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MODELLING OF 1D BIOHEAT TRANSFER WITH PERFUSION COEFFICIENT DEPENDENT ON TISSUE NECROSIS

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Abstract. The temperature distribution in the tissue subjected to a flash fire is dependent, first of all on its thermophysical parameters. In particular, the blood perfusion coefficient is dependent on the degree of tissue necrosis described by tissue injury integral. In the paper a bioheat model basing on the Pennes equation and the Arrhenius scheme is presented.

1. Governing equations

Up to the present, the most common model describing temperature distribution in the tissue domain subjected to heating, as a rule, bases on the Pennes bioheat equation in the form (1D problem) [1-3]:

$$0 < x < L: \quad c\frac{\partial T(x,t)}{\partial t} = \lambda \frac{\partial^2 T(x,t)}{\partial x^2} + c_B G_B \left[T_B - T(x,t)\right] + Q_{met} \tag{1}$$

where λ [W/(mK)] is the thermal conductivity, c [J/(m^3 K)] is the volumetric specific heat, G_B [(m^3 blood/s)/(m^3 tissue)] is the blood perfusion rate and Q_{met} [W/m^3] is the metabolic heat source. The blood parameters c_B [J/(m^3 K)] and T_B denote the volumetric specific heat and the artery temperature, respectively, while T, x, t correspond to the tissue temperature, spatial co-ordinate and time.

One of the flaws of this model is that destruction of tissue during heating has not any effect on parameters values. When tissue temperatures reach 60 to 65°C, proteins are denatured and tissue necrosis can be expected. Over 100°C, water in tissue changes phase, increasing pressure in tissue resulting in explosive vaporization and shutting down the vasculature (thrombosis). When temperature is above 150°C proteins are broken down, releasing hydrogen, nitrogen and oxygen, leaving layer of carbonization.

Tissue damage could be predicted by means of an Arrhenius integral formulation [1, 4]

$$\theta(x) = \int_0^{t^F} A \exp\left[-\frac{\Delta E}{RT(x,t)}\right] \mathrm{d}t \tag{2}$$

where A is the pre-exponential factor [s^{-1}], ΔE is the activation energy for the reaction [J/mole] and R is universal gas constant [J/(mole·K)].

The accepted criterion for complete tissue necrosis is [1, 4]

$$\theta(x) \geq 1 \tag{3}$$

Because necrosis results from damage of tissue vasculature, so the perfusion coefficient G_B is dependent on the tissue injury integral (2). The following approximation of function G_B can be assumed [4]

$$G_B(\theta) = \begin{cases} (1 + 25\theta - 260\theta^2) G_{B0}, & 0 < \theta \leq 0.1 \\ (1 - \theta) G_{B0}, & 0.1 < \theta \leq 1 \end{cases} \tag{4}$$

where G_{B0} denotes the rate of perfusion in totally undamaged tissue.

The first equation in (4) shows that perfusion rate increases as tissue is heated and vasodilation occurs, while the second reflects blood flow decrease as the vasculature begins to shut down (thrombosis).

Taking into account the equations (1) and (2) the bioheat transfer can be written in the form

$$0 < x < L: \quad c \frac{\partial T(x,t)}{\partial t} = \lambda \frac{\partial^2 T(x,t)}{\partial x^2} + c_B G_B(\theta)\left[T_B - T(x,t)\right] + Q_{met} \tag{5}$$

and $G_B(\theta)$ is described by formula (4).

