



# Numerical investigation of blood flow in three-dimensional porcine left anterior descending artery with various stenoses



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## ARTICLE INFO

### Article history:

Received 20 August 2013

Accepted 4 January 2014

### Keywords:

CFD

Coronary stenosis

Flow rate distribution

Flow-dependent boundary condition

Wall shear stress

## ABSTRACT

Coronary heart disease causes obstruction of coronary blood flow and is the leading cause of death worldwide. The effect of focal stenosis on downstream flow pattern in the coronary arterial tree is not well understood. Here, the blood flows in normal and diseased porcine left anterior descending (LAD) arterial tree were modeled and compared to determine the effects of stenosis on the blood flow distribution and hemodynamic parameters. The anatomical model of LAD was extracted from a porcine heart by computed tomography (CT), which was comprised of a main trunk and nine side branches. Stenoses with various severities were imposed into the main trunk between the first and second side branches, and the boundary condition at each outlet accounted for the effect of stenosis on the flow rate in the downstream vasculature. It was found that only significant stenosis ( $\geq 75\%$  area reduction) considerably altered pressure drop and total flow rate distribution in branches and at each bifurcation. The effect of significant stenosis on bifurcations, however, diminished at downstream locations. As demonstrated by distributions of oscillatory shear index and relative residence time, non-significant stenosis ( $< 75\%$  area reduction) has the potential to induce atherosclerosis near the ostium of downstream side branch, while significant stenosis can promote atherosclerosis in its wake.

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## 1. Introduction

Coronary heart disease (CHD) is one of the leading causes of death worldwide, which is typically caused by formation of plaques on a coronary arterial wall. Sudden plaque rupture may lead to an acute myocardial infarction or even sudden death [1]. The relevant retrospective pathologic studies show that a plaque prone to rupture is mostly an inflamed thin-cap fibroatheroma [2]. The stable plaque growth typically stiffens the host artery and narrows the lumen diameter (stenosis), leading to stable angina pectoris [2]. Stenosis is considered to be clinically significant when the area reduction is more than 75%, at which the supply of oxygenated blood to myocardium is inadequate [3–5]. Besides lifestyle changing and therapeutic medications (e.g. aspirin), percutaneous coronary intervention is a common non-surgical procedure to treat coronary artery stenosis, which can be an alternative to conventional coronary artery bypass grafting [6].

It has been found that the development of CHD is related to hemodynamic parameters [7–9]. Adverse parameters are usually observed at arterial bifurcations and curvature, where flow separation or flow reversal occurs [10,11].

Due to the difficulty of measuring hemodynamic parameters directly in coronary arteries, computational fluid dynamics (CFD) has been widely adopted, and the predicted hemodynamic parameters can assist diagnosis and prognosis [8,11–17]. Typically, only a segment or single bifurcation is considered, while the remaining arteries are represented by the boundary conditions. However, pressure or velocity waveforms at outlets of coronary artery are usually unavailable. A common method is to calculate the percent-distribution of the total flow rate among branches based on Murray's law [18], which correlates lumen diameter to flow rate. This method is only limited to studies in normal arteries. Groen et al. [19] compared outflow ratios of stenotic carotid arteries predicted by Murray's law with measured values and found that Murray's law was invalid for the area stenosis larger than 65%. A more sophisticated method is to represent downstream vasculature by a flow-dependent formulation; i.e. the static pressure at an outlet is calculated from the corresponding flow rate [20–25].

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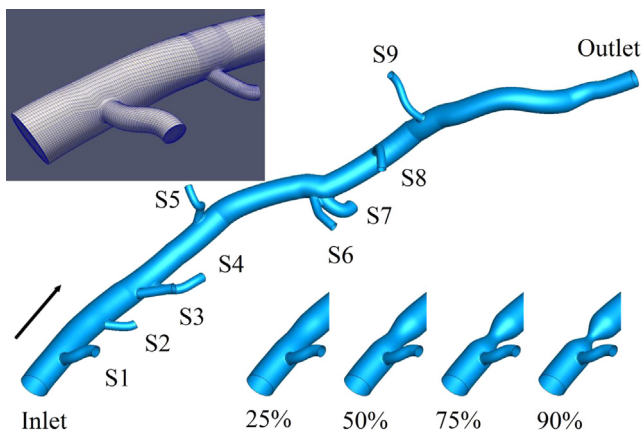
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Although extensive simulations on coronary arteries have been conducted during past decades, the studies on comparisons of left anterior descending (LAD) arterial trees without and with various degrees of stenosis are still limited. Shanmugavelayudam et al. [26] developed both 2D and 3D models of normal and simplified diseased LAD without side branch. They concluded that 2D model was incompetent to predict similar hemodynamic parameters in diseased LAD. Katritsis et al. [22] studied LAD models with different degrees of stenosis using the interpolated pressure boundary conditions, in order to correlate vortices and flow recirculation with thrombus formation. Swillen et al. [14] only modeled a LAD with 75% and 90% stenoses, since their main purpose was to estimate the effect of LAD stenosis on the flow in left internal mammary artery to LAD bypass surgery. Kim et al. [24] simulated patient-specific coronary arteries with sophisticated boundary conditions to examine the effect of stenosis on the total flow rate in LAD artery. In this study, a porcine epicardial LAD arterial tree model under both pulsatile and steady flows were modeled with different degrees of stenosis (0%, 25%, 50%, 75% and 90% area reduction) imposed into the main trunk between first and second side branches. In this study, we aimed to investigate: (i) differences between pulsatile and steady flow simulations; (ii) effects of stenosis on the hemodynamic parameters in the porcine LAD model.

