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# MODEL OF HUMAN SKIN OXYGENATION UNDER THERMAL ACTIONS

**S. A. Gubarev, A. A. Makhanek, and Z. P. Shul'man** UDC 536.24:675.014

*The problem of diffusion transfer of oxygen from an individual capillary into the surrounding skin with regard for the inhomogeneity of the blood composition in the vessel (plasma and erythrocytes) has been solved. The approximate dependence of the average concentration of oxygen in the tissue cylinder on its radius (density of active capillaries) and the circulation rate has been obtained. The model has been generalized to the macromolecule of a biotissue segment with a multitude of exchange vessels, and the dependence of the average concentration of oxygen in the biotissue bulk on the blood flow intensity (perfusion) has been obtained.*

**Introduction.** The processes of vital activity of biological tissues require timely delivery of oxygen $O_2$ and removal of carbon dioxide. The human organism cannot produce $O_2$ on its own. Without supply from the outside its amount suffices for only a few minutes. Therefore, man has to breath incessantly. The respiration process can be subdivided into three stages:

1. External respiration — in the lungs the air oxygen combines with blood. In so doing, the degree of saturation of hemoglobin in arterial blood reaches 97%.

2. Transport of oxygen — $O_2$ is carried by the blood flow to the tissues.

3. Internal (tissue) respiration — in exchange vessels oxygen from the blood is transferred to the surrounding cells.

The oxygen-transport properties of blood are determined by its predominant form elements — erythrocytes containing respiratory pigment hemoglobin (Hb). The erythrocyte is a nucleus-free biconcave cell of volume about 90 $\mu m^3$ and surface area 130–160 $\mu m^2$. Hemoglobin is a protein consisting of four subunits each of which contains an $Fe^{2+}$ atom capable of combining with oxygen $O_2$. The reaction is reversible and depends on the oxygen tension (partial pressure) $pO_2$. In the alveoli $pO_2$ = 100 Torr; here Hb combines with $O_2$ to form oxyhemoglobin ($HbO_2$). In exchange vessels the inverse process occurs — oxyhemoglobin dissociates into Hb and molecular $O_2$. An erythrocyte normally contains about 250 million Hb molecules.

In blood $O_2$ is present in two forms — bound (as part of oxyhemoglobin) and free (molecular oxygen dissolved in the blood plasma and erythrocyte cytoplasm). The $O_2$ solubility in biological fluids is rather low. The quantity of bound oxygen in arterial blood is almost 70 times higher than that of free oxygen (one liter of oxygenated blood contains about 200 ml of bound $O_2$ and only 3 ml of free $O_2$). Consequently, erythrocytes are the chief carriers of $O_2$.

A hemoglobin molecule can combine with up to four oxygen molecules to form compounds $Hb_4O_2$, $Hb_4O_4$, $Hb_4O_6$, and $Hb_4O_8$. The complete description of the $O_2$–Hb interaction is given by the Adair scheme [1]. It contains eight reaction rate constants, whose determination is problematic. Therefore, in model calculations this scheme is simplified — instead of one $Hb_4$ molecule one considers four independent heme groups of 4Hb. Then the $O_2$–Hb interaction is described as

$$\mathrm{Hb} + \mathrm{O_2} \underset{k'}{\overset{k}{\leftrightarrow}} \mathrm{HbO_2}\,, \tag{1}$$

where $k$, $k'$ are the rates of production and dissociation of oxyhemoglobin.

A. V. Luikov Heat and Mass Transfer Institute, National Academy of Sciences of Belarus, 15 P. Brovka Str., Minsk, 220072, Belarus. Translated from Inzhenerno-Fizicheskii Zhurnal, Vol. 80, No. 1, pp. 70–75, January–February, 2007. Original article submitted October 10, 2006.

