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# MODELLING OF LIGHT AND HUMAN SKIN INTERACTION USING KUBELKA-MUNK THEORY

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**Abstract.** The numerical analysis of thermal processes and light distribution in human skin is presented. The Kubelka-Munk theory has been used for modeling diffused light in tissue, while the Arrhenius scheme for modeling coagulation changes in human skin. At the stage of numerical realization, the boundary element method has been used. In the final part of the paper, the results obtained are presented.

## Introduction

The analysis of interaction between light and human skin is important because of the constantly improving techniques to cure various skin lesions like port wine stain (PWS), pigment naevi or tattoo removal. In many cases, the laser treatment of skin is connected with the necessity of blood vessel coagulation, so the selection of a wavelength that coagulates the full thickness of the vessels and simultaneously minimizes damage of the surrounding tissue is the main goal of researches in this area [1, 2].

There are many works in which the modeling of the coagulation phenomenon based on the Arrhenius scheme, usually via some mathematical function describing the blood perfusion rate is presented [3-5].

Human skin has light-scattering properties, which provides some possibilities to model light distribution. In the current paper, the Kubelka-Munk theory is applied, a quite simple but still useful approach. It should be pointed out that this theory also has a few disadvantages [1, 6]. First of all, this theory as almost all theories of the "multi-flux" class, is restricted to one-dimensional tasks. Secondly, the theory is based on the assumption that incidental light is already diffused. No less important is that the coefficients defining absorption and scattering in the Kubelka--Munk theory are not "classical" absorption and scattering coefficients introduced in other approaches, and the relations between them are the major problem of the Kubelka-Munk theory [6].

## 1. Governing equations

Transient heat transfer in successive layers of skin tissue is described by the system of Pennes equations in the form [3-5]

$$L_{e-1} < x < L_e: \quad c_e \dot{u}_e = \lambda_e u_{e,ii} + Q_{perf\,e} + Q_{met\,e} + Q_{las\,e}, \tag{1}$$

where $e$ corresponds to the layers of skin (1 - epidermis, 2 - dermis), $\lambda_e$ [$\mathrm{Wm^{-1}\,K^{-1}}$] is the thermal conductivity, $c_e$ [$\mathrm{Jm^{-3}K^{-1}}$] is the volumetric specific heat, $Q_{perf\,e}$, $Q_{met\,e}$ and $Q_{las\,e}$ [$\mathrm{Wm^{-3}}$] are the heat sources connected with perfusion, metabolism and laser radiation, respectively, $u_e$ is the temperature, $t$ is the time and $x$ denotes the spatial co-ordinate. In equation (1):

$$\dot{u}_e = \frac{\partial u_e}{\partial t}, \quad T_{e,ii} = \nabla^2 u_e \,. \tag{2}$$

In the current work, the metabolic heat source is assumed as a constant value while the parameter appearing in the perfusion heat source (the perfusion rate) depends on the tissue injury expressed by the Arrhenius integral [3, 4]

$$\theta_e(x) = \int_0^{t^F} A_e \exp\left[-\frac{\Delta E_e}{R u_e}\right] \mathrm{d}t\,, \tag{3}$$

where $A_e$ is the pre-exponential factor [$\mathrm{s^{-1}}$], $\Delta E_e$ is the activation energy for the reaction [J $\mathrm{mole^{-1}}$] and $R$ is the universal gas constant [J $\mathrm{mole^{-1}\,K^{-1}}$].
The accepted criterion for complete tissue necrosis is [3, 4]

$$\theta_e(x) \geq 1\,. \tag{4}$$

The perfusion heat source is as follows [4, 5]

$$Q_{perf\,e} = c_B G_{Be}(u_B - u_e)\,, \tag{5}$$

where $G_{Be}$ [($\mathrm{m^3}_{\,blood}$ $\mathrm{s^{-1}}$)/($\mathrm{m^3}_{\,tissue}$)] is the blood perfusion rate, $c_B$ [$\mathrm{Jm^{-3}K^{-1}}$] is the volumetric specific heat of blood while $u_B$ denotes the artery blood temperature [5].

