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Oxygen Transport in Human Alveolar Sacs.

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The calculations of resistance of oxygen access to the surface of alveolar sacs in human respiratory system were performed. The airways morphology was described by Weibel's model. The changes of alveolar sac diameter during breathing cycle and the unsteady flow conditions were taken into account. The resistance of oxygen transport for healthy case was compared with several pathological cases, namely insufficiency of breathing, emphysema, congestion-thrombotic diseases and interstitial diseases. Results of calculations show, that in each pathological case, the resistance of oxygen transport in human alveolar sacs is higher than for healthy one.

1. Introduction

The Constructal law states that if a system has a freedom to morphs it develops in time the flow architecture that maximizes the heat and flow transport (Bejan, 2000). Recently Przekop (2009) has shown that the best oxygen access to the tissues, where it reaches the blood and best carbon dioxide removal is performed by a flow structure composed of 23 levels of bifurcation. The calculations were performed for healthy case. The aim of this study is to investigate how the pathological cases, namely insufficiency of breathing, emphysema, congestion-thrombotic diseases and interstitial diseases influence the oxygen transport in human alveolar sacs.

2. Lungs model

Taking Weibel's (1963) lung model with 23 generations we can distinguish the following terminology (Figure 1). Generation zero is called the trachea. The first generation is called the main bronchus, the second is called the lobar bronchus, the third the segmental bronchus and the fourth the subsegmental bronchus. Generations 5 till 16 are called the lobuli and consist of small bronchioli (generations 5-9), and terminal bronchioli (14-16). Generations 17 till 24 are called the acini and consist of respiratory bronchioli (17-19), alveolar ducts (20-22) and alveolar sacs (23). The radius and branching angles of the tubes as they occur naturally in the lungs can be clarified if a few conditions are satisfied which may be applicable to branched tube flow. The minimum flow resistance is achieved if the ratio between consecutive duct diameters is:

$$\frac{D_n}{D_{n-1}} = 2^{-\frac{1}{3}} \tag{1}$$

And the respective duct lengths

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$$\frac{L_n}{L_{n-1}} = 2^{-\frac{1}{3}} \tag{2}$$

Typical value of trachea diameter and length are D_0=1.5 cm and L_0=15 cm, respectively. The optimal branching angle =37.5 degrees, what is close to the 34 degrees found in measurement of human lungs (Colebatch and Ng, 1992).

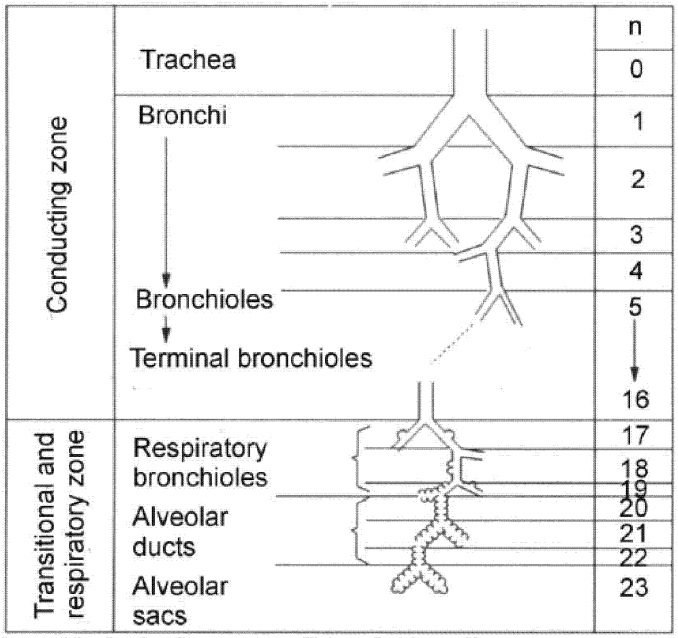

Fig. 1 - Model of the respiratory tree.

3. Alveolar sac model

In this study the alveolar sac is assumed to be sphere connected to the last generation of bronchial tree. In the beginning of inspiration the alveolar sac has got the diameter equal to the diameter of the last generation tube (d_0). Assuming breathing frequency and tidal the volumetric temporary air flow can be written:

$$Q(t) = A\sin(Bt)\,. \tag{3}$$

The temporary tidal volume is:

$$V(t) = \frac{A}{B}\left[1 - \cos(Bt)\right] \tag{4}$$

and the alveolar sac temporary diameter:

$$d(t) = \sqrt[3]{d_o^{\,3} + 6\pi \frac{A}{2^{23} B}\left[1 - \cos(Bt)\right]} \tag{5}$$

The model constants for different cases are summarized in Tab. 1.