**2. Materials and methods**

The preparation of porcine heart and the procedure of computed tomography scan were reported in our previous study [17]. Using centerline and lumen diameter at each segment extracted from CT images, the LAD arteries were reconstructed using computer-aided design (CAD) software as illustrated in Fig. 1. Table 1 shows the lumen diameters of trunk and side branches. To investigate effects of stenosis, the trunk segment between S1 and S2 was narrowed by 25%, 50%, 75% and 90% in area with respect to the host artery. The stenoses imposed were all concentric and of the same length (4 mm).

The reconstructed CAD geometries were meshed with structured grids as illustrated in Fig. 1. It is worth noting that the same topology was applied to each model so that the mesh quality was identical to each numerical model except the stenosis region. To ensure mesh quality, skewness angles were between 30° and 150°. In order to minimize the computational time, the normal model under steady-state was selected for the grid independence test. Models with  $5 \times 10^5$ ,  $8 \times 10^5$ ,  $11 \times 10^5$ , and  $1.4 \times 10^6$  grid cells were simulated, and the one with  $8 \times 10^5$  elements was chosen based



**Fig. 1.** Reconstructed CAD model of porcine LAD arterial tree with nine side branches and structured mesh.

**Table 1**

Percent-distribution of total flow rate among branches in the normal LAD model defined by scaling law, values calculated from steady and pulsatile flows and back pressure at side branch outlet.

LAD	Diameter (mm)	Scaling (%)	Steady (%)	Unsteady (%)	Back pressure (Pa)
Inlet	3.3	100	100	100	–
S1	1.3	13.0	13.0	13.2	6504
S2	1.0	6.0	6.1	6.1	6505
S3	1.1	8.0	7.9	7.9	6475
S4	1.1	8.0	8.0	8.0	6493
S5	0.9	5.0	5.0	5.0	6484
S6	1.0	6.0	5.9	5.9	6479
S7	1.6	16.0	16.2	16.5	6508
S8	1.0	4.0	4.0	4.0	6464
S9	0.9	3.0	3.0	3.0	6464
Outlet	2.0	31.0	30.9	30.5	6481

on the criterion that the relative difference of the maximum velocity with respect to the one with  $1.4 \times 10^6$  elements was less than 0.5%.

The porcine blood was assumed to be Newtonian and incompressible with constant dynamic viscosity of 4 cP and density of  $1235 \text{ kg/m}^3$  [17]. The 3D blood flow in LAD is governed by the Navier–Stokes equations and continuity equation written as

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

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

where  $\rho$  is the fluid density;  $v$  is the velocity vector;  $t$  is the time;  $P$  is the pressure; and  $\tau$  is the stress tensor. The commercial CFD solver, Fluent (Version 6.3.26), was adopted for the numerical simulations.

The LAD models were assumed to be stationary and rigid with no-slip boundary condition applied to walls. In the normal LAD model, the scaling law of coronary vascular tree proposed by Kassab [27] was implemented. The scaling exponent of porcine LAD was taken as 2.18, based on which the percent-distribution of the total flow rate among nine side branches was calculated as illustrated in Table 1.

In order to obtain the back pressure and flow resistance at each side branch, a number of steady-state simulations were conducted with the same outflow ratios (Table 1) but different inlet flow rates. For steady-state simulations, only flow resistance played a role in the flow rate distributions, leading to a linear relationship between pressure and flow rate. For each outlet, a linear trend line was plotted using the flow rates and pressures obtained from these steady-state simulations, and the flow behavior of the corresponding downstream vasculature was described as

$$P_{outlet}^i = P_{back}^i + R^i Q_{outlet}^i \tag{3}$$

where  $P_{outlet}^i$  is the outlet pressure;  $R^i$  is the flow resistance of downstream vasculature;  $Q_{outlet}^i$  is the flow rate; and  $P_{back}^i$  is the back pressure at  $i$ th outlet. Eq. (3) was implemented by user define function (UDF), which was compiled and loaded into the CFD solver. As a result, the downstream vasculature was coupled to the LAD model, and the static pressure at each outlet was solved iteratively. In this study, the implicit algorithm was applied with the under-relaxation scheme written as

$$P_{outlet}^{i,n+1,k+1} = P_{outlet}^{i,n+1,k} + \omega^{n+1,k} (R_{outlet}^i Q_{outlet}^{i,n+1,k+1} + P_{back}^i - P_{outlet}^{i,n+1,k}) \tag{4}$$

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