Regarding the necrotic changes in tissue, the blood perfusion coefficient is defined as

$$G_{Be} = G_{Be}(\theta_e) = G_{B0e}\, w(\theta_e)\,, \tag{6}$$

where $G_{B0e}$ is the initial perfusion rate and the function of $\theta_e$ is assumed as a polynomial one [3, 4]

$$w(\theta_e) = \sum_{j=1}^{3} m_j \theta_e^{j-1} \tag{7}$$

where $m_j$ are the coefficients.

Equation (1) is supplemented by the boundary condition on the tissue surface subjected to laser irradiation (external surface)

$$x = 0: \quad q_1 = \alpha(u_1 - u_{amb}), \tag{8}$$

where $\alpha$ [$Wm^{-2}K^{-1}$] is the heat transfer coefficient and $u_{amb}$ is the temperature of the surroundings, while on the internal tissue surface ($x = L$) the adiabatic condition is assumed.

The initial distribution of temperature is also known

$$t = 0: \quad u = u_e \tag{9}$$

## 2. Kubelka-Munk theory

The main assumption of the Kubelka-Munk theory is that the whole light is diffused. Light distribution in the tissue at coordinate $x$ is indicated by two fluxes: $J_n$ and $J_p$ (Fig. 1). Because in theory, convention $x = 0$ corresponds to the rear side of the sample, the fluxes $J_n$ and $J_p$ could be named as the fluxes in a negative and positive direction respectively [1, 6].

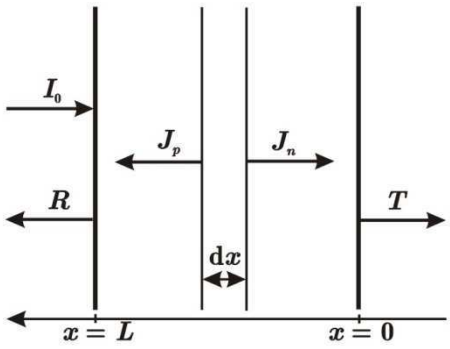


Fig. 1. Two fluxes in Kubelka-Munk theory

Flux $J_n$ corresponds to the forward scattered light, while flux $J_p$ to the backscattered light. The forward scattered flux $J_n$ describes the transmission behavior of the medium represented by transmission coefficient $T$. In a similar way flux $J_p$ gives information about the reflection of tissue stated by reflection coefficient $R$.

The following set of differential equations could be formed

$$\begin{cases} -\dfrac{\mathrm{d}J_n}{\mathrm{d}x} = -\left(A_{KM} + S_{KM}\right) J_n + S_{KM} J_p \\ \dfrac{\mathrm{d}J_p}{\mathrm{d}x} = -\left(A_{KM} + S_{KM}\right) J_p + S_{KM} J_n \end{cases} \tag{10}$$

where $A_{KM}$ and $S_{KM}$ [$\mathrm{m}^{-1}$] are defined for absorption and scattering respectively, but it should be pointed out that these coefficients must be distinguished from a normal coefficient for absorption and scattering which are defined in other approaches [6].
For the set of boundary conditions in form [1]

$$\begin{cases} x = 0: \quad J_p(x) = 0 \\ x = L: \quad J_n(x) = 1 \end{cases} \tag{11}$$

the solution is [1]

$$\begin{cases} J_n(x) = \dfrac{a\sinh(\phi) + b\cosh(\phi)}{a\sinh(\Phi) + b\cosh(\Phi)} \\ J_p(x) = \dfrac{\sinh(\phi)}{a\sinh(\Phi) + b\cosh(\Phi)} \end{cases} \tag{12}$$

where

$$a = \frac{A_{KM} + S_{KM}}{S_{KM}}, \quad b = \sqrt{a^2 - 1} \tag{13}$$

while

$$\Phi = bS_{KM}L, \quad \phi = bS_{KM}x \tag{14}$$

and finally

$$J(x) = J_n(x) + J_p(x) = \frac{(a+1)\sinh(\phi) + b\cosh(\phi)}{a\sinh(\Phi) + b\cosh(\Phi)} \tag{15}$$