Table 1: Model constants for different cases

	Tidal volume [dm^3]	Inspiration time [s]	Expiration time [s]	A [dm^3/s]	B[1/s]
Healthy	2.4	2.0	2.0	1.8849	1.5707
Insufficiency of breathing	1.7	2.0	2.0	1.3351	1.5707
Emphysema	0.45	2.0	2.8	Inspiration 0.3534 Expiration 0.2524	Inspiration 1.5707 Expiration 1.1221
Interstitial diseases	2.5	1.0	1.0	3.9269	3.1415
Congestion-thrombotic diseases	2.0	1.3	1.3	2.4165	2.4165

The temporary volumetric flow rate, tidal volume and alveolar sac diameter are shown in Figures 2-4.

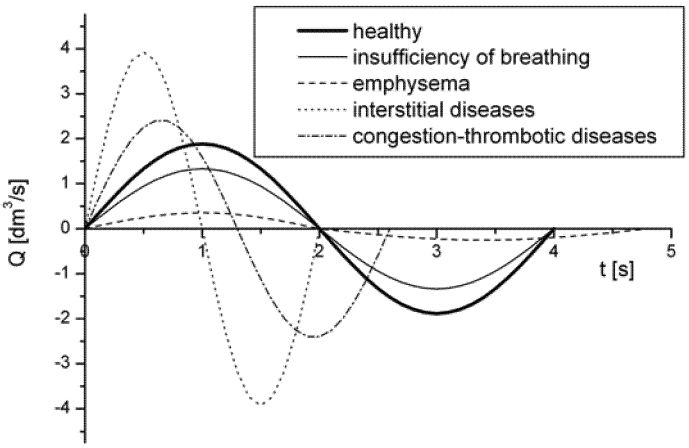

Figure 2: Temporary volumetric flow rate for different cases.

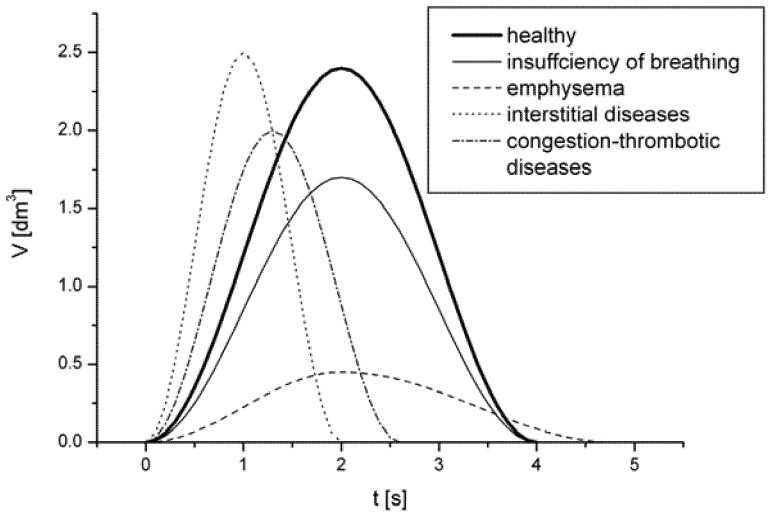

Figure 3: Temporary tidal volume for different cases.

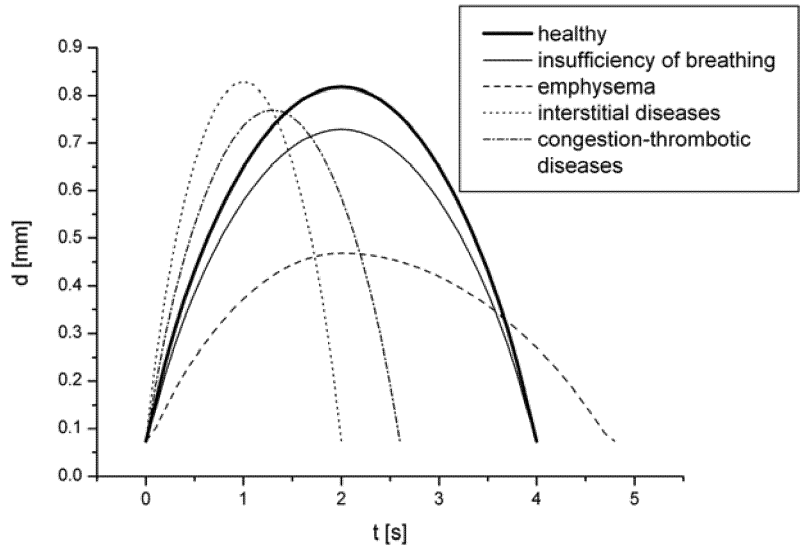

Figure 4: Temporary alveolar sac diameter for different cases.