 

The balance dependence between the quantities of bound and free $O_2$ in an erythrocyte is approximated by the Hill equation:

$$\frac{C_{HbO_2}}{C_{Hb}} = \frac{(C_{O_2}/C_{50})^{n_H}}{1 + (C_{O_2}/C_{50})^{n_H}}. \quad (2)$$

The walls of large arteries and veins are impenetrable to $O_2$ and other substances, and gas and water exchange with interstitially through them is absent. The transport of free or bound $O_2$ in these vessels is convective. In the microcirculation system, the walls become thinner and penetrable to water and substances dissolved in the plasma and tissue fluid, $O_2$ begins to flow from the vessels into the biotissue. In general, in exchange vessels $O_2$ is carried to the environment by diffusion and convection (jointly with water filtration). The relative contribution of these mechanisms is organospecific. In most biotissues, the distance between cells and nutrient vessels does not exceed 100 μm, as a rule. At such small distances diffusion effectively competes with convection.

Oxygen molecules are smaller than the size of capillary pores; they are electrically neutral and highly soluble in lipids. This enables them to easily penetrate through the membranes of endothelial cells, as well as through intercellular spaces. Diffusion of oxygen proceeds over the entire surface of the walls of exchange vessels. The limited density of interendothelial pores has a weak effect on the $O_2$ flow [2].

Water exchange of blood and biotissue is of paramount importance for living cells and is an essential part of homeostatis. Together with water low-molecular dissolved gases and ions (including $O_2$) are carried through three types of channels: interendothelial spaces, enlarged pores, and aquaporins [3, 4].

Central to the modern quantitative theories of water exchange between vessels and the surrounding biotissue is the Starling hypothesis [5]: the speed of ultrafiltration of water through vascular walls is determined by the difference between the hydrodynamic blood pressure and the osmotic plasma pressure. The blood plasma and the intercellular fluid are almost isotonic and differ by the value of the oncotic pressure, which is regulated mainly by the content of proteins (and to a lesser extent by the content of salts and other substances) and constitutes only 0.5% of the osmotic pressure. The hydrodynamic and oncotic pressures in a vessel have opposite directions. In the arterial part, the hydrodynamic pressure is higher; therefore, water with low-molecular substances dissolved in it is partly forced through the wall into the intercellular medium. In the venous part of the vessel, the inverse process occurs — the fluid is drawn into a capillary under the action of the oncotic pressure excess. About 15% of the fluid is removed through the lymphatic system.

The quantitative analysis of the transcapillary exchange was developed by Kedem and Katchalsky in [6]. They employed the nonequilibrium thermodynamics methods for the processes of transfer through biomembranes and expressed the water and dissolved substance flows in terms of three phenomenological coefficients: hydraulic conductivity, permeability, and reflection.

Beginning with the pioneer works of Krogh on oxygenation of biotissues, only capillary walls were considered to be permeable to $O_2$. The appearance of local methods of $O_2$ tension measurement in microvessels and biotissues made it possible to ascertain the participation of other vessels in the gas exchange [7, 8]. In the opinion of the authors of [9], an important role is played by minute arterioles and veinules of diameter 10–30 μm. In [7], comparison of the data of numerous *in vivo* measurements of longitudinal gradients of $pO_2$ in hamsters, rats, cats, dogs, and rabbits was made. A gradual decrease in $pO_2$ with thinning arterioles, beginning with a diameter of 100 μm, was observed. The transfer of oxygen into the interstitial medium is enhanced as capillaries are approached.

Oxygenation of biotissues is a hard-to-control process depending on many biophysical and physicobiological factors: the degree of hemoglobin saturation in the arterial blood, the shape of the oxyhemoglobin dissociation curve, the volume rate of blood flow in exchange vessels, the blood and biotissue temperature, the rate of oxygen consumption by the tissue cells, etc.

