Numerical analysis of soft tissue damage process caused by laser action

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Abstract

The numerical analysis of thermal damage process proceeding in biological tissue during laser irradiation is presented. Heat transfer in the tissue is assumed to be transient and two-dimensional. The internal heat source resulting from the laser irradiation based on the solution of diffusion equation is taken into account. The tissue is regarded as a homogeneous domain with perfusion coefficient treated as dependent on tissue injury. At the stage of numerical computations the boundary element method and the finite difference method have been used. In the final part of the paper the results obtained are shown.

Keywords: bioheat transfer, tissue injury, laser-tissue interaction, boundary element method, finite difference method

1. Introduction

Laser irradiation on biological tissue often leads to the temperature elevation that can cause irreversible damage by the alteration of thermophysical and optical properties of tissue. Consequently, parameters applied in mathematical models of heat transfer in biological tissue domain can be regarded as temperature-dependent or tissue damage-dependent. Such kind of processes are usually modeled by the so-called Arrhenius injury integral in which the reaction rate increases exponentially with the temperature [2].

To describe the light propagation in biological tissues the different mathematical models can be taken into account. It is also known that tissues are characterized by a strong scattering and weak absorption in the so-called therapeutic window (wavelengths between 650 and 1300 nm). For this reason in this paper the diffusion approximation is applied [3,4].

2. Governing equations

The 2D domain of homogeneous biological tissue of rectangular shape Ω subjected to the laser action, as shown in Figure 1, is considered.

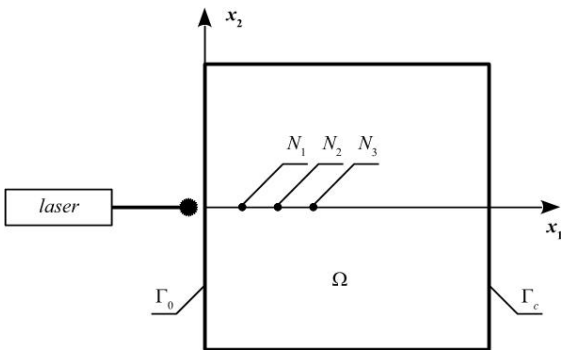

Figure 1: The domain considered

A transient heat transfer in biological tissue is described by the Pennes equation in the form [1-3]

$$\mathbf{x} \in \Omega: \quad c\dot{T} = \lambda \nabla^2 T + Q_{perf} + Q_{las} + Q_{met} \tag{1}$$

where λ [$\mathrm{Wm^{-1}K^{-1}}$] is the thermal conductivity, c [$\mathrm{Jm^{-3}K^{-1}}$] is the volumetric specific heat, Q_{perf}, Q_{met} and Q_{las} [$\mathrm{Wm^{-3}}$] are the heat sources connected with the perfusion, metabolism and laser radiation, respectively, while $T = T(\mathbf{x}, t)$ is the temperature.

In the current work the metabolic heat source is assumed as a constant value while perfusion heat source is described by the formula

$$Q_{perf}(\mathbf{x},t) = c_B w\left[T_B - T(\mathbf{x},t)\right] \tag{2}$$

where w [$\mathrm{s^{-1}}$] is the perfusion coefficient, c_B [$\mathrm{Jm^{-3}K^{-1}}$] is the volumetric specific heat of blood and T_B corresponds to the arterial temperature [2,3].

The source function Q_{las} connected with the laser heating is defined as follows [3]

$$Q_{las}(\mathbf{x},t) = \mu_a \phi(\mathbf{x}) p(t) \tag{3}$$

where μ_a [$\mathrm{m^{-1}}$] is the absorption coefficient, $\phi(\mathbf{x})$ [$\mathrm{Wm^{-2}}$] is the total light fluence rate and $p(t)$ is the function equal to 1 when the laser is *on* and equal to 0 when the laser is *off*.

The total light fluence rate ϕ is the sum of collimated part ϕ_c and diffuse part ϕ_d [3]

$$\phi(\mathbf{x}) = \phi_c(\mathbf{x}) + \phi_d(\mathbf{x}) \tag{4}$$

The collimated fluence rate is given as [1,2]

$$\phi_c(\mathbf{x}) = \phi_0 \exp\left(-\frac{2x_2^2}{r^2}\right)\exp(-\mu_t' x_1) \tag{5}$$

where ϕ_0 [$\mathrm{Wm^{-2}}$] is the surface irradiance of laser, r is the radius of laser beam and μ'_t [$\mathrm{m^{-1}}$] is the attenuation coefficient defined as [2-4]

$$\mu_t' = \mu_a + \mu_s' = \mu_a + (1-g)\mu_s \tag{6}$$

where μ_s and μ'_s [$\mathrm{m^{-1}}$] are the scattering coefficient and the effective scattering coefficient, respectively, while g is the anisotropy factor.

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To determine the diffuse fluence rate ϕ_d the steady-state optical diffusion equation should be solved [3,4]

$$\mathbf{x}\in\Omega: \quad D\nabla^2\phi_d(\mathbf{x})-\mu_a\phi_d(\mathbf{x})+\mu_s'\phi_c(\mathbf{x})=0 \tag{7}$$

where

$$D=\frac{1}{3\left[\mu_a+(1-g)\mu_s\right]}=\frac{1}{3\mu_t'} \tag{8}$$

is the diffusion coefficient.

