COUPLED OXYGEN TRANSPORT TO THE AVASCULAR WALL OF A PRE- AND POST-ANGIOPLASTY CORONARY ARTERY STENOSIS

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ABSTRACT

Coupled oxygen transport to the avascular wall of a coronary artery stenosis is studied by numerically solving the convection and diffusion equations. The O_2 flux to the wall is compared between pre- and post- PTCA (Percutaneous Transluminal Coronary Angioplasty) stenosis arteries to evaluate the effect of the degree of stenosis on the O_2 transport. The factors affecting the O_2 transport, such as, consumption of oxygen in the avascular wall region, *w*, the avascular thickness, δ, supply of oxygen from *vasa vasorum*, nonlinear oxygen binding capacity of the hemoglobin, are taken into account. The O_2 flux increases in the flow acceleration region. It drops at the location of flow separation and reattachment after the stenosis. The drop in the flux at reattachment location for pre- PTCA stenosis is 4 times larger than that for post- PTCA stensosis while it is 3 times larger at flow separation location.

INTRODUCTION

Avascular region of the arterial wall is primarily dependent on the oxygen supply from the blood lumen and supplies from other sources (adventitial vessels and *vasa vasorum*) vary according to species and size of vessels [1]. Zemplenyi et al. [2] have shown that the *vasa vasorum* grows in the subintimal thickening and thus plays a critical role against hypoxia. Banerjee et al. [3, 4] have studied the hemodynamics in the pre- and post- PTCA stenoses arteries. The purpose of this study is to evaluate the effect of varying degree of stenosis on the O_2 transport. Two stenosis geometries representing pre- and post- angioplasty coronary artery stenosis are analyzed and the O_2 flux to the wall is compared between two stenosis geometries.

METHOD

The geometrical details for post- PTCA artery stenosis are taken from Banerjee et al. [3]. The details for pre- PTCA stenosis are taken from Banerjee et al. [4]. The O_2 transport equation in the lumen is [5, 6]:

$$(1+\phi')(Dc/Dt) = D_b \nabla^2 c \qquad (1)$$

and that in the avascular wall region is:

$$(Dc/Dt) = D_w \nabla^2 c - w \qquad (2)$$

where c is the oxygen concentration in ml_o/ml_{blood}. It is related to Po_2 as $c = (\alpha \, . \, Po_2)$, where the value of the solubility coefficients in blood, α_b, and in the wall, α_w, is $3x10^{-5}$ ml_o/ml-mmHg. The quantity ϕ' introduces nonlinearity in the convective transport in equation (1) since it is related to the slope of the nonlinear hemoglobin saturation curve by the relation

$$\phi' = ([H]/\alpha)(\partial S/\partial Po_2) \qquad (3)$$

where, [H] = 0.2 ml_o/ml_{blood} is total oxygen carrying capacity of hemoglobin in blood and S is the saturation function which introduces the oxygen carried by hemoglobin into the equation (1) [5]. Value of O_2 consumption rate in the avascular wall, w, is $1.3x10^{-4}$ ml_o/ml_{tissue}-s as measured by Crawford et al. [7] with a microcathode in normal dog femoral arteries, which are roughly similar in size to human coronary vessels. The blood and wall density is 1.05 gm/cm^3, while diffusivity of oxygen in blood and in the wall is $1.0x10^{-5}$ cm^2/sec. The Carreau model for shear rate thinning non-Newtonian viscosity, having infinite shear rate viscosity of 3.45 cP, is used for the blood. For velocity field calculations, constant flow rate of 50 ml/min (basal), with parabolic profile for velocity, is applied at inlet. No slip boundary condition is applied at the lumen-wall interface. Reynolds number, Re_e, based on diameter is 100 and Peclet number, Pe is $3.45x10^5$. The Schmidt number Sc is 3450. 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. The oxygen transport from the blood in the lumen to the wall has continuity of flux across the endothelial wall. Galerkin finite element method is used for the calculations.

RESULT

Fig 1a and 1b show the oxygen flux to the wall and wall shear stress along the axial length for pre- and post- PTCA stenosis,

respectively. The overall profile of the oxygen flux and wall shear stress along the axial length for post- PTCA stenosis artery is qualitatively similar to that for pre- PTCA stenosis artery.

In the proximal straight arterial section of the pre- PTCA stenosis (fig 1a, thick solid line), the magnitude of the oxygen flux decreases from $2.50\text{x}10^{-6}$ ml_o/cm^2-sec at z = 0.03 cm to $2.26\text{x}10^{-6}$ ml_o/cm^2-sec at z = 0.99 cm. This is because the radial concentration gradient, $\partial c/\partial r$, reduces with length in this section as the concentration boundary layer thickness, ∂r increases. At the start of the converging section, the O_2 flux reduces sharply to $1.02\text{x}10^{-6}$ ml_o/cm^2-sec at z = 1.015 cm. This is because, the concentration gradient drops as a result of the drop in, ∂c, caused by the sudden rise in wall concentration. The oxygen flux increases along the length of the converging section and it reaches to a maximum value of $2.14\text{x}10^{-6}$ ml_o/cm^2-sec at z = 1.58 cm. This increasing trend of the flux is because, towards the end of converging section, concentration boundary layer thickness, ∂r is reduced. The increase in the concentration gradient results in a rise in the oxygen flux. The wall shear stress also increases in the converging section from 23.7 dyne/cm^2 at z = 1.0 cm to 931 dyne/cm^2 at z = 1.6 cm.