An important applied and theoretical problem is measurement of the $O_2$ concentration in the biotissue under various kinds of physiotherapeutic actions. This is of paramount importance in the analysis of oxygen-dependent methods, e.g., photodynamic therapy of malignant tumors — its effect directly depends on the $O_2$ content in a tumor. Biotissue heating in the course of irradiation by a therapeutic laser intensifies the blood flow and, therefore, the delivery

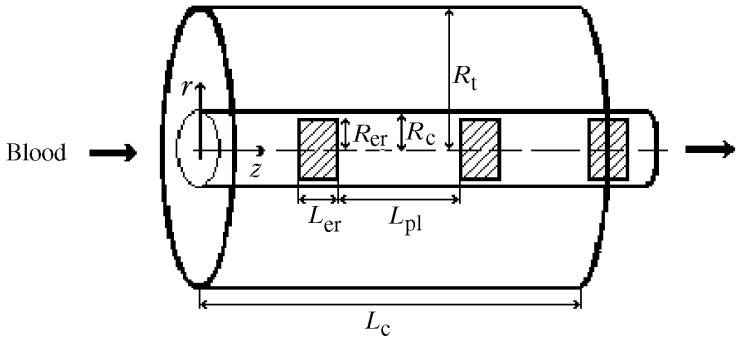


Fig. 1. Tissue cylinder model.

of oxygen. The aim of the present work is to obtain the dependence of the average concentration of oxygen in the biotissue on the blood flow intensity in it under thermal actions as applied to skin tissues.

**Formulation of the Problem.** The physico-mathematical model of biotissue oxygenation is based on the $O_2$ transport from an individual exchange vessel. The problem has been solved on the following assumptions:

1) a cylindrical coordinate system is used (Fig. 1);

2) the blood in the capillaries is a two-phase medium; all erythrocytes in the vessel are cylindrical and move along it at equal velocities on a thin wall lubricating layer of plasma;

3) the velocity profile of plasma in the erythrocyte–wall gap is linear, and between particles it is parabolic;

4) oxygen transport through the capillary wall and in the biotissue is diffusive;

5) all biophysical properties of $O_2$, tissue, and blood are constant and homogeneous.

The proposed model is a modification of the classical Krogh's tissue cylinder with allowance for the multiphase character of blood in microvessels. The symmetry of the $O_2$ and $HbO_2$ concentration fields about the angular coordinate is fully justified. In the capillaries, only the motion of erythrocytes was considered, and the contributions of other particles were neglected. Effects complicating the analysis are possible: they arise when leukocytes are forced through a microvessel because of temporary channel choking. These effects are the subject of the further development of the model. In the first approximation erythrocytes in the capillaries can be taken to be cylindrical, while their shape is actually parachute-like. Taking into account the smallness of the erythrocyte–wall gap (tenths of a micrometer), the linearity of the plasma velocity profile is fully justified. Between particles the plasma flow is swirled at the back surface of erythrocytes [10]. However, in [11] a slight influence of plasma convection between erythrocytes on the $O_2$ transfer into the biotissue was noted (since the main mass transfer occurs through the thin wall layer between the particle membranes and the capillary wall). Therefore, the replacement of the real velocity profile by a parabolic one is quite feasible with the preservation of the total consumption of plasma. For the skin and muscular tissue, in analyzing the $O_2$ transfer in the first approximation the water filtration through the capillary walls and in the biotissue can be ignored, which has been confirmed in many papers [10, 12–14]. In a number of tissues (kidneys, liver, intestine, brain, etc.), by virtue of their functional specificity, the water exchange of vessels with the environment is more intensive, and account in them of only the diffusion mechanism of mass transfer is insufficient. The model presupposes a uniform distribution of exchange vessels in the biotissue. The real structure can be heterogeneous, especially in rapidly growing malignant tumors. This leads to a nonuniformity of the blood flow and the $O_2$ concentration field. Further development of the theory of biotissues oxygenation requires knowledge of the detailed structure of the microcirculation system and complex account of individual vessels in the $O_2$ mass transfer, which is beyond the scope of the present paper.