On the basis of equation (15), using boundary conditions in the form of (11), one obtains the formulas for the reflection and transmission coefficient [1]

$$R = \frac{\sinh(\Phi)}{a\sinh(\Phi) + b\cosh(\Phi)} \tag{16}$$

and

$$T = \frac{b}{a\sinh(\Phi) + b\cosh(\Phi)} \tag{17}$$

and light intensity $I$ at coordinate $x$ is equal to

$$I(x) = I_0 J(x) \tag{18}$$

where $I_0$ denotes the light incident irradiation on the surface of the medium.
For the two-layer model which is under consideration in the current paper, at first an additional coefficient must be introduced [1]:

$$\tau_1 = \frac{T_1}{1 - R_1 R_2} \tag{19}$$

$$\tau_2 = \frac{T_1 T_2}{1 - R_1 R_2} \tag{20}$$

$$\rho_1 = R_1 + \frac{T_1^2 R_2}{1 - R_1 R_2} \tag{21}$$

$$\rho_2 = \frac{T_1 R_2}{1 - R_1 R_2} \tag{22}$$

Coefficients $\tau_1$ and $\rho_1$ are the reflection and transmission coefficients of the epidermis, while $\tau_2$ and $\rho_2$ correspond to the reflection and transmission coefficients of dermis, respectively. It should be also pointed out that

$$T_{skin} = \tau_2 \tag{23}$$

while

$$R_{skin} = \rho_1 \tag{24}$$

The irradiance inside the epidermis consists of two components: the first connected with light flux entering the tissue from the surroundings, and the second term connected with light flux reflected from the surface of the dermis and entering the epidermis from the rear side. So, we have

$$I_1(x) = I_0 \left[ \frac{(a_1 + 1)\sinh(\phi_1) + b_1\cosh(\phi_1)}{a_1\sinh(\Phi_1) + b_1\cosh(\Phi_1)} + \rho_2 \frac{(a_1 + 1)\sinh(\psi_1) + b_1\cosh(\psi_1)}{a_1\sinh(\Phi_1) + b_1\cosh(\Phi_1)} \right] \tag{25}$$

where

$$\psi_1 = b_1 S_{KM1}(L_1 - x) \tag{26}$$

The dermis sub-domain is subjected to the light incident associated with the light transmitted through the epidermis and then

$$I_2(x) = I_0 \tau_1 \frac{(a_2 + 1)\sinh(\phi_2) + b_2 \cosh(\phi_2)}{a_2 \sinh(\Phi_2) + b_2 \cosh(\Phi_2)}. \tag{27}$$

Finally, returning to the co-ordinate system used in chapter 2, one has

$$Q_{lase}(x) = A_{KMe} I_e(L_e - x). \tag{28}$$

## 3. Boundary element method

The problem has been solved using the 1st scheme of the BEM for 1D transient heat diffusion [4, 7]. This method for equation (1) and transition $t^{f-1} \to t^f$ leads to the formula (for successive sub-domains of skin)

$$\begin{aligned} u_e(\xi, t^f) + \left[ \frac{1}{c_e} \int_{t^{f-1}}^{f} u_e^*(\xi, x, t^f, t) q_e(x,t)\,\mathrm{d}t \right]_{x=L_{e-1}}^{x=L_e} \\ = \left[ \frac{1}{c_e} \int_{t^{f-1}}^{f} q_e^*(\xi, x, t^f, t) u_e(x,t)\,\mathrm{d}t \right]_{x=L_{e-1}}^{x=L_e} + \int_{L_{e-1}}^{L_e} u_e^*(\xi, x, t^f, t^{f-1}) u_e(x, t^{f-1})\,\mathrm{d}x \\ + \frac{1}{c_e} \int_{L_{e-1}}^{L_e} [Q_{perf\,e} + Q_{met\,e} + Q_{las\,e}] \int_{t^{f-1}}^{t^f} u_e^*(\xi, x, t^f, t)\,\mathrm{d}t\,\mathrm{d}x \end{aligned} \tag{29}$$

where $u_e^*$ is the fundamental solution [7]:

