graph 2 - Oxygen versus time with normal blood flow (no obstruction).

For $\alpha = 0.5$, that is, with 50% of obstruction, it can be seen in graph 3 a temperature decrease of approximately 0.5°C between control volumes 1 and 6, however for the other control volumes the difference in temperature is not significant. For oxygenation it is possible to observe, in graph. 4, a percentage increase in control volumes 1 and 3, more markedly in this one than in that one, and a decrease in oxygenation in control volumes 5 and 6, as expected given the reduced blood flow.



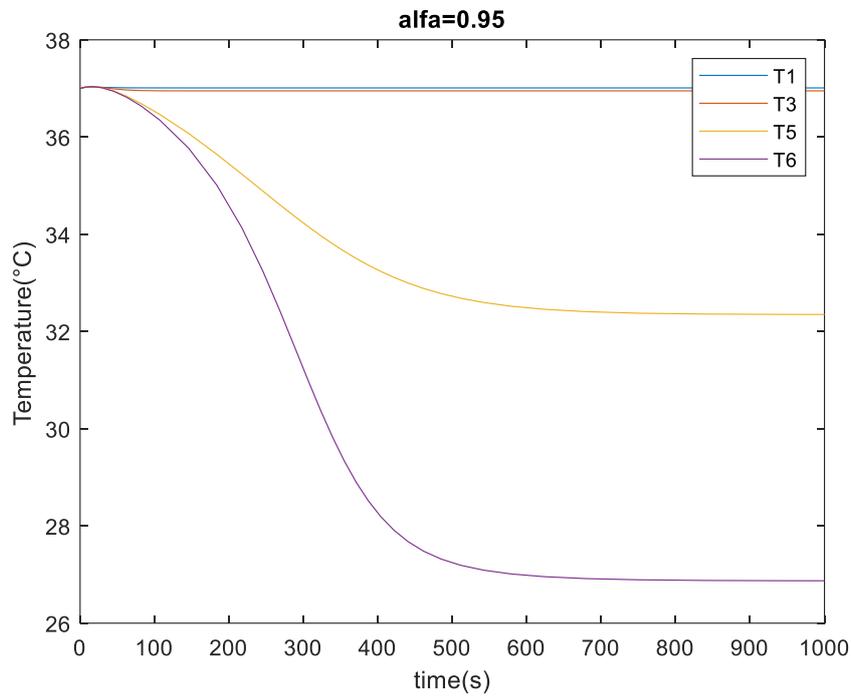
Graph 3 - Temperature as a function of time for blood flow obstruction by 50%.



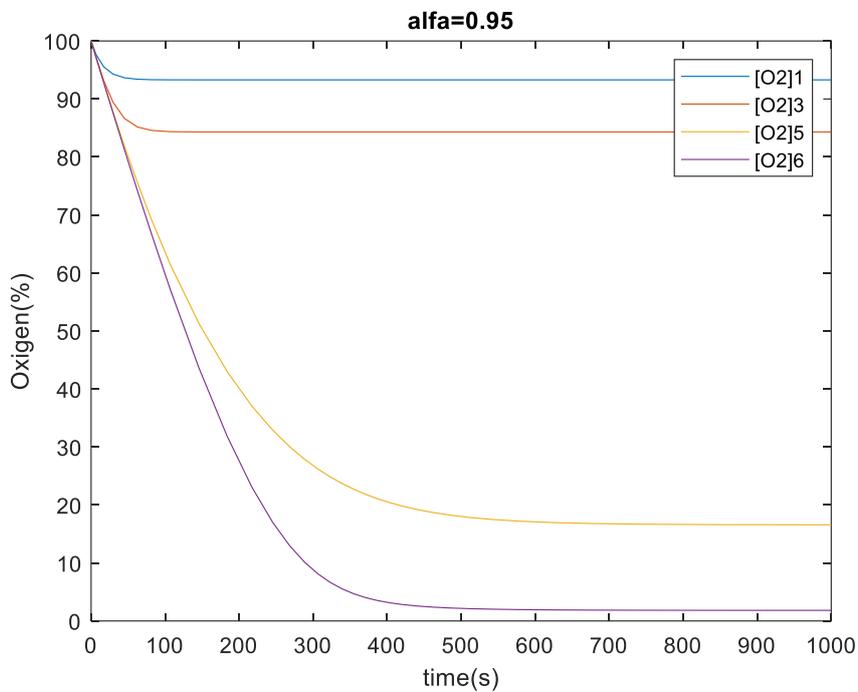
Graph 4 – Oxygen versus time for 50% blood flow obstruction.

With an obstruction close to total, $\alpha = 0.95$, that is, with 95% of obstruction, a sharp increase in the temperature difference can be noticed, represented in the graph 5. While Control Volumes 1 and 3 are maintained at optimal body

temperature, control volumes 5 and 6 have their temperatures lowered dramatically, with a difference of approximately 10°C between control volume 6 and its analog control volume, 3. Regarding oxygenation, visible in graph 6, in control volumes 5 and 6 there is an extremely sharp drop, with values close to 0% for volume 6, indicating a possible tissue death by hypoxia.



