Coupled oxygen transport analysis in the avascular wall of a post-angioplasty coronary artery stenosis

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Abstract. The coupled oxygen transport in the avascular wall of a coronary artery stenosis is studied by numerically solving the convection–diffusion equations. Geometry, replicating residual stenosis after percutaneous transluminal coronary angioplasty (PTCA), is used for the analysis. Important physiological aspects, such as oxygen consumption in the wall, oxygen carried by the hemoglobin, non-Newtonian viscosity of the blood, and supply of oxygen from the vasa vasorum are included. Mean blood flow rate in the lumen is varied from basal to hyperemic conditions. The results show that the $P_{O_2}$ in the medial region of the arterial wall is $\sim10$ mmHg. The oxygen flux to the wall increases in the flow acceleration region, whereas it decreases at the flow reattachment zone. Near the location of flow separation there is a small rise and a sharp fall in the oxygen flux. The minimum $P_{O_2}$ in the avascular wall, $P_{O_2,\text{min}}$, at the point of flow reattachment reduces to $\sim6$ mmHg for a 300 micron wall thickness. For a thinner wall of 200 micron, the $P_{O_2,\text{min}}$ at the location of flow reattachment increases to 6 times that of a 300 micron wall. The $P_{O_2,\text{min}}$ in the wall decreases by 60% when volumetric oxygen consumption is increased by 30% for the same avascular wall thickness.

Keywords: Percutaneous transluminal coronary angioplasty (PTCA), oxygen transport, hypoxia, stenosis, coronary artery, angioplasty, computational fluid mechanics (CFD), numerical methods, oxygen consumption, arterial wall, hemodynamics, convective transport, diffusive transport

1. Introduction

The complete wall of the small arteries and major portion of the wall for large arteries is avascular. Oxygen is supplied to this avascular region by radial diffusion from the vessel lumen and from other sources such as adventitial vessels and vasa vasorum [24]. The abnormalities in the supply of oxygen to the wall are linked with the conditions of hyperoxia or hypoxia, which accelerates atherosclerosis by initiating metabolic abnormalities (e.g., [3,10], and others). Understanding the significance of a wide range of factors that affect the oxygen transport to the arterial wall becomes critical to investigate the pathogenesis of atherosclerosis. Some of these factors are: consumption of oxygen in the wall, thickness of the avascular region and changes in the geometry due to a curvature or presence of plaques.
The available studies on this work can be broadly classified into experimental and numerical studies. For experimental studies, researchers have measured the oxygen tensions in normal or diseased arterial walls either in vivo or ex vivo. The numerical studies deal with the analysis of oxygen transport to the wall in an axi-symmetric or 3D representation of an artery.

Heughan et al. [12], in an experimental study of oxygen tension in atherosclerotic lesions in rabbits, measured the oxygen tensions in atheromata. They found that the lowest tension in the 2-week old injury was 10 mmHg and it was 12 mmHg for a 5-week-old injury. Schneiderman et al. [17] measured the oxygen tensions ex vivo inside the wall of a rabbit thoracic aorta. They showed that the location of minimum \( P_{O_2} \) in the wall indicates that the mid to inner media would be the most prone to hypoxic injury if oxygen transport from the lumen were to become impaired. Jurrus and Weiss [13] measured the oxygen tension profile to be discontinuous at intima, Jurrus and Weiss found that the oxygen tension profiles were smooth continuous curves. They found that the minimum \( P_{O_2} \) decreases from 55 mmHg for the thinnest tissue to zero for 720 micron tissue thickness. Crawford et al. [9] experimentally measured the \( P_{O_2} \) in vivo in dog femoral arteries. They concluded that a significant \( P_{O_2} \) gradient exists between free stream arterial blood and the intima, forming a measurable lumen boundary layer. They further confirmed their earlier finding that the lumen side resistance may be a significant determinant of arterial wall oxygenation. Zemplenyi et al. [24] experimentally showed that the in-growth of vasa vasorum counteracts the impairment of oxygen supply caused due to subintimal thickening. Thus, it is an important mechanism against hypoxia and may be an essential protective factor in atherogenesis.

Back [1] numerically investigated the transport of oxygen to the wall from the blood flowing in a converging-diverging tube without an oxygen consuming wall at a velocity averaged over a cardiac cycle. This study found that there is an increase in oxygen transport to the wall in the accelerated flow regions, while it decreases in decelerated flow regions. Also, the oxygen transport was found to occur primarily across the cell free plasma layer adjacent to the endothelium. Back [2] numerically analyzed the coupled relationship between oxygen transport in the lumen and in the inner wall. The important conclusion of this study was that the lumen side resistance may be more than the avascular wall side resistance, i.e. the hemodynamics primarily controls the oxygen supply to the arterial wall. Back et al. [3] numerically studied the transport of oxygen to the arterial wall in multiple non-obstructive plaque regions over the complete cardiac cycle. They found that the oxygen transport to the wall varied considerably over the cardiac cycle. This study further confirmed that the lumen side resistance is at least an order of magnitude greater than the wall side resistance at the incipient separation locations; while at the reattachment, the lumen side resistance is equal or less than the wall side resistance.

Schneiderman and Goldstick [18] numerically calculated the thickness of the oxygen concentration boundary layer in the lumen in the presence of the oxygen consuming wall. They noted that significant oxygen diffusion gradients extend into the flowing blood well beyond any luminal plasma layer as also found by Back et al. [3], contradicting the earlier finding of Back [1]. Schneiderman et al. [19] numerically studied the mass transport to the walls of stenosed arteries without considering the oxygen consuming wall thickness. They assumed a constant oxygen concentration along the wall. They found that there are regions of both enhanced and impaired mass flux in the diverging part of a constriction. The critical limitation of this study was that it did not consider the effect of oxygen consuming wall thickness and the effect of nonlinear oxygen binding capacity of hemoglobin. Schneiderman et al. [20] studied the effect of pulsatility on the oxygen transport to the oxygen consuming wall by superimposing Womersley type pulsatility on fully-developed flow. They concluded that the pulsatile flow negligibly affects the oxygen transport to the wall.
Rappitsch and Perktold [15] numerically studied the oxygen transport in a stenosed artery at a constant flow rate with shear rate dependent and constant permeability of the wall. They found that in the flow region downstream of the stenosis, oxygen concentration decreases to 75% of the inlet concentration and the wall flux also reduces in this region, thus confirming findings of Back et al. [3]. In another study by the same authors [16], analyzing the transport of albumin in a stenosed artery, they concluded that the endothelial resistance is more compared to the lumen side boundary layer resistance.

