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Effect of porous media of the stenosed artery wall to the coronary physiological diagnostic parameter: A computational fluid dynamic analysis

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ABSTRACT

Functional assessment of a coronary artery stenosis severity is generally assessed by fractional flow reserve (FFR), which is calculated from pressure measurements across the stenosis. The purpose of this study is to investigate the effect of porous media of the stenosed arterial wall on this diagnostic parameter.

To understand the role of porous media on the diagnostic parameter FFR, a 3D computational simulations of the blood flow in rigid and porous stenotic artery wall models are carried out under steady state and transient conditions for three different percentage area stenoses (AS) corresponding to 70% (moderate), 80% (intermediate), and 90% (severe). Blood was modeled as a non Newtonian fluid. The variations of pressure drop across the stenosis and diagnostic parameter were studied in both models.

The FFR decreased in proportion to the increase in the severity of the stenosis. The relationship between the percentage AS and the FFR was non linear and inversely related in both the models. The cut-off value of 0.75 for FFR was observed at 81.89% AS for the rigid artery model whereas 83.61% AS for the porous artery wall model. This study demonstrates that the porous media consideration on the stenotic arterial wall plays a substantial role in defining the cut-off value of FFR. We conclude that the effect of porous media on FFR, could lead to misinterpretation of the functional severity of the stenosis in the region of 81.89 %–83.61% AS.

1. Introduction

A progressive narrowing in the arterial system of human or animal heart is known as stenosis, which impairs blood flow to the heart muscle and eventually results in atherosclerotic plaque rupture and life threatening myocardial infarction [1]. Assessment of physiological severity of an intermediate stenosis in a single vessel or branched vessel using usual coronary angiogram or multi slice computed tomography is more complex [2,3]. The true functional severity of coronary artery stenosis is assessed by pressure drop and flow [4–6]. Coronary flow reserve index [6] (CFR; ratio of hyperemic flow to the flow at resting conditions) and fractional flow reserve index [7] (FFR; ratio of distal coronary pressure to aorta pressure under hyperemic condition) are the two parameters that provide physiological information about the severity of the coronary artery stenosis so that appropriate therapy can be initiated to the patient during cardiovascular intervention. Among them, the FFR is currently used as a gold standard for the assessment of functional significance of stenosis severity. The FFR has a value of 1 for every patient, every artery and every segment under no stenotic condition and is highly reproducible [8]. The FFR is easy to measure during coronary intervention and well validated [9]. A 0.014 inch diameter guide wire with the pressure sensor at its tip is advanced across the stenosis to measure distal pressure under hyperemic conditions, which is induced by a vasodilator agent, adenosine or papaverine to measure FFR [2]. It is linearly related to the maximum achievable blood flow [7]. In the presence of stenosis, a cut-off value of FFR <0.75 was almost able to induce myocardial ischemia, whereas FFR >0.8 was never associated with exercise induced ischemia [10,11]. There is a well recognized uncertainty region for FFR lies between 0.75 and 0.8.

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The uncertainty region for the FFR is of great interest, where misinterpretation of physiological significance of stenosis severity is a possibility. Many studies have been performed in the uncertainty region during the past years. Insertion of sensor tipped guide wire [12], downstream collateral flow [13] and by other factors such as micro vascular resistance, aortic and coronary outflow pressure [14], arterial wall compliance and plaque characteristics [15] have been significantly affecting the FFR value.

One of the major limitations of the well published studies is that the coronary artery wall is impervious to blood. From the literature, it is clear that all the human tissues are porous in nature [16–19] and the plaque region mainly includes a large lipid core and a thin fibrous cap [20]. The normal arterial wall consists of endothelium, intima, internal elastic lamina (IEL), media and adventitia [21]. It is believed that the permeability of endothelium wall increases with deposition of cholesterol due to the damaged or inflamed arterial wall.

Blood is a moving column with suspended cells. Normal endothelium did not allow passage of cells. However, it has been shown that atherosclerotic endothelium is highly permeable to white cells and platelets in the event of plaque rupture. Recells are also shown to enter to tunica media [22,23].

It is useful to study the pulsatile non-Newtonian blood flow through stenotic arteries taking into account of blood transport through the porous arterial wall, and to investigate the effect of porous media on the diagnostic parameter, FFR. The FFR does not contain any empirical relations, but it is a ratio which simply depends on measurable quantities of distal pressure and proximal pressure. Pressure drop-flow (Δp–Q) relation and FFR in the stenosis region have been studied by many researchers by considering the arterial wall as rigid [24–26]. However, variations in the values of FFR due to porous stenotic arterial wall have not been previously studied. In this study, a 3D computational rigid stenosed artery (RA) and porous stenosed artery (Fluid Porous Interface–FPI) models have been considered. For the given percentage area stenosis, we examine the pressure drop across the stenosis and estimate the value of FFR in both models for the possible misdiagnosis region.