Equation (5) is supplemented by following boundary conditions

$$\begin{aligned} &x = 0: \quad T(x,t) = T_b, \quad \text{or} \quad q(x,t) = q_b, \\ &x = L: \quad q(x,0) = 0 \end{aligned} \tag{6}$$

and the initial one

$$T(x,0) = T_p \tag{7}$$

2. Examples of computations

On the stage of numerical realization the 1st scheme of boundary element method for linear elements has been applied [5]. In computations the following values of tissue parameters have been assumed: λ = 0.75 W/(mK), $c = 3 \cdot 10^6$ J/(m^3 K), G_{B0} = 0.00125 (m^3 blood/s)/(m^3 tissue), Q_{met} = 245 W/m^3 and L = 35 mm. Parameters appearing in Arrhenius integral equal $A = 3.1 \cdot 10^{98}$ s^{-1}, $\Delta E = 6.27 \cdot 10^5$ J/mole and R = 8.314 J/(mole·K). Blood parameters: $c_B = 3.9962 \cdot 10^6$ J/(m^3 K) and T_B = 37°C. Tissue domain has been divided into 100 elements and the time step Δt = 0.5 s.

In example 1 the constant temperature for $x = 0$ has been assumed $T_b = 75°C$, while for the $x = L$ the no-flux condition could be accepted. Initial distribution of temperature has been assumed as the constant temperature $T_p = 37°C$.

In Figure 1 the temperature distribution in the tissue domain is presented. The two next figures concern results connected with tissue damage. Figure 2 shows the distribution of perfusion coefficient for different times. The effect of tissue necrosis corresponds to left hand side of the peak where value of perfusion coefficient fall down to zero, and on the right side of the peak is visible increase of perfusion rate caused by vasodilation. In Figure 3 the profiles of injury integrals are shown. On the basis of those values and criterion for tissue necrosis (c.f. equation (3)) the depth of necrosis after time 240 seconds was calculated as equal 8.4 mm.

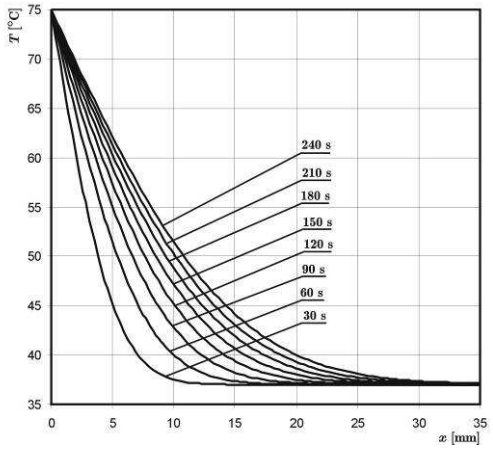

Fig. 1. Temperature distribution (example 1, $T_b = 75°C$)

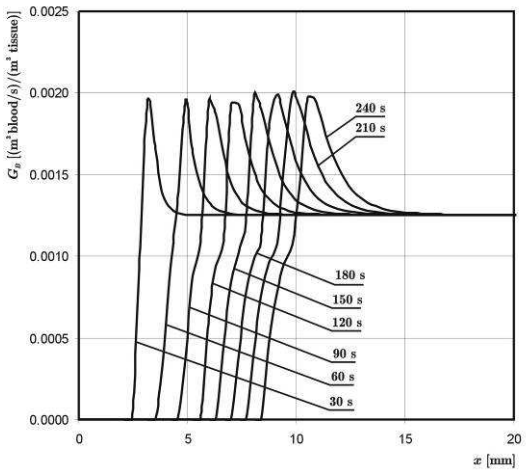

Fig. 2. Perfusion rate distribution (example 1, $T_b = 75°C$)

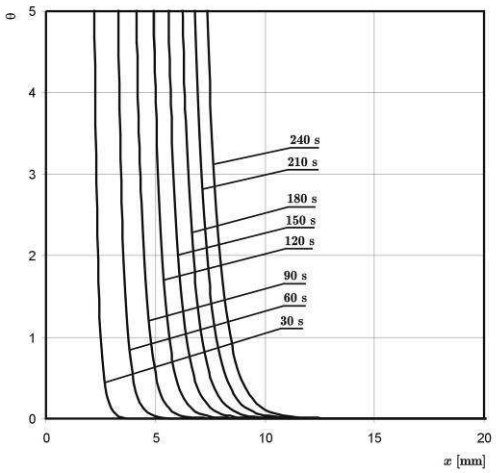

Fig. 3. Profiles of tissue injury integral (example 1, T_b = 75°C)