Moore and Ethier [14] numerically studied the transport of oxygen in large arteries taking into consideration important physiological factors such as the oxygen consuming wall thickness and non-linear oxyhemoglobin saturation curve. They found the $P_{O_2}$ curve to be continuous showing a luminal oxygen concentration boundary layer and a minimum $P_{O_2}$ in the medial region. The important conclusion of this study was that the oxygen transport is primarily determined by the wall-side effects and the hemodynamics plays a secondary role in oxygen transport to the wall. The important limitations that keep this study from being a comprehensive study are: assumption of Newtonian viscosity for blood, steady flow rate in the lumen neglecting pulsatility and low Reynolds number. Stangeby and Ethier [21], in a recent study, numerically analyzed the transport of macromolecules including porous oxygen consuming wall at a steady flow of blood in the lumen. They found that the mass flux to the wall increases in the acceleration region and it decreases in the deceleration region, reaching minimum at the point of flow separation.

Thus, the conclusions from the previous studies, which are well accepted are: (1) The oxygen concentration boundary layer in the lumen extends well beyond the single plasma layer; (2) The radial oxygen concentration curve is continuous from the middle of the lumen till the outer surface of arterial wall; and (3) The oxygen flux to the wall increases in the acceleration region and decreases in the deceleration region.

The present analysis is intended to be more exhaustive by studying the coupled blood–wall oxygen transport in presence of a moderate stenosis and blood flow varying from basal to hyperemic coronary blood flow conditions with Reynolds number, $Re$ ranging from 100 to 360. Further, the use of more physiological conditions such as oxygen consuming avascular wall, supply of oxygen from other sources such as vasa vasorum, non-Newtonian viscosity of blood makes this study more comprehensive and physiologically realistic. The non-linear oxygen binding capacity of the hemoglobin will have an effect of releasing more oxygen at lower $P_{O_2}$, which, in turn leads to more oxygen available for diffusion. Hence, this non-linearity due to oxygen hemoglobin saturation curve is retained while solving the concentration equation.

2. Methodology

2.1. Geometry

Figure 1 shows the geometry used for the analysis in moderately stenosed coronary artery representing post-PTCA condition. The geometrical details for the plaque are taken from the in vivo data set of Wilson et al. [23] in a group of 32 patients undergoing PTCA and the analysis by Banerjee et al. [5]. It is an axi-symmetric model of an artery of mean diameter $d_e = 3$ mm ($A_e = 7.0 \pm 0.1 \text{ mm}^2$). After PTCA, the average minimum diameter in the plaque is, $d_m = 1.8$ mm ($A_m = 2.5 \pm 0.1 \text{ mm}^2$) forming 64% area stenosis. The length of converging section is $l_c = 6$ mm, that of throat section is $l_m = 3$ mm. The length of diverging section is $l_r = 1.5$ mm. Distal to the stenosis, 60 mm length of the artery is included in the geometry to avoid the influence of the downstream boundary condition. Zemplenyi et al. [24] showed by experimental measurements that the vasa vasorum grows in the plaque as a counter mechanism to the
hypoxia. Hence, the thickness of avascular region, $\delta$, is 300 micron along the complete axial length. In the avascular wall region, the diffusion velocities are an order of magnitude greater than the convective velocity [1]. Hence, the oxygen transport in the avascular region is mainly by diffusion, and the wall is considered to be rigid. The physical properties such as density, diffusivity and oxygen consumption of the avascular region are considered to be spatially uniform. The \textit{vasa vasorum} is considered to be symmetric around the arterial axis thus forming an annulus of avascular, oxygen consuming wall region. Smaller values of $\delta$ are also studied.

2.2. Problem formulation

The oxygen concentration distribution in an artery is determined by the velocity field. Hence, in order to establish the velocity distribution, the following continuity and momentum conservation equations are solved.

Continuity equation:

$$ \rho \nabla \cdot (\vec{v}) = 0, \quad \text{(1)} $$

where $\rho$ is the density of blood and $\vec{v}$ is the velocity vector.

Momentum conservation equation:

$$ \rho \frac{\partial}{\partial t} (\vec{v}) + \rho \nabla \cdot (\vec{v} \vec{v}) = -\nabla p + \nabla \cdot (\bar{\tau}), \quad \text{(2)} $$

where $p$ is the static pressure and $\bar{\tau}$ is the stress tensor. The stress tensor $\bar{\tau}$ is given by,

$$ \bar{\tau} = \mu \left[ (\nabla \vec{v} + (\nabla \vec{v})^T) \right], \quad \text{(3)} $$

where $\mu$ is the viscosity.
The transport of oxygen from its point of intake in the lungs to the point of its consumption in the living tissue takes place by convection and diffusion. The diffusivity of oxygen in blood being very low, then the transport of oxygen in the lumen is a convection dominated process with Peclet number, $Pe$, being a very high value ranging from $3.54 \times 10^5$ to $12.7 \times 10^5$.

The oxygen is carried by the blood in two forms; the dissolved oxygen in plasma and the oxygen that is bound with the hemoglobin. Each hemoglobin molecule can carry four molecules of oxygen. The saturation of the hemoglobin, $S$, defined as the ratio of the oxyhemoglobin to total hemoglobin, is a function of oxygen partial pressure, $P_{O_2}$. The variation of the hemoglobin saturation function, $S$ with $P_{O_2}$, is nonlinear. Further, this variation curve shows reduced saturation at lower $P_{O_2}$ and increased saturation at higher $P_{O_2}$ [11]. Thus, due to the nature of the oxy-hemoglobin saturation curve, more oxygen is available for diffusion at lower $P_{O_2}$ and less oxygen is available at higher $P_{O_2}$. The transport of oxygen across the red cell membrane is assumed to be instantaneous.

Taking into account the oxygen carried by the hemoglobin and that dissolved in plasma, the oxygen mass conservation equation in the lumen becomes

$$\frac{\partial}{\partial t}(c + \chi) + u \frac{\partial}{\partial z}(c + \chi) + v \frac{\partial}{\partial r}(c + \chi) = D_b \left[ \frac{\partial^2 c}{\partial r^2} + \frac{1}{r} \frac{\partial c}{\partial r} + \frac{\partial^2 c}{\partial z^2} \right], \quad (4)$$

where, $c$, is the oxygen concentration in ml of oxygen/ml of blood. $\chi$ is the oxygen carried by the hemoglobin. The terms on the left hand side of the equation represent variation with time and the convective transport. The terms on the right hand side of the equation represent the diffusive transport. Since the hemoglobin is carried only by convection and not by diffusion, the term $\chi$ is absent from the right side of the equation. The oxygen concentration, $c$, in this equation, is related to $P_{O_2}$ by the solubility relation.

$$c = (\alpha \cdot P_{O_2}), \quad (5)$$

where, $\alpha$, is the solubility coefficient for oxygen.