The calculation region consisted of three parts — erythrocytes, plasma, and biotissue. Oxygen in erythrocytes can be of two kinds: free (dissolved in the cytoplasm) and bound (as part of oxyhemoglobin $HbO_2$). As a particle is moving through the capillary, $HbO_2$ dissociates and $O_2$ is released. In the blood plasma, oxygen is carried by diffusion and convection together with the medium flow. In the biotissue, $O_2$ is consumed in metabolic reactions. In erythrocytes, it is necessary to consider the $O_2$ and $HbO_2$ concentration fields [15], and in the plasma and biotissue — only the $O_2$ concentration field:

$$\partial C_{O_2}/\partial t + V\nabla C_{O_2} = D_{O_2}\nabla^2 C_{O_2} + s_{O_2}, \tag{3}$$

$$\partial C_{HbO_2}/\partial t = D_{HbO_2}\nabla^2 C_{HbO_2} + k_{HbO_2}\left\{(C_{Hb} - C_{HbO_2})\,(C_{O_2}/C_{50})^{n_H} - C_{HbO_2}\right\}, \tag{4}$$

Equation (3) is written in the general form for erythrocytes, plasma, and biotissue. The velocity of motion relative to particles $V$ takes values 0, $(-V_{er} + V_{pl})$, $-V_{er}$; the diffusion coefficient $D_{O_2}$: $D_{O_2,er}$, $D_{O_2,pl}$, $D_{O_2,t}$; the oxygen source $s_{O_2}$: $-k_{HbO_2}\,((C_{Hb} - C_{HbO_2})\,(C_{O_2}/C_{50})^{n_H} - C_{HbO_2})$, 0, and $s_{O_2,t}$, respectively. The velocity profile of the plasma between erythrocytes and the vessel wall is assumed to be linear because of the smallness of the gap (~0.1 μm), and between erythrocytes — parabolic.

The $HbO_2$ flow at the particle boundary is zero. On the erythrocyte and vessel walls we give the condition of conjugation of the concentrations and diffusion flows of oxygen:

$$C_{O_2}\Big|_{r=r^*-0} = C_{O_2}\Big|_{r=r^*+0}, \quad C_{O_2}\Big|_{z=z^*-0} = C_{O_2}\Big|_{z=z^*+0},$$

$$\left(D_{O_2}\frac{\partial C_{O_2}}{\partial r}\right)\Big|_{r=r^*-0} = \left(D_{O_2}\frac{\partial C_{O_2}}{\partial r}\right)\Big|_{r=r^*+0}, \quad \left(D_{O_2}\frac{\partial C_{O_2}}{\partial r}\right)\Big|_{z=z^*-0} = \left(D_{O_2}\frac{\partial C_{O_2}}{\partial r}\right)\Big|_{z=z^*+0}, \tag{5}$$

where $z^*$ and $r^*$ the coordinates of the erythrocyte boundaries or the vessel walls. At the tissue cylinder boundaries:

$$C_{O_2}\Big|_{\substack{z=0\\0<r<R_c}} = C_{in}, \quad \frac{\partial C_{O_2}}{\partial z}\Big|_{\substack{z=0\\R_c\le r\le R_t}} = 0, \quad \frac{\partial C_{O_2}}{\partial z}\Big|_{z=L_c} = 0; \tag{6}$$

$$\frac{\partial C_{O_2}}{\partial r}\Big|_{r=0} = 0, \quad \frac{\partial C_{O_2}}{\partial r}\Big|_{r=R_t} = 0. \tag{7}$$

According to (6), arterial blood enters the vessel with a constant concentration of oxygen $C_{in}$. For the biotissue at the arterial boundary, as well as for the whole of the vein boundary (inside and outside the capillary) the conditions of zero flows are given. On the vessel axis the flow is zero because of symmetry (7). At the outer boundary of the tissue cylinder minimum $C_{O_2}$ is given.

At the initial instant of time, the $O_2$ concentration in the vessel is assumed to be homogeneous and linearly decreasing along the vessel axis from the known concentration $C_{in}$ to that presumably equal to $C_{out}$. We calculate $S_{HbO_2}$ in erythrocytes by the Hill formula (2). We assume the $O_2$ concentration in the biotissue to be homogeneous and known. At the tissue cylinder boundaries, we give conditions (6), (7). At the erythrocyte–plasma and plasma–biotissue boundaries, we equalize the oxygen flows (5).