4. Oxygen transport in alveolar sacs

The temporary oxygen current to the surface of alveolar sacs can be expressed as:

$$m_{ox}(t) = 2^{23} K_g(t)\pi d^2(t)\Delta\rho_{ox}, \tag{6}$$

where K_g is mass transfer coefficient and $\Delta\rho_{ox}$ is the difference between the oxygen concentration in the entrance of the alveolar sac and the alveolar surface. The average oxygen flux during breathing cycle is than

$$\overline{m}_{ox} = \frac{2^{23}}{t_b}\int_0^{t_b} K_g(t)\pi d^2(t)\Delta\rho_{ox}\,dt \tag{7}$$

where t_b is time of breathing cycle and K_g is mass transfer coefficient. Taking into account that

$$\Delta\rho_{ox} = \frac{\phi_{ox}\rho\Delta\mu_{ox}}{(R_g)_{ox}T}, \tag{8}$$

where ϕ_{ox} is relative concentration of oxygen in the alveoli, ρ the air density, $\Delta\mu_{ox}$ chemical potential, $(R_g)_{ox}$ is the oxygen constant and T absolute temperature, we can therefore calculate the resistance of oxygen transport in alveolar sac:

$$R_{ox} = \frac{(R_g)_{ox}Tt_b}{2^{23}\int_0^{t_b} K_g(t)\pi d^2(t)\Delta\rho_{ox}\,dt}. \tag{9}$$

The value of mass transfer coefficient can be calculated from the correlation given by Sherwood *et. al* (1975) for mass transfer in bubbles as a function of Reynolds, *Re*, and Schmidt, *Sc*, numbers:

$$Sh = 0.664\,\mathrm{Re}^{1/2}\,Sc^{1/3}. \tag{10}$$

Sherwood number is defined as:

$$Sh = \frac{K_g d}{D_{ox}}, \tag{11}$$

where D_{ox} is oxygen diffusivity. The characteristic velocity and dimension for computing Reynolds number are the values for the last tube of bronchial tree.

5. Results and discussion

The results of calculation of oxygen transport resistance in alveolar sacs, for healthy and different pathological cases, are presented in Figure 5. As one can see, each pathological case causes a significant increase in oxygen transport resistance. This is particularly

interesting when we take into account the different nature of various pathological cases. In most cases, the tidal volume decreases, but congestion-thrombotic diseases are an exception. Some pathological cases are caused by decrease of alveolar sacs elasticity (emphysema), while the source of others is elasticity increase, as for insufficiency of breathing or interstitial diseases. The increase in oxygen transport resistance is observed for both cases of shortening and extension of breathing cycle time. The results indicate that respiratory system is not only the optimal geometry, but also the tidal volume and frequency of breathing maximize the oxygen current from the air into the blood.

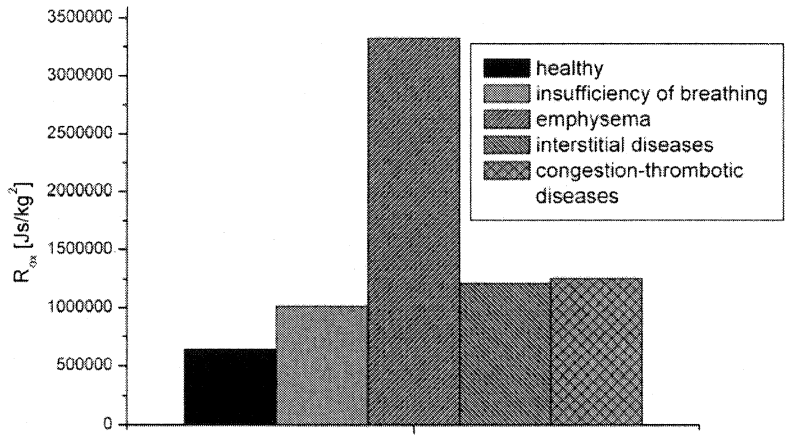

Figure 5: Oxygen transport resistance in alveolar sacs for different cases.

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