Equation (4) can be re-written as

$$(1 + \phi') \frac{Dc}{Dt} = \nabla \cdot (D_b \nabla c). \quad (6)$$

The non-dimensional term $\phi'$ in this equation is related with the slope of the oxyhemoglobin saturation curve through the relation,

$$\phi' = \frac{\partial \chi}{\partial c} = \left( \frac{H}{\alpha} \right) \left( \frac{\partial S}{\partial P_{O_2}} \right). \quad (7)$$

Here $H$ is total oxygen carrying capacity of hemoglobin in blood, given as

$$[H] = 1.34 C_{hb,sat}, \quad (8)$$

where, 1.34 is the number of milliliter of oxygen per gram of hemoglobin and $C_{hb,sat}$ is the density of saturated hemoglobin, the value of which is 0.15 g/ml of blood for normal blood [3].

Thus, the term $(1 + \phi')$ in Eq. (6), which is otherwise the simple species conservation equation, takes into account the oxygen released by the oxyhemoglobin. The slope of the oxyhemoglobin saturation
curve \((\partial S/\partial P_{O_2})\) is non-linear and hence, the left hand side of the Eq. (6) becomes non-linear making it more complex. The values for the slope \((\partial S/\partial P_{O_2})\) are taken from Colton and Drake [8].

The lumen side oxygen transport equation is coupled with oxygen transport and consumption in the avascular wall. The oxygen concentration conservation equation in the wall region is

\[
\frac{\partial c}{\partial t} = \nabla \cdot (D_w \nabla c) - \dot{q},
\]

where, \(\dot{q}\), is a constant volumetric consumption rate of oxygen by the cells within the wall region. It may be noted that the convective transport terms and the term \(\phi'\) are absent in Eq. (9).

2.3. Material properties

The density of the blood is 1.05 g/cm\(^3\). The diffusivity of oxygen in the blood, \(D_b\), and that in the wall region, \(D_w\), is taken to be \(1.0 \times 10^{-5}\) cm\(^2\)/s. The value of the solubility coefficient for oxygen in blood and in the wall region is \(3 \times 10^{-5}\) ml\(_o\)/ml-mmHg [15]. The value of the constant volumetric consumption rate of oxygen in the arterial wall, \(\dot{q}\), is \(1.3 \times 10^{-4}\) ml\(_o\)/ml-tissue-s (Crawford et al. [9], as obtained from microcathode measurements \textit{in vivo} in normal dog femoral arteries which are roughly the size of some human coronary arteries). Lower and higher values of \(\dot{q}\) are also studied.

For viscosity of the blood, the Carreau model is used with shear-rate-dependent non-Newtonian viscosity. The local shear rate is calculated from the velocity gradient through the second invariant of the rate of strain tensor [7]. The corresponding equations for local shear rate \(\dot{\gamma}\) and blood viscosity \(\eta\) are given by,

\[
\dot{\gamma} = \sqrt{\frac{1}{2} \left[ \sum_i \sum_j \dot{\gamma}_{ij} \dot{\gamma}_{ji} \right]},
\]

where \(\dot{\gamma}_{ij}\) is the rate of strain tensor and \(i, j = 1, 2\) and 3 for three dimensional flows.

\[
\eta = \eta_{\infty} + (\eta_0 - \eta_{\infty}) \left[ 1 + (\lambda \dot{\gamma})^2 \right]^{(n-1)/2},
\]

where time constant \(\lambda = 3.313\) s, constant \(n = 0.3568\), zero shear viscosity \(\eta_0 = 0.56\) g/cm\(-s\) and infinite shear viscosity \(\eta_{\infty} = 0.0345\) g/cm\(-s\). The model constants were obtained by curve-fitting blood viscosity data in the literature. From these relations, the local shear stress \(\tau = \eta \dot{\gamma}\) is calculated.

2.4. Boundary conditions

Velocity boundary condition: For velocity distribution calculations, no-slip boundary condition is applied at the lumen-wall interface. No radial flow boundary condition is applied at the axis. Constant flow rates of 50 (basal), 100 and 180 ml/min (hyperemic), with parabolic profile for axial velocity, are applied at inlet. Reynolds number, \(Re_e\), based on inlet diameter, range from 100 to 360. The Schmidt number \(Sc\) is 3500.

Species boundary condition: For species distribution, uniform concentration of oxygen, corresponding to normal blood \(P_{O_2}\) of 95 mmHg, is applied at inlet [13]. At \textit{vasa vasorum} oxygen concentration corresponding to \(P_{O_2,V}\) of 45 mmHg is applied [9]. The oxygen concentration is calculated using Eq. (5). Zero flux boundary condition is applied at the axis along the lumen. The oxygen transport from the blood in the lumen to the wall has continuity of species and flux across the endothelial wall.
2.5. Finite element method

Four node quadrilateral element mesh is generated with \(~80,000\) number of elements. To capture the oxygen concentration boundary layer in the lumen and accurately calculate the oxygen flux to the wall, mesh grading is used while meshing the lumen and wall regions such that the first node from the lumen-wall interface lies at \(3.33 \times 10^{-4}\) times the lumen diameter. The skewness of the elements is not allowed to increase over 0.25.

Galerkin Finite element method [4] is used for the calculations. The computation is carried out in two steps. First, velocity distribution is calculated in the complete geometry. In the second step this velocity distribution is used to calculate oxygen concentration distribution. Second order streamline upwinding scheme is used for the oxygen concentration calculations while no upwinding is used for the flow computations.

Mesh independency was checked by increasing the number of elements by 20\% over the previous mesh and both the results were compared. The mesh with increased number of elements showed less than 3\% difference in oxygen concentration values. Computations were carried out on a Microsoft Windows\textsuperscript{XP} Professional (Version 5.1) workstation with Intel (Pentium IV) 2.4 GHz processors with 1.0 GB RAM and 80 GB hard drive available at the Bio-Fluids Heat and Mass Transfer Laboratory at the University of Cincinnati. The average CPU time for one set of calculations was 60 min.

3. Results

To check the accuracy of the numerical computations, a simulation was done to replicate the results obtained by Schneiderman et al. [20]. The geometry, material properties and boundary conditions used for this simulation were the same as used by Schneiderman et al. Figure 2 shows the radial oxygen partial pressure profiles obtained by these two studies. The difference between the oxygen concentration values is less than 1\%. This replication of the past results validates and provides the confidence in the results obtained during the present study.