In the throat section, as in the proximal section, the drop in the radial concentration gradient causes reduction in the flux. The flux reduces from $2.20\text{x}10^{-6}$ ml_o/cm^2-sec at z = 1.57 cm to $1.17\text{x}10^{-6}$ ml_o/cm^2-sec at z = 1.65 cm. The wall shear stress also drops in this region from 931 dyne/cm^2 at z = 1.6 cm to 525 dyne/cm^2 at z = 1.65 cm.

Near the start of the diverging section at z = 1.66 cm, the high velocity flow is still in contact with the wall. Thus, the convective effect causes a local rise in the oxygen flux to $1.23\text{x}10^{-6}$ ml_o/cm^2-sec. There is a local rise in wall shear stress at this axial location to 605 dyne/cm^2. At about z = 1.68 cm the flow separates and the reduction in the convective flux causes the oxygen flux to drop sharply. It reaches to $8.06\text{x}10^{-7}$ ml_o/cm^2-sec z = 1.81 cm. The magnitude of negative wall shear stress is also close to zero (-1.7 dyne/cm^2) at this location. Qualitatively similar variations in the oxygen flux are observed in the diverging section for post- PTCA stenosis artery. Due to the increase in the negative velocity, as an effect of the recirculation zone, the oxygen flux increases along the diverging section. It reaches to $8.63\text{x}10^{-7}$ ml_o/cm^2-sec near the end of the diverging section at z = 1.82 cm. In the distal region of the stenosis, i.e. from z = 1.825 cm onwards, the oxygen flux increases to $2.34\text{x}10^{-6}$ ml_o/cm^2-sec at z = 3.13 cm, due to the increase in the negative velocity, as a result of the flow recirculation. The magnitude of the negative wall shear stress also increases in the distal region of the stenosis from -2 dyne/cm^2 at z = 1.83 cm to -24 dyne/cm^2 at z = 2.5 cm. Subsequent to this location, the oxygen flux reduces along the length until the point of flow reattachment.

At the point of flow reattachment, i.e. at z = 4.5 cm, it decreases sharply to $1.65\text{x}10^{-6}$ ml_o/cm^2-sec due to reduced convective flow near the wall. At this axial location the wall shear stress is 0 dyne/cm^2. As expected, the location of flow reattachment is farther downstream from the end of the stenosis than that for post- PTCA stenosis. In addition, the drop in the oxygen flux at the location of flow reattachment, for pre- PTCA stenosis, is about 4 times larger than that for post- PTCA stenosis. The oxygen flux again increases to $2.30\text{x}10^{-6}$ ml_o/cm^2-sec at z = 5.12 cm, due to the rise in the positive velocity after the flow reattachment. Subsequent to this location, similar to the straight arterial region proximal to the stenosis, the flux reduces along the axial length because of the increasing thickness of the oxygen concentration boundary layer, ∂r.

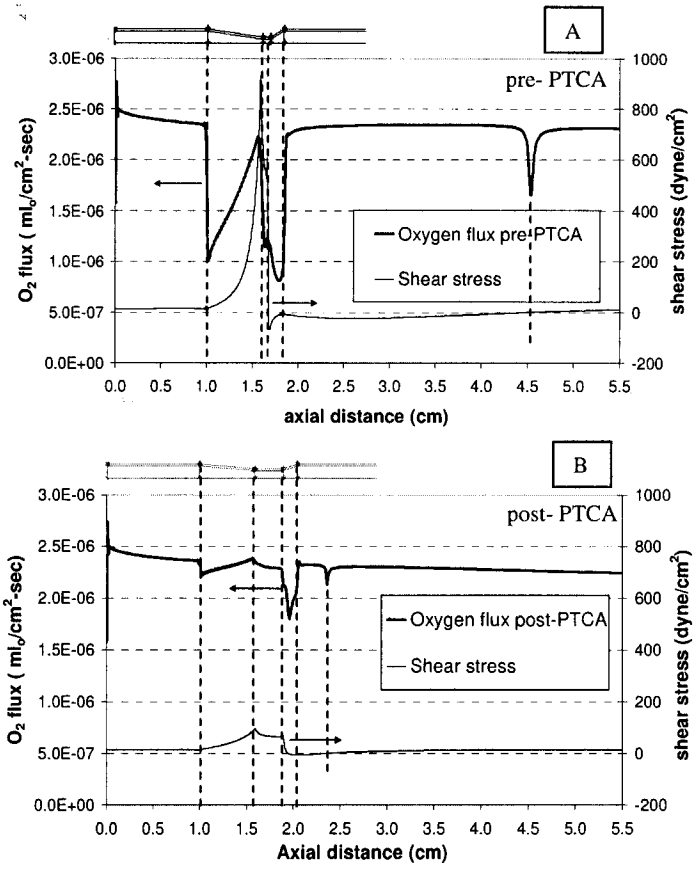

Fig 1: Comparison of O_2 flux to the wall and wall shear stress along axial length between pre- (a) and post- (b) PTCA stenosis.

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