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Numerical study of the effect of left coronary artery stenosis on vascular tissue oxygenation

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ABSTRACT: Cellular metabolism is strongly dependent on the sufficient and continuous supply of oxygen. Cellular oxygenation is performed by blood flow. Blood flow and oxygen delivery to cells can be influenced under several conditions such as arterial stenosis. In the present study, the oxygen delivery to the arterial wall of a precise model of the main left coronary artery and its two main branches for normal artery and stenosis degrees of 75% and 84% is numerically investigated. For all simulations, it is assumed that the flow is steady, blood is Newtonian, and the arterial wall is rigid. Transported oxygen by hemoglobin as well as oxygen consumption in the vessel wall are considered for determining the oxygen content in the vascular tissue. The results of oxygen concentration in the lumen and vascular tissue are validated with a benchmark study. The results indicate that centrifugal forces and secondary flow are formed due to curvature and results in a significant reduction in the mass transfer of oxygen to the myocardial wall relative to the epicardial one. Arterial stenosis results in locations of low oxygen concentration with 3-4 mmHg less than the normal artery, that increases the likelihood of hypoxia in these areas. Finally, the results show that 12.8% reduction in P_{50} does not have a significant effect on the oxygen concentration in the lumen and vascular tissue.

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1- Introduction

Many researchers have pointed to the association between atherosclerosis, lack of blood supply, and insufficient oxygenation to the heart tissue [1-3].

Moore and Ethier [4] investigated a straight and symmetrical artery with a maximum stenosis degree of 88.9%. The results of this study showed that oxygen transported by hemoglobin had a significant effect on the rate of oxygen transfer. Banerjee et al. [3] studied oxygen delivery in the coronary artery with stenosis before and after angioplasty. Their results showed that the oxygen delivery to the vessel wall increases in the area where blood flow accelerates. Liu et al. [5] investigated the effect of severe aortic curvature on oxygen transfer. Their results show that aortic curvature causes strong rotational flow, which results in increased oxygen flow to the arterial wall. Yan et al. [6] investigated the effect of amputation on blood flow and oxygenation in the femoral artery. Zhang et al. [7] investigated the effects of cardiac output on the distribution of oxygen concentration in the aorta for patients undergoing VA-ECMO support.

In this study, the steady-state flow in a model constructed based on the laboratory information of the left coronary artery with its main branches in non-stenosis and several degrees of stenosis is investigated. The effect of oxygen transport by hemoglobin, the thickness of the vessel wall, and oxygen consumption in the wall have also been considered. Therefore, in this study, the simultaneous effect of curvature, bifurcation, and stenosis on a 3D model of the left coronary artery is investigated.

2- Methods

The geometry considered in this study is a 3D model of the left main coronary artery and its two main branches.

The length and radius of the vessels are based on laboratory measurements [8, 9] and only a direct portion is added to the end of the artery to reduce the effects of boundary conditions uncertainty [8, 10]. The thickness of the vessel wall was varied from 0.3 mm in the non-stenosis area to 0.47 mm in the stenosis area, indicating the distance between the inner lumen and the Vasa-vasorum layer [3]. Three geometrical models are constructed. One model of the coronary artery is without stenosis and two models are with stenosis degrees of 75% and 84% in left anterior descending artery (LAD) and circumflex branch of the left coronary (LCX).

We assumed that the flow is steady, blood is Newtonian, and the arterial wall is rigid. Under these assumptions, three-dimensional mass conservation and Navier-Stokes equations were solved to describe blood flow in the artery. Oxygen transfer within the blood is expressed by the following equation [4]:

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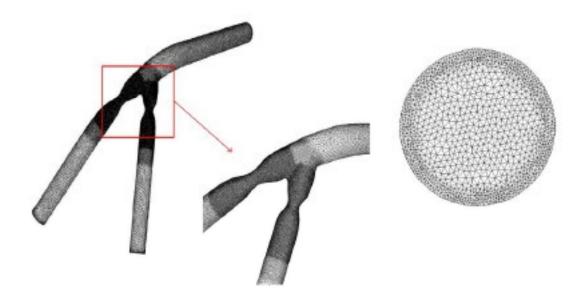


Fig. 1. Schematics of constructed model and generated mesh

$$\left(1 + \frac{[Hb]}{\alpha} \frac{dS}{dPO_2}\right) \vec{u} \cdot \nabla PO_2 =
\nabla \cdot \left[D_b \left(1 + \frac{[Hb]}{\alpha} \frac{D_c}{D_b} \frac{dS}{dPO_2}\right) \nabla PO_2\right]$$
(1)

where α is the solubility of oxygen in plasma, S is the oxyhemoglobin saturation function, PO_2 is the plasma oxygen tension, [Hb] is the total oxygen-carrying capacity of hemoglobin in the blood, and D_b and D_c are the diffusivities of free oxygen and oxyhemoglobin in blood, respectively. The saturation function, S, defined as the ratio of oxyhemoglobin to total hemoglobin, is approximated by the Hill equation [11]:

$$S = \frac{PO_2^n}{PO_2^n + P_{50}^n} \tag{2}$$

where n=2.7 and $P_{50}=26.6$ mmHg. A typical convective velocity within the artery wall is 10^{-6} cm/s, while oxygen diffusion velocity is of order 10^{-5} to 10^{-4} cm/s [12]. Therefore, Convective transport of oxygen within the artery wall was neglected. Thus, the equation governing wall-side oxygen transport was [4]:

$$\alpha_T \frac{dPO_2}{dt} = \alpha_T \nabla \cdot (D_T \nabla PO_2) + \dot{q}$$

where \vec{q} represents a constant volumetric consumption rate of oxygen by cells within the arterial wall. D_T and α_T is the oxygen diffusivity and solubility of oxygen in tissue, respectively.

(3)

For all three cases, a uniform inlet velocity profile is used with a mean Reynolds number of 270 [11]. In addition, at the inlet and Vasa-vasorum layer, the boundary condition of the oxygen partial pressure was specified as 95 mmHg and 45 mmHg, respectively [3]. Fig. 1 shows an overview of the constructed model and generated mesh for the stenosis degrees 75%.

3- Results and Discussion

Fig. 2 shows the distribution of oxygen partial pressure in the radial direction at two different sections in LAD and LCX branches. In the bloodstream region, the partial pressure of oxygen is not affected by coronary artery bifurcation and

the arterial curvature. Due to oxygen consumption in the vessel wall, the partial pressure of oxygen in the vessel wall initially decreases, but with approaching toward the Vasavasorum, the oxygen partial pressure increases to reach 45 mmHg at the outermost part of the artery wall.

As blood flows through the artery curvature, the maximum axial velocity shifts toward the outer wall of the curvature, which induces secondary flows. This causes the partial pressure of oxygen through the inner wall of the curvature to be lower than the outer wall of the curvature. After passing

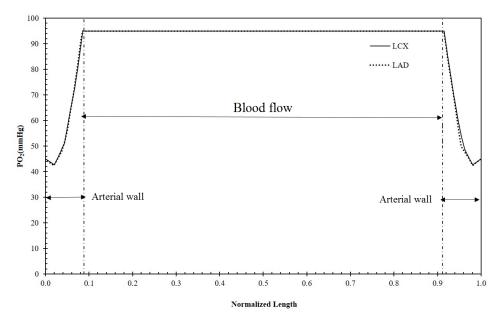


Fig. 2. Distribution of oxygen partial pressure in the radial direction

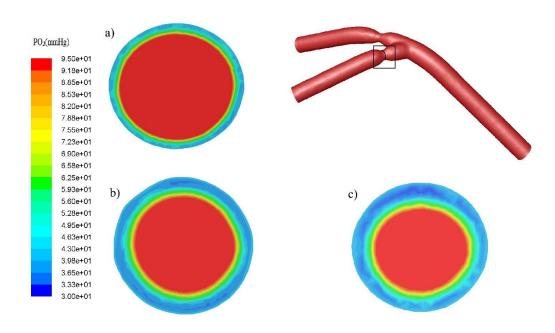


Fig. 3. Oxygen partial pressure distribution in left circumflex branch for a) Without stenosis, b) 75% stenosis, and c) 84% stenosis

the curved section, the oxygen partial pressure distribution in the arterial wall gradually approaches a uniform distribution.

Fig. 3 shows the distribution of partial pressure of oxygen in the left circumflex branch in three different modes; normal (without stenosis), 75% stenosis, and 84% stenosis. In the region of the stenosis, due to the increased thickness of the vessel wall, oxygen consumption increases. On the other hand, due to stenosis, the flow velocity is increased and the

thickness of the boundary layer is reduced, which causes more oxygen delivery to the vessel wall. Therefore, the interaction between these two factors will determine the amount of oxygen partial pressure in this region. The stenosis has little effect on the distribution of partial pressure of oxygen in the bloodstream, so in all three cases in Fig. 3, the amount of partial pressure of oxygen within the vessel is approximately constant at 95 mmHg.

4- Conclusions

In the present study, oxygen transport in the blood and the walls of the coronary artery with its two main branches are investigated. The results of this study show that the curvature of the artery causes a secondary flow, which has a significant effect on the distribution of oxygen concentration through the vessel wall. The formation of this secondary flow causes the partial pressure of oxygen on the inner wall to be lower than the partial pressure of oxygen on the outer wall of the arterial curvature. In addition, with increasing curvature, the difference of oxygen concentration on the inner and outer walls of the arterial curvature is increased. In addition, Different degrees of stenosis have little effect on the pattern of oxygen distribution in the vessel wall. At stenosis areas, the thickness of the vessel wall is increased. This, in turn, increases areas with low oxygen partial pressure. However, almost the minimum amount of oxygen partial pressure does not change. In the present study, 13% variation in P_{50} is also investigated; the results indicate that the changes in this parameter have little effect on the oxygen distribution.

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