![Fig. 2. Comparison between results obtained by the present study and those obtained by Schneiderman et al. [20] using same geometry, properties and boundary conditions.](image)
Figure 3 shows oxygen species contours similar to the flow streamlines, distal to the stenosis at basal flow condition. These contours are observed as a result of the flow recirculation distal to the stenosis. The flow separates immediately after the stenosis throat causing convection of oxygen away from the wall. Since the magnitudes of the velocities are lower towards the core of the recirculation, the oxygen concentration reduces radially towards the core. It decreases to \( \sim 81\% \) of the inlet concentration. The magnitude of negative velocity increases from the core of the recirculation region towards the arterial wall, and, hence, the oxygen concentration also increases near the wall. The oxygen concentration is \( \sim 91\% \) of the inlet concentration near the wall region.

In the medial region of the avascular wall, the oxygen concentration drops considerably, to \( \sim 9\% \) of the inlet concentration due to the consumption of oxygen in the wall (Fig. 3). Since the \textit{vasa vasorum} acts as an additional supply of oxygen, the oxygen concentration increases radially towards the outer surface of the arterial wall. This behavior in the avascular region remains the same even in the throat because of the in-growth of the \textit{vasa vasorum}. The oxygen concentration contours shown in the lumen are similar to those observed by Moore and Ethier [14] and Rappitsch and Perktold [15].

Figure 4 shows the radial \( P_{O_2} \) variation at key points along the axial length in a moderately stenosed artery at basal flow condition. These points are chosen such that they represent all key geometrical variations in a stenosed artery. Proximal to the stenosis, at \( z = 0.95 \) cm, i.e. in the normal artery, the \( P_{O_2} \) is equal to the inlet \( P_{O_2} \) towards the luminal axis. \( P_{O_2} \) starts decreasing near the wall forming an oxygen concentration boundary layer thickness of \( \sim 60 \) microns. The \( P_{O_2} \) reaches to 76.5 mmHg at the wall \( (P_{O_2,w}) \). Thus, there is a reduction of 18.5 mmHg from mean arterial blood \( P_{O_2} \) to \( P_{O_2,w} \). This is in good agreement with the experimental measurements of Crawford et al. [9]. Their experimental results show that there is a difference of 19 mmHg between mean arterial \( P_{O_2} \) and \( P_{O_2,w} \). The \( P_{O_2} \) reduces further in the avascular region due to the consumption by tissue for its metabolic activities, causing \( P_{O_2,\text{min}} \) of 10.3 mmHg. The \( P_{O_2,\text{min}} \) occurs at about the middle of the avascular section but slightly shifted towards the outer surface of the arterial wall, since the oxygen flux from the lumen is larger as compared to that
Fig. 4. Radial $P_{O_2}$ profiles at different locations along the axis. 64% area stenosis, $Q = 50$ ml/min, $\delta = 300$ micron, $\dot{q} = 1.3 \times 10^{-4}$ ml/mtissue-sec.

from the *vasa vasorum*. The $P_{O_2,\text{min}}$ occurs at 166 micron from the lumen–endothelium interface. The $P_{O_2}$ starts increasing radially towards the outer surface of the arterial wall as *vasa vasorum* acts as an additional source of oxygen.

At the middle of the throat, i.e. at $z = 1.75$ cm in Fig. 4, the $P_{O_2}$ variations remain similar to those observed in the proximal region. The oxygen concentration boundary layer reduces to $\sim 27$ micron due to the high magnitude of velocity gradients. The value of $P_{O_2,\text{min}}$ increases marginally from 10.3 mmHg to 10.9 mmHg, but it occurs at radially the same location, i.e. at 166 micron from the throat lumen–endothelium interface.

At $z = 2.15$ cm, i.e. in the recirculation region, immediately distal to the stenosis, the radial $P_{O_2}$ decreases to 79 mmHg in the lumen (Fig. 4). This reduction in $P_{O_2}$ is because of the flow recirculation. $P_{O_2}$ increases radially towards the wall, away from the core of the recirculation, due to the increase in convective flow in the negative direction. It again reduces very near to the wall, forming a relatively thinner lumen boundary layer of $\sim 40$ micron. In the avascular region $P_{O_2}$ reduces to a value of $P_{O_2,\text{min}} = 8.8$ mmHg. The radial location of $P_{O_2,\text{min}}$ is 166 micron from the lumen–endothelium interface. The $P_{O_2}$ curve at $z = 7$ cm, i.e. the region far distal to the stenosis, remains similar to that at $z = 0.95$ cm. Since
Fig. 5. Effect of the flow rate on the radial $P_{O_2}$ profiles, $z = 2.15$ cm, $\delta = 300$ micron, $\dot{q} = 1.3 \times 10^{-4}$ ml/o/mltissue-sec.

there is no flow recirculation at this point the profile shows no change in the lumen until the boundary layer starts. The thickness of the oxygen concentration boundary layer, at this location, is $\sim 90$ micron. The value of $P_{O_2, \text{min}}$ is lower than that at $z = 0.95$ cm due to the continuous consumption along the axial distance. The $P_{O_2, \text{min}}$ reaches a very low value of 4.7 mmHg. The radial location of $P_{O_2, \text{min}}$ is 166 micron from lumen–endothelium interface, as in all other cases.

The critical observation from the Fig. 4 is that the medial region of the avascular wall always has a low value of $P_{O_2}$, indicating that this region is most prone to hypoxic injury if the transport of oxygen from any source, either the lumen or vasa vasorum, is impaired. This is in agreement with the experimental results shown by Schneiderman et al. [17].

Figure 5 shows the effect of the flow rate on the radial $P_{O_2}$ profiles at $z = 2.15$ cm, just distal to the stenosis. The flow rates are increased from basal to hyperemic conditions. For higher flow rate, the oxygen concentration is higher in the recirculation zone. This is due to the larger magnitudes of flow velocities in the recirculation zone for higher flow rate. The value of $P_{O_2, w}$ increases from 73 mmHg for basal flow to 78 mmHg for hyperemic flow.

The important observation in the Fig. 5 is that the flow rate affects the $P_{O_2}$ values in the avascular region. The $P_{O_2, \text{min}}$ value increases by 24% from basal to hyperemic flow conditions. For basal flow, the value of $P_{O_2, \text{min}}$ is 8.8 mmHg, while that for hyperemic flow is 10.9 mmHg. The changes in the radial position of $P_{O_2, \text{min}}$ are insignificant for higher flow rates. For hyperemic flow, it is 165 micron from the lumen–endothelium interface versus 166 micron for the basal flow rate. Thus, in spite of the increase in the $P_{O_2, \text{min}}$ with flow rate, the medial region in the avascular wall has a low value of $P_{O_2}$ from basal to hyperemic flow conditions.