$$u_e^*(\xi, x, t^f, t) = \frac{1}{2\sqrt{\pi a_e (t^f - t)}} \exp\left[ -\frac{(x-\xi)^2}{4a_e(t^f - t)} \right] \tag{30}$$

where $\xi$ is the point in which the concentrated heat source is applied and $a_e = \lambda_e / c_e$ while the heat flux resulting from the fundamental solution is equal to

$$q_e^*(\xi, x, t^f, t) = -\lambda_e u_{e,i}^* n_i = \frac{\lambda_e (x - \xi)}{4\sqrt{\pi}[a_e(t^f - t)]^{3/2}} \exp\left[ -\frac{(x-\xi)^2}{4a_e(t^f - t)} \right] \tag{31}$$

and $q_e(x, t) = -\lambda_e u_{e,i} n_i$.

For $\xi \to L_{e-1}^{+}$ and $\xi \to L_e^{-}$ for each sub-domain one obtains the system of equations

$$\begin{bmatrix} g_{11}^e & g_{12}^e \\ g_{21}^e & g_{22}^e \end{bmatrix} \begin{bmatrix} q_e(L_{e-1}, t^f) \\ q_e(L_e, t^f) \end{bmatrix} = \begin{bmatrix} h_{11}^e & h_{12}^e \\ h_{21}^e & h_{22}^e \end{bmatrix} \begin{bmatrix} u_e(L_{e-1}, t^f) \\ u_e(L_e, t^f) \end{bmatrix} + \begin{bmatrix} p_e(L_{e-1}) \\ p_e(L_e) \end{bmatrix} + \begin{bmatrix} z_e(L_{e-1}) \\ z_e(L_e) \end{bmatrix} \tag{32}$$

where

$$h_e(\xi, x) = \frac{1}{c_e} \int_{t^{f-1}}^{f} q_e^*(\xi, x, t^f, t)\,\mathrm{d}t, \quad g_e(\xi, x) = \frac{1}{c_e} \int_{t^{f-1}}^{f} u_e^*(\xi, x, t^f, t)\,\mathrm{d}t \tag{33}$$

while

$$p_e(\xi) = \int_{L_{e-1}}^{L_e} u_e^* u_e(x, t^{f-1})\,\mathrm{d}x, \quad z_e(\xi) = \int_{L_{e-1}}^{L_e} [Q_{perf\,e} + Q_{met\,e} + Q_{las\,e}] g_e(\xi, x)\,\mathrm{d}x \tag{34}$$

Finally, the temperatures at the internal points can be found using the formula

$$\begin{aligned} u_e(\xi, t^f) = h_e(\xi, L_e) u_e(L_e, t^f) - h_e(\xi, L_{e-1}) u_e(L_{e-1}, t^f) - \\ - g_e(\xi, L_e) q_e(L_e, t^f) + g_e(\xi, L_{e-1}) q_e(L_{e-1}, t^f) + p_e(\xi) + z_e(\xi) \end{aligned} \tag{35}$$

## 4. Results of computations

The values of the thermophysical parameters for successive layers of skin are collected in Table 1, while for the blood: $c_B$ = 3.9962 MJ m$^{-3}$ K$^{-1}$ and $u_B$ = 37°C [8].

Table 1

**Thermophysical parameters of skin**

| Parameter | Unit | Epidermis | Dermis |
|---|---|---|---|
| $\lambda$ | Wm$^{-1}$ K$^{-1}$ | 0.235 | 0.445 |
| $c$ | MJ m$^{-3}$ K$^{-1}$ | 4.308 | 3.96 |
| $G_{B0}$ | s$^{-1}$ | 0 | 0.00125 |
| $Q_{met}$ | Wm$^{-3}$ | 0 | 245 |
| $L$ | mm | 0.1 | 2 |
| $A$ | s$^{-1}$ | 3.1e+98 | 3.1e+98 |
| $\Delta E$ | J mole$^{-1}$ | 6.27e+5 | 6.27e+5 |

The coefficients appearing in polynomial function $w(\theta)$ are as follows [3-5]:

$$\begin{aligned} &0 < \theta \le 0.1: \quad m_1 = 1, \quad m_2 = 25, \quad m_3 = -260 \\ &0.1 < \theta \le 1: \quad m_1 = 1, \quad m_2 = -1, \quad m_3 = 0 \end{aligned} \tag{36}$$

The values of these coefficients for the interval from 0 to 0.1 correspond to the increase of the perfusion rate caused by vasodilatation, while for the interval from 0.1 to 1 they reflect a blood decrease as the vasculature begins to shut down (thrombosis) [3].