We calculate the value of $L_{pl}$ by the given hematocrit Ht (volume concentration of erythrocytes) in the capillary

$$L_{pl} = L_{er}\left(R_{er}^2 - \mathrm{Ht}_c R_c^2\right)\Big/\left(\mathrm{Ht}_c R_c^2\right). \tag{8}$$

The tissue cylinder radius is determined from the known density of vessels

$$R_t = (\pi N_c)^{-1/2}. \tag{9}$$

Problem (3)–(7) was solved by the finite difference method with splitting for spatial variables. For the variable $r$, the mesh width was given to be different in the regions of erythrocytes, the gap, and the biotissue. For the

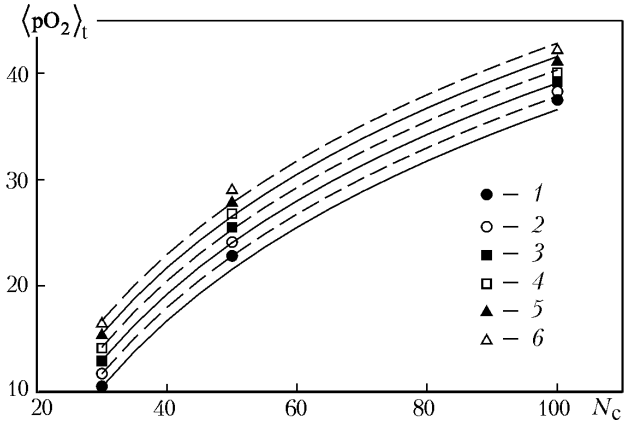


Fig. 2. Oxygenation of the tissue cylinder: 1) $V_{er}$ = 150; 2) 200; 3) 250; 4) 300; 5) 350; 6) 400 μm/sec. $N_c$, mm$^{-2}$; $\langle pO_2 \rangle_t$, Torr.

variable $z$, the mesh was chosen to be uniform. The derivatives of $r$ and $z$ were approximated implicitly with regard for the conservatism. The nonlinearity of the source terms in (3) and (4) required introduction of the iteration process at each time step to refine the concentrations of $O_2$ and $HbO_2$. Going out of it occurred upon reaching the given accuracy. The difference scheme converged conditionally at a time step of no more that $10^{-5}$ sec.

In the numerical experiment, we varied the particle velocity and the value of $N_c$. The results of the calculations are presented in Fig. 2. They are expressed more graphically in terms of oxygen tension $pO_2$. Passing to the concentration is realized by the formula $C_{O_2} = Bu \cdot pO_2$, where Bu = $1.56 \cdot 10^{-2}$ mole·Torr$^{-1}$ m$^{-3}$ is the solubility coefficient of $O_2$. The values of the erythrocyte velocity and the capillary length were chosen so that the particle stayed in the vessel for about 2 sec. The densities of active capillaries of 30, 50, and 100 mm$^{-2}$ correspond to the radii of tissue cylinders of 103, 80, and 56 μm. The increase in $pO_2$ in the biotissue due to the acceleration of the blood flow through the vessel by a factor of 2.7 is less than 60%, and when the number of open capillaries is increased three times, it is 250–350%. Therefore, the change in the oxygenation level with increasing perfusion is mainly due to the involvement of reserve capillaries and, to a much lesser extent, is a result of the increase in the blood velocity in the capillaries. Acceleration of the blood flow causes a more homogeneous oxygenation of the tissue, decreasing the $O_2$ pressure drops at the arterial and venous ends of the vessel. In the biotissue, in the immediate vicinity of the capillary wall $pO_2$ oscillations due to the alternation of erythrocytes and plasma were observed. They attenuate in going deeper into the tissue cylinder. We managed to express the dependences of the oxygen tension averaged over the biotissue volume $\langle pO_2 \rangle_t$ on the density of active capillaries at different velocities of erythrocytes $V_{er}$ by a single dependence (curves in Fig. 2) at a maximum difference of less than 3 Torr:

$$\langle pO_2 \rangle_t = a_1 + a_2 V_{er} + a_3 \ln (N_c) , \tag{10}$$

where $a_1$ = −367.3337 Torr, $a_2$ = 25,000 Torr·sec·m$^{-1}$, and $a_3$ = 21.7244 Torr are the approximation coefficients.