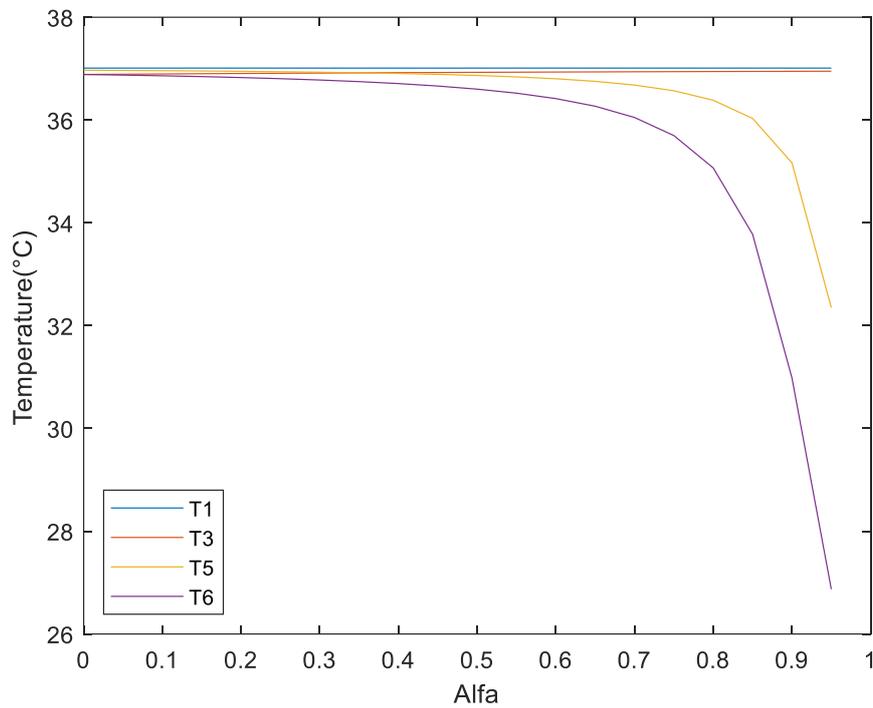
Graph 5 - Temperature versus time for 95% blood flow obstruction.



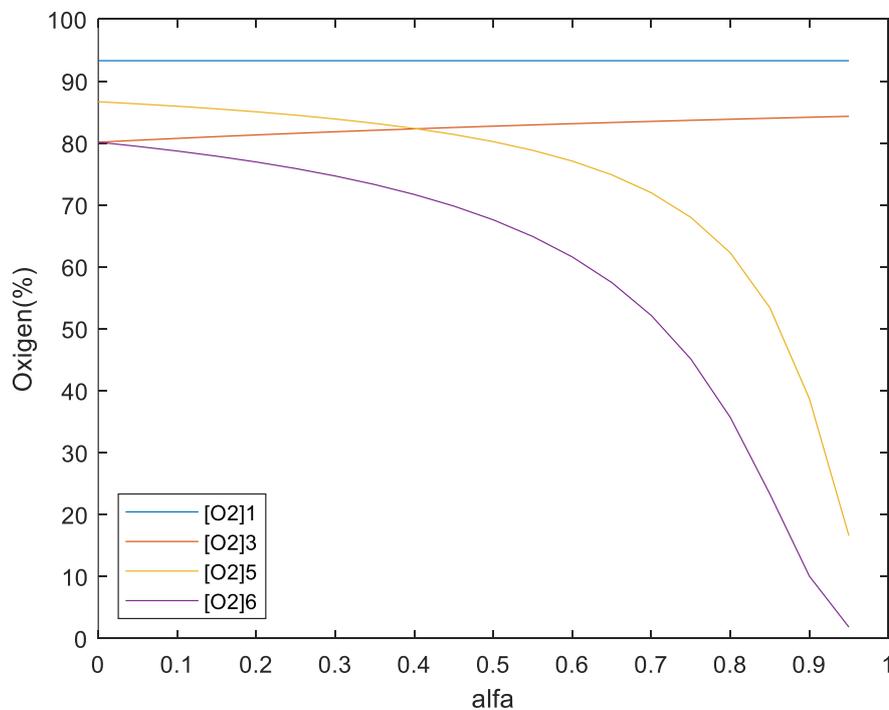
Graph 6 - Oxygen versus time to 95% blood flow obstruction.

Graphs 7 and 8 represent temperature and oxygenation, respectively, as a function of obstruction α . There is evidence of an exponential behavior in the temperature drop from 70% of obstruction, however, with a difference of approximately

0.5°C, it would already be possible to identify obstructions from 50% with a thermographic mapping of the region. It is also clear that oxygenation, in this model, is much more sensitive to obstruction than temperature, and if it were possible to map tissue oxygenation, it would be possible to identify obstructions starting at 20%.



Graph 7 – Temperature as a function of obstruction.



Graph 8 – Oxygen as a function of obstruction.

4. CONCLUSION

In this work, a simplified mathematical model was presented in a tissue area and with variations of obstructions in the flow of blood passage, consequently of oxygen. It can be seen that the results achieved are similar to reality. Temperature and oxygenation in the control volumes that had blood flow restricted by obstruction were reduced, while oxygenation and temperature were maintained in the other control volumes. It can also be concluded that using temperature as a way of verifying the existence of obstruction is viable for obstructions greater than 50% and using methodologies and apparatus that allow the identification of changes in the order of 0.5°C. Thermal images associated with mathematical models of the skin's thermal response have the potential to be effective tools to aid in the diagnosis of vascular alterations, which implies a more adequate predictive approach.

5. ACKNOWLEDGMENTS

The authors are grateful for the support of the national scientific and technological development council, CNPq.

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