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OXYGEN TRANSPORT TO THE AVASCULAR WALL OF A CORONARY ARTERY STENOSIS FOR VARYING BLOOD VISCOSITY

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ABSTRACT

Numerical study of oxygen (O_2) transport to the avascular wall of a stenosed artery is carried out for varing red blood cell concentration, i.e., hematocrit (Hct). Comprehensive physiological factors, such as, oxygen supply from vasa vasorum, nonlinear O_2 binding capacity of the hemoglobin, and O_2 consumption by the wall region, are also taken into account. As Hct increases, the $pO_{2,\ wall}$ and O_2 flux to the avascular wall are decreased. In particular, the O_2 flux for the Hct 65 % at the point of flow reattachment is significantly decreased from the value obtained for the Hct 25%. Moreover, for an increased area occlusion and arterial wall thickness, a hypoxic condition might occur in the avascular wall region as Hct increases.

INTRODUCTION

Blood is a non-Newtonian fluid that its viscosity changes with the shear rate. The blood viscosity is primarily dependent on Hct [3]. Hct in a given individual may change significantly as a part of physiological and pathological processes [2]. Previous results have been found that there is a decrease in O_2 transport to the wall in the decelerated flow regions in a stenosed artery and the region might be prone to hypoxic injury [5, 6]. However, there is little research regarding optimal Hct for maximum O_2 transport to the avascualr wall. Thus, the purpose of this study is to evaluate the effect of varying Hct on the O_2 transport through the avascular wall region of the stenosed coronary artery.

METHOD

The O_2 transport equation in the lumen is [1]:

$$\left(1+\phi'\right)\frac{Dc}{Dt}=\nabla\cdot\left(D_b\nabla c\right) \tag{1}$$

and that in the avascular wall region is:

$$\frac{\partial c}{\partial t}=\nabla\cdot\left(D_w\nabla c\right)-\dot{q} \tag{2}$$

where c is the O_2 concentration in ml_o/ml_{blood}. It is related to pO_2 as $c = \alpha\cdot pO_2$, where the value of the solubility in blood, α, is 3×10^{-5} $ml_o/ml/mmHg$. The O_2 diffusivities in blood, D_b, and in the wall, D_w, are 1.37×10^{-5} and 0.9×10^{-5} cm^2/s, respectively [5]. The quantity ϕ' is related to the slope of the nonlinear hemoglobin saturation curve by the relation:

$$\phi'=\frac{\partial\chi}{\partial c}=\left(\frac{H}{\alpha}\right)\left(\frac{\partial S}{\partial pO_2}\right) \tag{3}$$

where, [H] = 0.2 ml_o/ml_{blood} is total O_2 carrying capacity of hemoglobin in blood and S is the saturation function which introduces the O_2 carried by hemoglobin into the equation (1) [1]. Value of O_2 consumption rate in the avascular wall, $\dot{q}$, is 1.3×10^{-5} $ml_o/ml_{tissue}/sec$ as measured by Crawford et. al. [4] in normal dog femoral arteries, which are roughly similar in size to human coronary vessels.

For velocity field calculations, constant basal flow rate of 50 ml/min with fully developed parabolic profile for velocity, is applied at inlet. No slip boundary condition is applied at the lumen-wall interface. For concentration field calculations, uniform concentration of O_2, corresponding to normal blood pO_2 of 95 mmHg is applied at inlet and at vasa vasorum O_2 concentration corresponding to $pO_{2,\ v}$ of 45 mmHg is applied. Zero flux boundary condition is applied at the axis. In the avascular wall (thickness: 300 μm), the diffusion velocities are orders of magnitude greater than the convective velocity

[1]. Hence, the oxygen transport in the avascular region is mainly by diffusion, and a rigid wall model is chosen for this study.

The Carreau model is used with shear-rate-dependent non-Newtonian viscosity as shown in Fig. 1 [3]. As Hct increase, the blood viscosity increases.