2. Mathematical modeling

In this study, stenosis geometry was considered as trapezoidal as shown in Fig. 1. Lesion dimensions (Table 1) are taken from Konala et al. [15] and Rajabi et al. [25]. Stenosis regions consist of converging (of length l_c), throat (of radius r_m and length l_m) and diverging (of length l_r) sections. Moreover, proximal and distal radius are assumed to be identical ($r_e = r_d$). Three stenotic models–70% (moderate), 80% (intermediate) and 90% (severe) AS have been considered here for both RA and FPI models. The computational domain, a 3-D computational coronary artery models for simulation are as shown in Fig. 2(a) & (b).

Table 1
Artery wall and stenosis geometry. All dimensions are in mm.

Area stenosis (AS) (%)	t_a	$r_e = r_d$	r_m	l_c	l_m	l_r
70	1	1.5	0.82	6	3	1.5
80	1	1.5	0.67	6	1.5	1.5
90	1	1.5	0.47	6	0.75	1.5

Area stenosis (AS) % = Area of the blockage due to stenosis/Area of the lumen free from stenosis) $= ((\pi \times r_e^2) - (\pi \times r_m^2))/\pi \times r_e^2$.

3. Computational blood flow model

It will be assumed that the flow of blood is incompressible and governed by the Navier–Stokes equations.

$$\rho\left(\frac{\partial v}{\partial t} + v\cdot\nabla v\right) = -\nabla\cdot\tau - \nabla p \tag{1}$$

and the continuity equation for incompressible flow is

$$\nabla\cdot v = 0 \tag{2}$$

here v is the three dimensional velocity vector, t the time, ρ the blood density, p the pressure and τ the stress tensor. In this study, a non-Newtonian blood is assumed to follow the Bird-Carreau model [27] and the blood viscosity μ given in poise (P) as a function of shear rate $\dot{\gamma}$ given in s^{-1} is given by

$$\mu = \mu_\infty + (\mu_0 - \mu_\infty)\left[1 + (\lambda\dot{\gamma})^2\right]^{(n-1)/2} \tag{3}$$

where, λ (Time constant) = 3.313 s, n (Power law index) = 0.3568, μ_0(Low shear viscosity) = 0.56 P, μ_∞(High shear viscosity) = 0.0345 P. The density of the blood (ρ) is assumed as1050 kg/m^3.

4. Arterial wall

Fluid flow was modeled through a homogeneous porous stenotic artery wall. In this work, we use Darcy-Forchheimer model, which accounts for the inertial effects. The governing momentum loss equations can be written as [17]

$$\nabla p = -\frac{\mu}{K}V + c_F K^{-\frac{1}{2}}\rho|V|V \tag{4}$$

where ∇p is the pressure gradient, μ is fluid kinematic viscosity, K is the permeability of the wall, V is the superficial velocity vector, c_F is a dimensionless parameter related to inertial effects and ρ is a fluid density.

In our study, we set the thickness of the arterial wall which is represented by $t_a = 1$ mm [15], $K = 2.0 \times 10^{-14}$ cm^2 [16], porosity $\varepsilon = 0.15$ [16] and $c_F = -1.75/\sqrt{150 \times \varepsilon}$ [28].

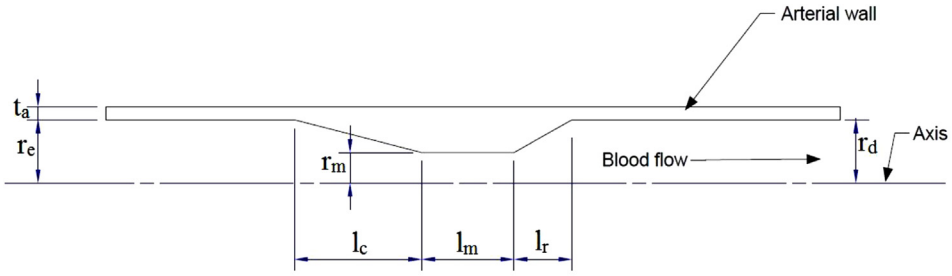

Fig. 1. Schematic diagram showing vivo lesion geometry. (Dimensional values are given in Table 1).