Figure 6 shows the effect of the flow rate on the oxygen flux at the lumen wall interface for the stenosed artery. The flow rate is increased from basal to hyperemic. The flux to the wall in an unstenosed artery (our previous study; [22]) of the same diameter ($d_e = 3$ mm) for basal flow condition is also shown in this figure (thin solid line). In the proximal region of the stenosis, the flux to the wall matches that of
Fig. 6. Effect of flow rate on the oxygen flux at the lumen–endothelium interface, $\delta = 300 \, \text{micron}$, $\dot{q} = 1.3 \times 10^{-4} \, \text{ml}_o/\text{ml}_tissue–\text{sec}$.

Fig. 6. Effect of flow rate on the oxygen flux at the lumen–endothelium interface, $\delta = 300 \, \text{micron}$, $\dot{q} = 1.3 \times 10^{-4} \, \text{ml}_o/\text{ml}_tissue–\text{sec}$.

In an unstenosed artery at basal flow. In this region, the magnitude of the flux reduces with axial distance from $2.50 \times 10^{-6} \, \text{ml}_o/\text{cm}^2–\text{sec}$ at $z = 0.03 \, \text{cm}$ to $2.33 \times 10^{-6} \, \text{ml}_o/\text{cm}^2–\text{sec}$ at $z = 1.0 \, \text{cm}$ (thick solid line). This is because, along the axial length, the oxygen boundary layer thickness increases, and due to the constant consumption of oxygen in the avascular region, $P_{O_2,w}$ reduces. There is a relatively larger increase in the denominator of the concentration gradient term, $\partial c/\partial r$, as compared with the increase in the numerator term. This, in turn, reduces the flux.

In the converging section of the stenosis, the increase in flow velocity causes $P_{O_2,w}$ to increase along the axial direction, as will be discussed subsequently in connection with Fig. 8. At the start of this section, the flux to the wall (Fig. 6) reduces sharply to $2.20 \times 10^{-6} \, \text{ml}_o/\text{cm}^2–\text{sec}$ at $z = 1.015 \, \text{cm}$. This is due to the increase in $P_{O_2,w}$, causing significant reduction in the numerator of the concentration gradient term, $\partial c/\partial r$, as compared with the reduction in the denominator. This, in turn, reduces the overall concentration gradient in radial direction, and thus, the oxygen flux. This rapid decrease in the oxygen flux at the start of the stenosis is the result of the sharp change in the arterial diameter. In more diffused lesions than used for the present analysis, the change in the arterial diameter can be more gradual. In that case the drop in the oxygen flux at the start of converging section will not be as sharp as observed in the present case. The flux increases along the length of the converging section to $2.37 \times 10^{-6} \, \text{ml}_o/\text{cm}^2–\text{sec}$ at $z = 1.57 \, \text{cm}$. This increasing trend of the flux is because, towards the end of converging section, the velocity gradient is higher and the oxygen concentration boundary layer is significantly reduced. This causes the denominator of the concentration gradient term, $\partial c/\partial r$, to decrease more than the decrease in
the numerator term. Hence, in spite of the increasing $P_{O_2, w}$ along the length, the increase in the gradient results in a rise in the flux in the converging section.

In the throat region (Fig. 6), similar to the proximal section, the thickness of the boundary layer grows, causing the denominator of the concentration gradient term $\partial c/\partial r$ to increase. This results in a drop in the radial concentration gradient, which, in turn, reduces the flux along the axial length from $2.33 \times 10^{-6}$ $ml/cm^2-sec$ at $z = 1.6$ cm to $2.27 \times 10^{-6}$ $ml/cm^2-sec$ at $z = 1.88$ cm. At the start of the diverging section at $z = 1.9$ cm the throat diameter increases, causing the wall shear stress to reduce. This reduces the flux to the wall extensively to $2.10 \times 10^{-6}$ $ml/cm^2-sec$. At about $z = 1.91$ cm, the early part of the diverging region, the high velocity flow is still in contact with the wall, thus, there is a local rise in the flux to $2.11 \times 10^{-6}$ $ml/cm^2-sec$. Downstream from this location, near $z = 1.95$ cm, there is a sharp drop in the flux to a local minima of $1.79 \times 10^{-6}$ $ml/cm^2-sec$. This behavior is similar to that observed by Schneiderman et al. [19]. At this location, there is a flow separation, causing a stagnation zone. The low wall shear stress (close to 0 dyne/cm$^2$) in this region results in a drop in the convective flux to the arterial wall.

Due to the increase in the negative velocities near the wall, as an effect of the recirculation zone, the oxygen flux increases along the diverging section (Fig. 6). It reaches a local maxima of $2.35 \times 10^{-6}$ $ml/cm^2-sec$ near the end of the diverging section at $z = 2.06$ cm. In the distal to stenosis region, i.e. from $z = 2.06$ cm onwards, the flux reduces gradually until the point of flow reattachment. At the point of flow reattachment, i.e. at $z = 2.37$ cm, it decreases sharply to $2.12 \times 10^{-6}$ $ml/cm^2-sec$ due to reduced convective flow near the wall. The flux again increases to $2.29 \times 10^{-6}$ $ml/cm^2-sec$ at $z = 2.54$ cm due to the increase in positive velocity, caused by the flow reattachment. Subsequent to this location, the flux reduces along the axial length similar to the proximal section. Thus, in the straight portions of the artery, such as proximal, throat and distal regions of the stenosis, the flux to the wall is mainly influenced by the concentration boundary layer thickness. In the varying diameter regions, such as, converging and diverging sections of the stenosis, the flux is controlled by the combined effect of the concentration difference near the wall and the boundary layer thickness associated with it.

The variations in the oxygen flux for hyperemic flow (thin dotted line with diamond) show similar trends as observed for the basal flow (Fig. 6). For the hyperemic flow, the peak oxygen flux in the distal region and flux at the end of proximal region of the stenosis, are higher by $\sim 2.5\%$ and $\sim 3.4\%$, respectively. In these regions, the larger velocity gradient for hyperemic flow results in higher oxygen flux to the wall. The drop in the flux at the start of the converging, throat and diverging sections for hyperemic flow is greater than that for the basal flow. The lowest oxygen flux in the converging and throat region of the stenosis is $\sim 14\%$ lower, and the local minimum of flux in the diverging section is 26% lower for the hyperemic flow. At the location of flow reattachment, at $z = 3.44$ cm, the oxygen flux reduces sharply to $2.07 \times 10^{-6}$ $ml/cm^2-sec$. As expected, this location is farther distal from the end of stenosis for hyperemic flow. In addition, the drop in the flux at this location is 58% more than that for the basal flow.