For boundary condition (8) the following input data have been introduced: $\alpha = 10$ Wm$^{-2}$ K$^{-1}$ and $u_{amb} = 20$°C, while the initial distribution of temperature has been assumed as the parabolic between temperature 32.5°C (external surface of skin) and 37°C (internal surface of skin) [8].

In Table 2, the values of absorption and scattering coefficients for different wavelengths are presented [1] as well as the coagulation time of the whole skin domain predicted for the data assumed and for incident intensity $I_0 = 1$ Wcm$^{-1}$. The coagulation time is predicted on the basis of the blood perfusion rate course at the point corresponding to the internal surface of skin (Fig. 2).

Table 2

**Optical parameters of skin and coagulation time**

| | Epidermis | | Dermis | | |
|---|---|---|---|---|---|
| Wavelength nm | $A_{KM}$ cm$^{-1}$ | $S_{KM}$ cm$^{-1}$ | $A_{KM}$ cm$^{-1}$ | $S_{KM}$ cm$^{-1}$ | $t_{coag}$ s |
| 400 | 85.5 | 30.5 | 25.8 | 0.05 | 25.8 |
| 500 | 52.5 | 28.6 | 12.9 | 14.9 | 27.9 |
| 600 | 32.5 | 28 | 3.66 | 17 | 33.4 |
| 700 | 20.5 | 29 | 0.38 | 17.8 | 53.8 |

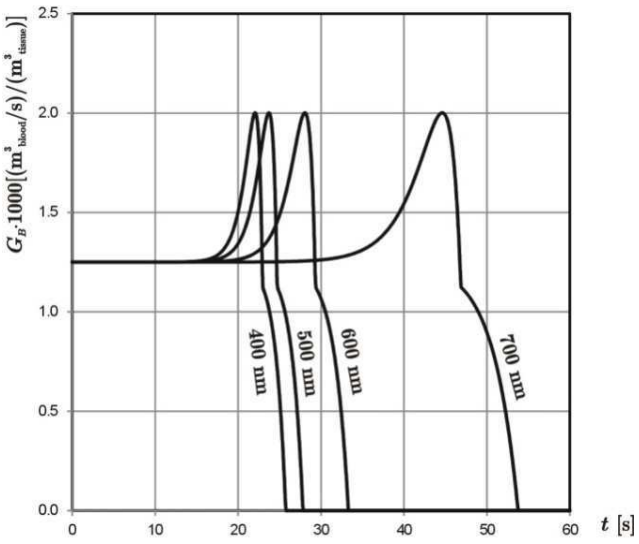


Fig. 2. Courses of perfusion rate $G_B$ at internal skin surface

In Figure 3 the courses of temperature at the internal skin surface for different wavelengths are shown, while in Figure 4 the coagulation energy density supplied to the skin domain within the coagulation time calculated as

$$E_{coag}(x) = I(x)t_{coag} \tag{37}$$

is shown.