The tissue cylinder model makes it possible in the first approximation to go from the consideration of an individual vessel to integrated indices for the biotissue volume. A simple dependence of the blood flow intensity on the tissue cylinder characteristics (its radius and the mean blood velocity) was proposed in [16]. However, it does not take into account the contribution of arteriovenous anastomoses (AVA) to the development of perfusion, although for many biotissues (especially for the skin) it is dominant. To take it into account, let us introduce the coefficient $K_{AVA}$ depending on the type of tissue and the current blood flow intensity (or accumulated thermal dose):

$$W = \frac{\pi R_c^2 V_{er}}{L_c} K_{ABA} N_c . \tag{11}$$

Under thermal actions the perfusion changes because of the vasodilation ($V_{er}$ increases) and involvement of reserve capillaries ($N_c$ increases) and thermoregulation mechanisms ($K_{AVA}$ changes). We have estimated the contribution

of these factors. Using the model of temperature-time analogy to describe the blood flow intensity under thermal actions [17], let us represent the perfusion dependence of the average tension $pO_2$ in the tissue in view of (11) as

$$\langle pO_2 \rangle_t = b_1 + b_2 \ln (W/K_{ABA}) , \tag{12}$$

where $b_1$ = 166.44 Torr, $b_2$ = 21.7244 Torr are the approximation coefficients.

**Conclusions.** A physico-mathematical model of oxygen mass transfer from an individual capillary into the surrounding biotissue with allowance for the inhomogeneity of the blood composition (erythrocytes and plasma) has been constructed. The dependence of the mean volume concentration of $O_2$ in the tissue on the particle velocity and the tissue cylinder radius (density of active capillaries) has been obtained. The numerical experiment has shown that under a thermal action on the tissue an increase in the density of active capillaries produces a much stronger effect on $\langle pO_2 \rangle_t$ than the blood flow acceleration does (a threefold amplification of the action of one of these factors leads to an increase in the $O_2$ tension by 250–350% and 60%, respectively).

The model of oxygen mass transfer from an individual capillary has been generalized to the case of a microportion of biotissue with allowance for the contribution of arteriovenous shunts. The perfusion dependence of the average concentration of $O_2$ on a portion of the skin has been obtained.

## NOTATION

Bu = $1.56 \cdot 10^{-3}$, solubility coefficient of oxygen, mole·Torr$^{-1}$·m$^{-3}$; $C_{50}$ = 0.0412, equilibrium concentration of oxygen at a 50% saturation of hemoglobin, mole·m$^{-3}$; $C_{Hb}$ = 20.3, heme concentration in blood, mole·m$^{-3}$; $C_{HbO_2}$, concentration of oxyhemoglobin, mole·m$^{-3}$; $C_{O_2}$, oxygen concentration, mole·m$^{-3}$; $D$, diffusion coefficient, m$^2$·sec$^{-1}$; Ht, packed cell volume (volume concentration of erythrocytes in blood); $I$, radiation intensity, W·m$^{-2}$; $K_{AVA}$, coefficient for taking into account the contribution of arteriovenous anamostoses to the change in perfusion under thermal actions; $k_{HbO_2}$ = 44, dissociation rate of oxyhemoglobin, sec$^{-1}$; $L_{pl}$, interval between two neighboring erythrocytes, m; $N_c$ = 30–100, density of active capillaries in the skin, mm$^{-2}$; $n_H$ = 2.65, exponent in the Hill equation; $pO_2$, oxygen tension (partial pressure), Torr; $R$, radius, m·sec$^{-1}$; $V$, velocity with respect to erythrocytes, m·sec$^{-1}$; $W$, tissue blood flow intensity (perfusion), sec$^{-1}$; $r$, radial variable, m; $s_{O_2}$, oxygen source-sink, mole·m$^{-3}$·sec$^{-1}$; $t$, time, sec; $z$, axial variable, m. Subscripts: in, inlet; out, outlet; c, capillary; pl, plasma; t, biotissue; er, erythrocyte.

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