Figure 7 shows the variation of $P_{O_2, min}$ along the axial length for the stenosed artery from $z = 0.1$ cm to $z = 2.8$ cm for basal flow condition for different wall thicknesses: 200, 250 and 300 micron. In conjunction with Fig. 6, a number of important observations may be noted from Fig. 7. In the proximal section, for 300 micron wall thickness, the $P_{O_2, min}$ drops from 14.2 mmHg at $z = 0.1$ cm to 10.3 mmHg at $z = 1.0$ cm. This drop is attributed to the drop in the oxygen flux. The $P_{O_2, min}$ increases along the converging section, as a result of the rise in the oxygen flux. It reaches a local maximum of 12 mmHg at $z = 1.6$ cm. The $P_{O_2, min}$ decreases along the throat section as the oxygen flux decreases, and it reaches 10.6 mmHg at $z = 1.9$ cm. Since the oxygen flux rises at the start of the diverging section, the $P_{O_2, min}$
Fig. 7. Effect of avascular wall thickness, $\delta$, on the variation of $P_{O_2, \text{min}}$ in the wall along the axial length at basal flow, $Q = 50 \text{ ml/min}$, from $z = 0.1 \text{ cm}$ to $2.8 \text{ cm}$. $q = 1.3 \times 10^{-4} \text{ ml}_o/\text{ml}_\text{tissue-sec}$. It also increases to 15.6 mmHg at $z = 1.92 \text{ cm}$. It reduces to 13.4 mmHg at $z = 1.95 \text{ cm}$ because of the reduction in the flux at the location of flow separation. The $P_{O_2, \text{min}}$ then increases to 14.5 mmHg at $z = 2.0 \text{ cm}$, as the flux increases due to the increased negative flow which is caused by flow recirculation. In the distal region of the stenosis, the $P_{O_2, \text{min}}$ drops significantly at the location of flow reattachment, i.e. at $z = 2.37 \text{ cm}$, to 5.7 mmHg. At this location, the oxygen flux to the wall drops sharply as shown in Fig. 6. The values of $P_{O_2, \text{min}}$ for a thinner wall of 250 and 200 micron are higher than those for a thicker wall of 300 micron. This is because the thinner wall has less overall consumption of oxygen. At the point of flow reattachment, the $P_{O_2, \text{min}}$ increases from 5.7 mmHg for a 300 micron wall to 34.3 mmHg for a 200 micron wall. The overall profile of $P_{O_2, \text{min}}$ values along the axial direction for 250 and 200 micron wall thicknesses remains qualitatively similar to that of a 300 micron wall thickness.

Thus, the locations of flow separation and reattachment are subjected to increased variations in the supply of oxygen from luminal blood. The point of flow reattachment where the $P_{O_2, \text{min}}$ drops to a low value may be especially prone to hypoxic injury.

Figure 8 shows the effect of flow rate on the $P_{O_2, \text{w}}$. The variation of $P_{O_2, \text{w}}$ with axial length is plotted for basal and hyperemic flow conditions. As a reference, the $P_{O_2, \text{w}}$ for an unstenosed artery for basal flow condition is also shown in this figure (our previous study; [22]). In the proximal region, the $P_{O_2, \text{w}}$ for basal flow matches with that in an unstenosed artery for the same flow. In this upstream region, $P_{O_2, \text{w}}$ decreases from the inlet value of 95 mmHg to 76 mmHg at $z = 1.0 \text{ cm}$, due to the constant consumption of oxygen in the avascular wall region.
In the converging region (Fig. 8), the $P_{O_2,w}$ increases from 76 mmHg at the start to 80 mmHg at $z = 1.61$ cm. In this section, due to the increase in wall shear stress along the length (shown in [5]), oxygen flux increases. This, in turn, increases the $P_{O_2,w}$. The $P_{O_2,w}$ decreases along the throat from 80 mmHg at $z = 1.61$ cm to 77.5 mmHg at $z = 1.9$ cm, due to the constant consumption of oxygen in the avascular region. In the diverging region, there is a sharp drop in $P_{O_2,w}$ to 66 mmHg at $z = 1.95$ cm, due to the reduced convective flux at flow separation. Distal to this drop, the $P_{O_2,w}$ increases in the diverging section as the negative wall shear stress increases due to recirculation, and it reaches 73 mmHg at $z = 2.17$ cm. Subsequent to this location, $P_{O_2,w}$ decreases until the point of flow reattachment. At $z = 2.37$ cm, i.e the point of reattachment, $P_{O_2,w}$ drops to 64 mmHg. At the reattachment zone the wall shear stress is close to 0 dyne/cm$^2$, causing the local minima for $P_{O_2,w}$. Distal to this point, the $P_{O_2,w}$ increases to 71 mmHg at $z = 2.65$ cm due to the rise in the positive wall shear stress after flow reattachment. The $P_{O_2,w}$ reduces distal to this location, due to the constant consumption of oxygen in the wall and increased concentration boundary layer thickness.

The variations in $P_{O_2,w}$ with axial distance for hyperemic flow (Fig. 8) are similar to those for the basal flow. In the diverging section there is a minimal drop in $P_{O_2,w}$ from 76.7 mmHg at $z = 1.93$ cm to 76.2 mmHg at $z = 2.07$ cm. This deviation, in comparison to the basal flow, is because the negative wall shear stress drops in this length reducing oxygen flux to the wall. The $P_{O_2,w}$ increases to 78.5 mmHg at $z = 2.5$ cm due to the increase in the negative wall shear stress. Distal to this location, $P_{O_2,w}$ reduces until the point of flow reattachment. At the location of flow reattachment, i.e. at $z = 3.4$ cm, the $P_{O_2,w}$ drops to 71.4 mmHg. This drop in $P_{O_2,w}$ at the reattachment point, is 20% smaller than that for the basal
flow. In general the values of the $P_{O_2,w}$ for hyperemic flow are always higher than those for basal flow, associated with the complicated oxygen flux to the wall.