In example 2 the Neumann condition for $x = 0$ has been assumed and the value of heat flux is equal q_b = 3000 W/m^2. Initial condition and condition for the second boundary were the same as in previous example. The results are presented in the Figures 4, 5 and 6. Similary to former simulation, both the tissue necrosis and perfusion increase produced by vasodilation are very well noticeable in the Figure 5. On the basis of knowledge of Arrhenius integral profiles (Fig. 6) the depth of complete tissue damage after 240 seconds was determinated as equal 5.6 mm, but on the contrary to example 1, the process of necrosis had begun after 76 seconds (in example 1, immediately after calculations starts i.e. after 0.5 second).

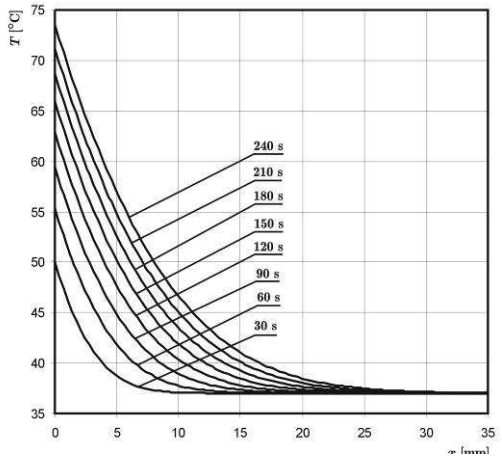

Fig. 4. Temperature distribution (example 2, q_b = 3000 W/m^2)

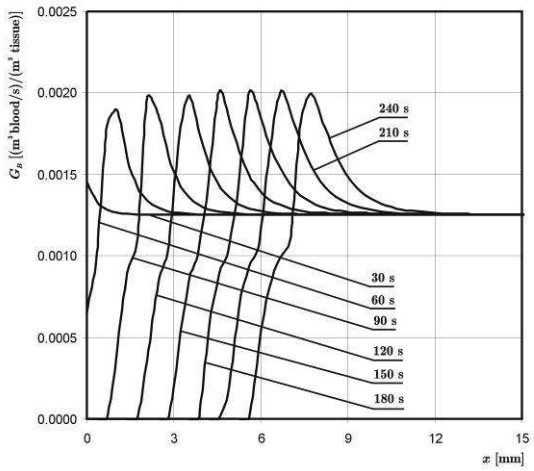

Fig. 5. Perfusion rate distribution (example 2, q_b = 3000 W/m^2)

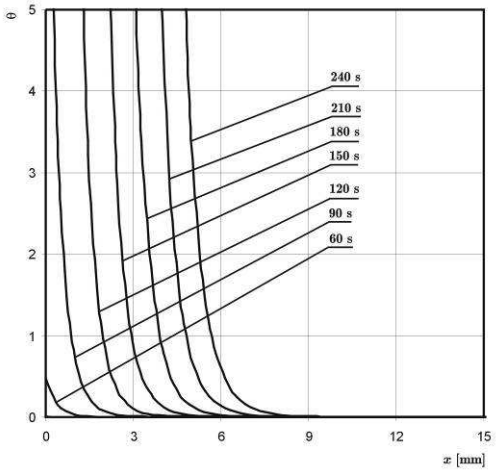

Fig. 6. Profiles of tissue injury integral (example 2, q_b = 3000 W/m^2)

3. Final remarks

The model proposed seems to be closer to the real conditions of heat transport in the living tissue subjected to intensive external heating. Negative effects of heating have visible influence on tissue perfusion rate. The Arrhenius integral formulation is quite convenient means to modelling such type of problems. It should be pointed out that equation, similarly to (4), could be introduced also for metabolic heat source.

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