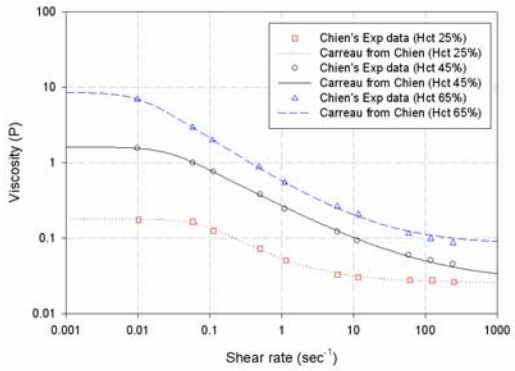

Fig. 1. Viscosity curve with different Hct values

RESULTS AND DISCUSSION

The oxygen concentration contours are shown in the Fig. 2. These contours are observed as a result of the flow recirculation distal to the stenosis. The area where the pO_2 is between 84 – 72 mmHg is smaller as Hct increases from 25 to 65 %. Also as Hct becomes high, the zone of 84 – 72 mmHg pO_2 shifts radially away from the center of arteries. These are because higher viscosity reduces the convective flow of the blood in the lumen. The change of O_2 flux with geometric variation along the axial distance was already analyzed in a paper of our research group [6].

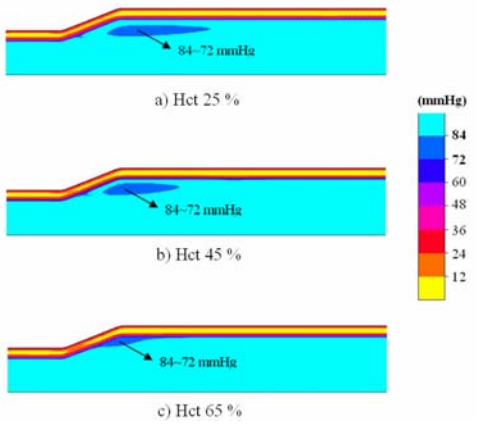

Fig. 2. Oxygen concentration contours

Fig. 3. shows the effect of the Hct variations on the oxygen flux and shear stress at the lumen wall interface. In the proximal section (z < 1 cm), the effect of Hct variations is negligible with a constant diameter of the artery.

In the converging section of the stenosis (1.0 < z < 1.6 cm), the amount of the flux decrease at the start of this section is reduced by ~ 33 % as Hct increases from 25 to 65 %. This is because the slope of the $pO_{2,\ wall}$ increase is gentle in the case of higher Hct and results in less reduction of the numerator of the concentration gradient($\partial c / \partial r$), compared to those of lower Hct cases. Then, the flux increases along the length of the converging section. This is because, along the converging zone, the velocity gradient is higher and the oxygen concentration boundary layer is significantly reduced. Therefore, at Hct 65 %, even though the $pO_{2,\ wall}$ remains lower than those of other two cases, the flux trend is almost similar to others. For the higher Hct, the wall shear stress curve shows the higher value due to the high viscosity, and it is found over the entire region. These analyses explain the result for the throat region in the same way.

In the diverging section (1.9 < z < 2.05 cm), the artery diameter increases and it causes the wall shear stress to reduce. After that, there is flow separation and the reduction in the convective flux causes the oxygen flux to drop drastically. At the flow separation point, the wall shear stress is also close to zero. This trend is similar for all three Hct cases, but the ~ 15 % drop of oxygen flux for the Hct 65 % is much greater than other lower Hct cases (8 % for Hct 25 %). This is because of the significant decrease of convective velocity and $pO_{2,\ wall}$ for the Hct 65 %. Thus, there is a relatively larger decrease in the numerator of the concentration gradient term, ($\partial c / \partial r$).

At the point of flow reattachment, the oxygen flux decreases sharply due to reduced convective flow near the wall. It is found that, for the lower Hct, the location of flow reattachment is further downstream from the end of the stenosis than that for the higher Hct. In addition, the magnitude of the oxygen flux at the reattachment point is decreased with increasing of Hct (from 2.29 to 2.18 ml_o / cm^2 /sec from Hct 25 to 65 %).

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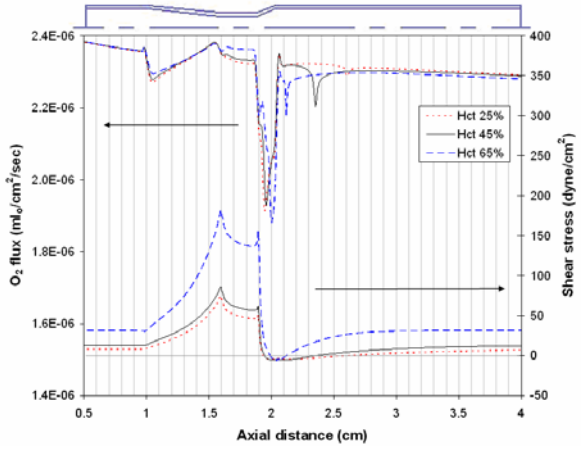

Fig. 3. Effect of Hct variations on O_2 flux and shear stress