Figure 9 shows the effect of variation in volumetric oxygen consumption, $\dot{q}$, in the avascular region on the radial $P_{O_2}$ profile, at $z = 2.15$ cm for basal flow condition. For a lower value of oxygen consumption, $\dot{q} = 1 \times 10^{-4}$ ml/oxygen/ml tissue/sec and wall thickness of 300 micron, the $P_{O_2,min}$ in the wall is 21.5 mmHg, and it occurs at 166 micron from the lumen–endothelium interface. For a medium value of consumption, $\dot{q} = 1.3 \times 10^{-4}$ ml/oxygen/ml tissue/sec and wall thickness of 300 micron, the $P_{O_2,min}$ reduces to 8.8 mmHg at 166 micron from the lumen–endothelium interface. For higher consumption, $\dot{q} = 2 \times 10^{-4}$ ml/oxygen/ml tissue/sec, the increased overall consumption of oxygen necessitates larger in-growth of vasa vasorum, and the thickness of the avascular wall reduces to 225 micron. Thus, with higher consumption but a relatively thinner wall, the $P_{O_2,min}$ reaches 14.1 mmHg at 126 micron from the lumen–endothelium interface. In the flow recirculation region in the lumen, the $P_{O_2}$ values increase with decreasing oxygen consumption. The lowest $P_{O_2}$ in the recirculation region for $\dot{q} = 1 \times 10^{-4}$ ml/oxygen/ml tissue/sec is 82 mmHg, and that for $\dot{q} = 2 \times 10^{-4}$ ml/oxygen/ml tissue/sec is 76.5 mmHg.

4. Discussion

This study presents a comprehensive analysis of the coupled oxygen transport to the avascular wall of a moderately stenosed coronary artery. Important physiological aspects, such as oxygen carried by the hemoglobin, non-linear viscosity of the blood, and consumption of oxygen in the wall, are accounted for. The lumen side and wall side factors, that influence the oxygen transport, are varied to quantify their effects.

The radial $P_{O_2}$ profiles show that the $P_{O_2}$ curve is continuous from the arterial lumen to the outer surface of the arterial wall. The lumen side oxygen concentration boundary layer is $\sim$80 micron thick.
The $P_{O_2}$ drops in the medial region to a value of $\sim 10$ mmHg and it rises radially towards the outer surface of the arterial wall. The location of the medial region of the arterial wall for a normal as well as stenosed artery always has a low value of $P_{O_2}$. Thus, this region is highly susceptible to hypoxic injury. The in-growth of *vasa vasorum* in a stenosis plays a key role in maintaining luminal patency and acts against atherosclerosis. It can also be inferred from the results that a situation where there is insufficient in-growth of *vasa vasorum* in the arterial wall can have an adverse effect due to reduced $P_{O_2}$ values in the avascular region. Presence of a stenosis in the artery, even in the post-PTCA condition, has residual stenosis which has a significant impact on the supply of oxygen to the arterial wall. The oxygen flux increases in the flow acceleration region. The point of flow separation and reattachment are prime locations where oxygen supply shows increased variations. The locations of flow stagnation (separation and reattachment), where the $P_{O_2}$ value is low, may be the sites of lesion growth.

The important conclusion from this study is that the transport of oxygen to the avascular region is complex and is affected by both lumen and wall parameters. Lumen-side factors include varying flow rates and different hemodynamic regions produced by the presence of a stenosis. The wall-side factors include volumetric oxygen consumption and avascular wall thickness. The change in the flow rate through the lumen from basal to hyperemic condition causes $P_{O_2}$ in the medial region to rise by $\sim 25\%$. The flow stagnation zones created by changes in the geometry, such as the presence of a stenosis, affect the oxygen supply to the wall. The oxygen flux drops sharply at the flow stagnation locations, causing $P_{O_2}$ in the medial region to drop. The position of outer surface of the arterial wall may also vary, causing the avascular thickness to increase or decrease. A thicker avascular region will have larger overall oxygen consumption. This will require increased oxygen supply in order to maintain normal metabolic activities. The results show that the $P_{O_2}$ values in the medial region are higher for a thinner wall. The $P_{O_2,\text{min}}$ at the location of flow reattachment for a thinner wall of 200 micron is 6 times larger than that for a 300 micron wall. The volumetric oxygen consumption in the avascular wall also has a significant impact on the $P_{O_2}$ values. Higher volumetric consumption results in larger overall oxygen consumption, requiring increased supply of oxygen. Sufficient in-growth of *vasa vasorum* is needed for higher volumetric consumption to maintain adequate $P_{O_2}$ values. For example, higher oxygen consumption, $\dot{q} = 2 \times 10^{-4}$ mlO$_2$/mltissue-sec, necessitates larger in-growth of *vasa vasorum* so that the avascular wall thickness is reduced to $\sim 225$ micron.

As mentioned earlier, this study is for an artery under a post-PTCA condition. A geometry representing the pre-PTCA condition with a higher degree of area occlusion can be studied as an extension to this study. Also, the physiological properties in the arterial wall are considered to be spatially uniform. An analysis with different properties for various arterial wall layers may result in other values of $P_{O_2}$ in the wall.

### 5. Nomenclature

- $A_e$ – cross sectional area based on the normal artery mean diameter (mm$^2$);
- $A_m$ – minimum cross sectional area based on the minimum diameter in the plaque (mm$^2$);
- $c$ – oxygen concentration (mlO$_2$/mlblood);
- $C_{\text{hb,sat}}$ – density of saturated hemoglobin;
- $d_e$ – normal artery mean diameter (mm);
- $d_m$ – minimum diameter in the plaque (mm);
- $D_b$ – diffusivity of oxygen in blood (cm$^2$/s);
- $D_w$ – diffusivity of oxygen in the wall region (cm$^2$/s);
$H$ – total oxygen carrying capacity of hemoglobin (ml$_o$/ml$_{blood}$);

$l_c$ – length of the converging section of the plaque (mm);

$l_m$ – length of the throat section of the plaque (mm);

$l_r$ – length of the diverging section of the plaque (mm);

$n$ – Power index;

$p$ – static pressure (dyne/cm$^2$);

$Pe$ – Peclet number;

$P_{O_2}$ – partial pressure of oxygen (mmHg);

$P_{O_2,\text{min}}$ – minimum partial pressure of oxygen in the avascular wall (mmHg);

$P_{O_2,w}$ – partial pressure of oxygen at the lumen–endothelium interface (mmHg);

$P_{O_2,v}$ – partial pressure of oxygen at the outer surface of the arterial wall (mmHg);

$q$ – volumetric consumption rate of oxygen by the cells within the wall region (ml$_o$/ml$_{tissue}$-sec);

$r$ – radial distance (cm);

$Re$ – Reynolds number;

$Sc$ – Schmidt number;

$S$ – saturation function for oxyhemoglobin;

$u$ – axial component of velocity (cm/s);

$v$ – radial component of velocity (cm/s);