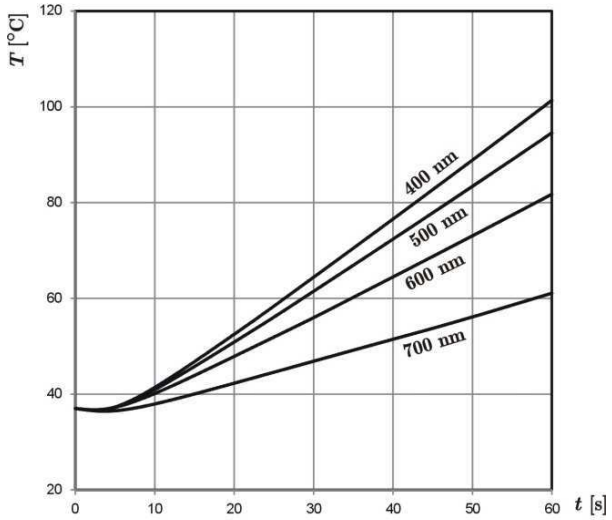


Fig. 3. Courses of temperature at internal skin surface

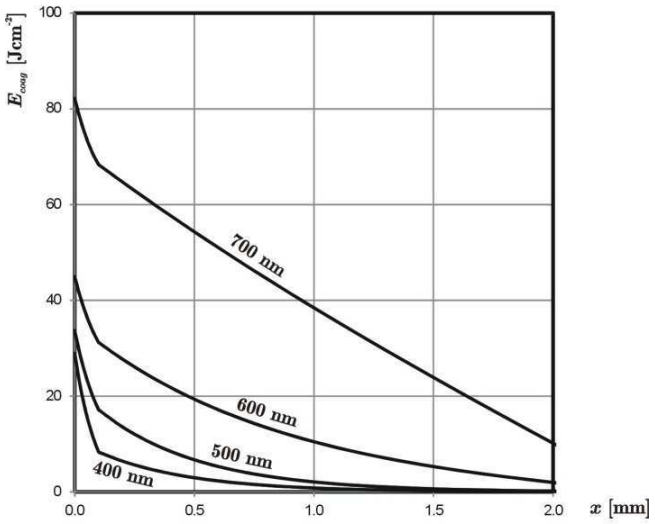


Fig. 4. Coagulation energy distribution in skin

## Final remarks

In the paper the coagulation time of the whole skin domain has been estimated on the basis of the Arrhenius scheme. This time is equal to the time in which the blood perfusion rate at the internal surface of the dermis drops to zero (c.f. Figure 2). Shorter coagulation times are the results of more violent temperature growth (c.f. Figure 3): the maximal temperature at the internal surface of the skin was above 100°C for the 400 nm wavelength, while for the 700 nm about 60°C. It is obvious that the temperature reached at 700 nm is much more comfortable from the patient's point of view, also in order to prevent scar formation and other complications that may take place during the healing process. Furthermore, shorter times of coagulation denote a lower energy density delivery into the tissue (c.f. Fig. 4).

Correct selection of the wavelength to obtain the best results in blood vessel damage in skin should take into account the absorption coefficient for blood [1, 2]. The wavelength selected ought to maximize the absorption of laser light in the blood and minimize absorption in the epidermis as well. It is well known that this coefficient has the greatest values (about few thousand $cm^{-2}$) at about 420 nm, a quite large value (about few hundreds of $cm^{-2}$) between 520 and 580 nm, but for a wavelength over 580 nm the values drop to about a few or less than one $cm^{-2}$ at 700 nm [1-3]. The most frequently suggested wavelength for blood vessel coagulation in skin are those about 580 nm [2], although there are some works which suggest rather a wavelength closer to 420 nm [1].

There are a few estimations in literature, based on experimental data, which allow one to estimate the depth of coagulation based on the energy density value provided to the tissue. For example in [1] the value of about 9 J $cm^{-2}$ is indicated as needed to destroy a vessel of 0.3 mm diameter.

If we use this criterion for the results obtained and presented in Figure 4, we can say that vessel coagulation is possible to the depth of: 0.08, 0.3, 1.1 and 2 mm for wavelengths 400, 500, 600 and 700 nm, respectively. The value for 400 nm corresponds to the epidermis sub-domain (no blood vessels), therefore there is no coagulation for this wavelength actually. It is clearly visible that these results do not exactly correspond to those obtained on the basis of the Arrhenius formula.

The source of the differences is probably in the approach used in the current work. The Kubelka-Munk theory is strictly a one-dimensional theory: it uses forward and backward fluxes of light only. The skin is treated as a two turbid layer model and some physiological features, e.g. differences in the melanin concentration in the epidermis or the influence of the stratum corneum and fat layer have not been taken into account.

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