The Eqn (7) is supplemented by boundary condition

$$\mathbf{x}\in\Gamma: \quad -D\,\mathrm{n}\cdot\nabla\phi_d(\mathbf{x})=\frac{\phi_d(\mathbf{x})}{2} \tag{9}$$

where n is the outward unit normal vector.

On the tissue surface Γ_0, subjected to a laser irradiation, the Pennes equation (1) is supplemented by boundary condition

$$\mathbf{x}\in\Gamma_0: \quad q(\mathbf{x},t)=\alpha(T-T_{amb}) \tag{10}$$

where α [$\mathrm{Wm^{-2}K^{-1}}$] is the convective heat transfer coefficient and T_{amb} is the temperature of surrounding, while on the internal tissue surface Γ_c, the no-flux condition is accepted. The initial distribution of temperature is also known.

Damage of biological tissue resulting from temperature elevation is modelled by Arrhenius injury integral, defined as [2, 3]

$$\Psi(\mathbf{x},t^F)=\int_0^{t^F}P\exp\left[-\frac{E}{RT(\mathbf{x},t)}\right]\mathrm{d}t \tag{11}$$

where R [J $\mathrm{mole^{-1}K^{-1}}$] is the universal gas constant, E [J $\mathrm{mole^{-1}}$] is the activation energy and P [$\mathrm{s^{-1}}$] is the pre-exponential factor. The criterion for tissue necrosis is $\Psi(\mathbf{x})\geq 1$.

The main assumption of the Arrhenius formula is that the damage of tissue is irreversible, so even in the case of very little rise and lowering of temperature the tissue remain damaged. On the other hand, at the initial tissue heating, when the temperature is moderate, that is between 37°C and 45-55°C, the blood vessels in the tissue become dilated without being thermally damaged.

In order to modeling the possibility of withdrawal of tissue injury, when the laser action is ceased, the TTIW algorithm (thermal tissue injury withdrawal algorithm presented in Ref. [2] has been applied.

3. Results of computations

The bioheat problem (1) has been solved using the 1st scheme of the BEM for 2D transient heat diffusion while the optical diffusion equation has been solved by the finite difference method.

The domain considered Ω has size 4×4 cm. The laser irradiation ϕ_0 has been assumed as equal to 3 [W $\mathrm{cm^{-2}}$] with the duration 100 s.

Additionally the perfusion coefficient is treated as dependent on tissue injury, so it's defined as [2]

$$w=w(\Psi)=\begin{cases}\left(1+25\Psi-260\Psi^2\right)w_0, & 0\leq\Psi\leq 0.1\\ (1-\Psi)w_0, & 0.1<\Psi\leq 1\\ 0, & \theta>1\end{cases} \tag{12}$$

where w_0 is the initial perfusion coefficient.

The values of coefficients for the interval from 0 to 0.1 respond to the increase of perfusion coefficient caused by vasodilatation up to the value Ψ = 0.05 (maximum of the function) and the beginning of narrowing of blood vessels, while for the interval 0.1 to 1 they reflect blood flow decrease as the vasculature going to shut down (thrombosis).

In the Fig. 2 and 3 the courses of injury integral and perfusion coefficient at the selected points of the domain are presented (c.f. Fig. 1).

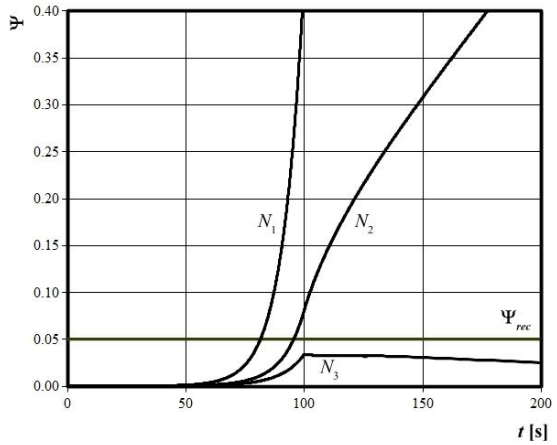

Figure 2: Courses of injury integral Ψ

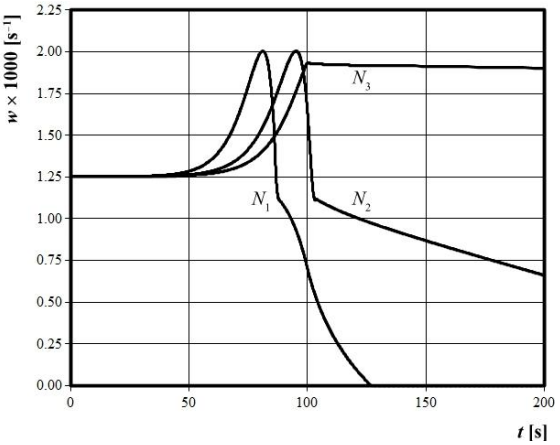

Figure 3: Courses of perfusion coefficient *w*

References

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