$\bar{v}$ – velocity vector (cm/s);

$z$ – axial distance (cm);

$\delta$ – thickness of the avascular wall (micron);

$\rho$ – density of the blood (g/cm$^3$);

$\bar{\tau}$ – stress tensor (dyne/cm$^2$);

$\mu$ – dynamic viscosity (cP);

$\chi$ – oxygen carried by hemoglobin (ml$_o$/ml$_{blood}$);

$\alpha$ – solubility coefficient for oxygen (ml$_o$/ml$_{blood}$-mmHg);

$\phi$ – term introduced to account for the oxygen carried by oxyhemoglobin;

$\dot{\gamma}$ – local shear rate (s$^{-1}$);

$\eta$ – blood viscosity (cP);

$\dot{\gamma}_{ij}$ – rate of strain tensor (s$^{-1}$);

$\lambda$ – time constant (s);

$\eta_0$ – zero shear rate viscosity (cP);

$\eta_\infty$ – infinite shear viscosity (cP);

$\tau$ – local shear stress (dyn/cm$^2$).

Appendix

Calculations of oxygen transport without and with convection due to variable arterial filtration velocity is carried out to determine the validity of the assumption that the convective transport of the oxygen in the wall could be neglected. Here, the baseline case refers to only diffusive transport in the arterial wall and thus, there is no convective transport due to arterial filtration velocity. Figure 10 shows the radial $P_{O_2}$ profiles obtained without and with convective oxygen transport in the arterial wall. The calculations are carried out in an unstenosed artery of radius $r = 0.15$ cm and wall thickness of 300 micron with basal flow rate and wall oxygen consumption of $1.3 \times 10^{-4}$ ml$_o$/ml$_{tissue}$-sec. The profiles are plotted at $z = 6$ cm from inlet.
Fig. 10. Radial $P_{O_2}$ profiles obtained with and without (baseline case) convective oxygen transport in the arterial wall at $z = 6$ cm. Flow rate in the lumen = 50 ml/min. Oxygen consumption in the wall = $1.3 \times 10^{-4}$ mlO$_2$/mltissue-sec. The wall thickness is 300 micron.

Table 1

<table>
<thead>
<tr>
<th>Arterial wall filtration velocity (cm/s)</th>
<th>$P_{O_2, min}$ (mmHg)</th>
<th>% Change in $P_{O_2, min}$ with respect to baseline case</th>
<th>Radial position of $P_{O_2, min}$ from lumen–endothelium interface (micron)</th>
<th>% Change in radial position of $P_{O_2, min}$ with respect to baseline case</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (Baseline case: No convective transport)</td>
<td>5.00</td>
<td>$-%$</td>
<td>164</td>
<td>$-%$</td>
</tr>
<tr>
<td>$4 \times 10^{-6}$</td>
<td>5.16</td>
<td>3.2</td>
<td>164</td>
<td>$-%$</td>
</tr>
<tr>
<td>$4 \times 10^{-5}$</td>
<td>6.62</td>
<td>32.4</td>
<td>164</td>
<td>$-%$</td>
</tr>
<tr>
<td>$4 \times 10^{-4}$</td>
<td>17.57</td>
<td>251.4</td>
<td>191</td>
<td>16.5</td>
</tr>
<tr>
<td>$8 \times 10^{-4}$</td>
<td>27.34</td>
<td>446.8</td>
<td>210</td>
<td>28.1</td>
</tr>
</tbody>
</table>

The filtration velocity in the normal arterial wall is reported to be $\sim 4 \times 10^{-6}$ cm/s. However, in the stenotic region, especially after PTCA procedure, the permeability of the wall could increases by more than 50%. Hence, to determine the effect of increased arterial perfusion on oxygen transport, the filtration velocity is parametrically increased by orders of magnitude. The maximum arterial filtration velocity is considered to be $8 \times 10^{-4}$ cm/s. As expected the magnitude of oxygen concentration in the wall is elevated with the increase in arterial filtration velocity (Fig. 10). For a normal arterial filtration velocity of $\sim 4 \times 10^{-6}$ cm/s, only $\sim 3\%$ change (Table 1) is observed in oxygen concentration when
compared with the baseline case whereas more than 30% change is observed when the filtration velocity is increased by an order of magnitude, i.e., $4 \times 10^{-3}$ cm/s. Further, for the maximum filtration velocity of $8 \times 10^{-4}$ cm/s, which is more than two orders of magnitude higher than the normal arterial filtration velocity, the $P_{O_2, min}$ is increased by $\sim 4.5$ times as compared to the baseline case.

The radial position of $P_{O_2, min}$ shifts further away from the lumen–endothelium interface with increasing arterial filtration velocity. For example, for filtration velocity of $4 \times 10^{-4}$ cm/s the shift is $\sim 17\%$ whereas it is $\sim 28\%$ for filtration velocity of $8 \times 10^{-4}$ cm/s. The shift is insignificant for lower filtration velocities. The increase in the $P_{O_2, min}$ and shift of its locations away from the lumen–endothelium with increasing filtration velocity is due to the increased oxygen flux due to the higher convective transport through the arterial wall.

In order to confirm that the non-Newtonian viscosity of the blood affects the transport of the oxygen to the wall authors calculated the oxygen transport with a constant blood viscosity of 0.0345 g/cm-s. The results were compared with those using Carreau model of blood viscosity with the constants given in the text. Figure 11 shows the comparison of the oxygen flux to the wall with two models of blood viscosity. The length of the recirculation zone for constant blood viscosity is 1.55 cm while that with Carreau model is 1.39 cm. The drop in the oxygen flux to the wall at the location of flow reattachment with constant blood viscosity is 23% of that with Carreau model of blood viscosity. Figure 12 shows the radial $P_{O_2}$ profiles at the location of flow reattachment with constant blood viscosity and Carreau model viscosity. The $P_{O_2, min}$ with constant viscosity is 10.7 mmHg while that with Carreau model is 9.0 mmHg (difference of 18.8%). The $P_{O_2}$ at the endothelium $P_{O_2,w}$ with constant blood viscosity is 77.61 mmHg while that with Carreau model viscosity is 73.37 mmHg (difference of 5.7%). Thus it can be concluded that the effect of non-Newtonian blood viscosity should be included while calculating the oxygen transport to the arterial wall.
Fig. 12. Radial $P_{O_2}$ profile at the location of flow reattachment with Newtonian and non-Newtonian blood viscosity. Flow rate $= 180$ ml/min. Oxygen consumption in the wall $= 1.3 \times 10^{-4}$ mL O$_2$/mL tissue/sec. The wall thickness is 300 micron.

References


