A hemodynamic model with a seepage condition and fluid–structure interactions for blood flow in arteries with symmetric stenosis

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Received: 21 September 2018 / Accepted: 25 March 2019 / Published online: 6 May 2019
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Abstract

To strengthen the detailed understanding of arterial stenosis, we construct a novel hemodynamic model. Frequently used symmetric stenosis is employed in this work. Being different from a traditional model, this numerical model adopts microcirculation resistance as an outlet boundary condition, which is called a seepage condition. Meanwhile, fluid–structure interactions are used in the numerical simulation considering the interrelationship of blood and arterial wall. Our results indicate that (i) the region upstream of stenosis experiences very high pressures during cardiac cycles, (ii) pressure drops much faster as the flow moves into the stenotic region, and (iii) high flow velocities and high shear stresses occur in the post-stenosis region. This work provides evidence that there is a strong effect of the function of microcirculation on stenosis. This contributes to evaluating potential stenotic behavior in arteries and is pivotal in guiding disease treatment.

Keywords Stenosis · Seepage · Artery · Hemodynamics · Fluid–structure interaction

1 Introduction

The understanding of physiologically realistic pulsatile flow through stenoses has profound implications for the diagnosis and treatment of vascular diseases. Arterial stenosis is one of the leading causes of death. The reason is that stenosis is usually accompanied by thrombus formation, atherosclerosis growth, and plaque cap rupture that leads directly to stroke and heart attack [1, 2]. Great efforts have been made by clinicians, mathematicians, and mechanics

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researchers towards stenotic arteries. In particular, computational models of stenotic flow have been developed through advanced computers and numerical methods in recent years. Considerable work has focused on the effects of stenosis severities on flow [3–9]. Many researches have been mainly performed using a traditional approach based on the specification of a constant or time-dependent pressure at an outlet. In previous work, we discussed the limitation of the traditional approach in numerical simulations of arterial flow and explained the role of microcirculation and the necessity of introducing it [10]. Namely, microcirculation is located at the outlet of arteries and provides a complicated outlet condition beyond a specific pressure profile. It is important to understand how the flow and microcirculation affect each other and how microcirculation changes with the development of stenosis.

Due to the high complexity of the mechanical properties of the arterial wall and the limitation of experimental and computational methods, the exact mechanism of the formation and development of stenosis is still not well understood. In this paper, we introduce a symmetric model with a seepage condition and fluid–structure interactions for pulsatile blood flow through a compliant stenotic artery and investigate the unsteady flow. Here, the seepage condition denotes that the resistance caused by the seepage flow in microcirculation is specified as a boundary condition based on a seepage theory. Though the acquisition of large and complex microvascular anatomy is onerous, a porous medium may replicate downstream capillary structures, which can capture the essential features of the seepage flow in microcirculation. A porous model has been widely and successfully used in simulating the flow in microcirculation [11–13]. Fluid–structure interaction is two-way in that the fluid affects the wall and the wall in turn affects the flow [14]. It is outlined that changes to vascular pressure induce a compliant response in the vessels as they cyclically stretch and relax and then the compliance influences the fluid flow throughout the system during the cardiac cycle [15]. The main purpose of this study is to improve our understanding of the mechanism. Compared with a traditional approach, the combination of a seepage boundary condition and fluid–structure interactions is closer to the nature of arterial flow and it may contribute to discovering the reality of stenosis.

2 Model and methods

The geometrical model was constructed as shown in Fig. 1, where D is the diameter of the tube. In this study, we specify $D = 20$ mm. Microcirculation is represented by the domain for the outlet length. Due to the tiny deformation of the vessel in microcirculation and arterial compliance, the wall in microcirculation is assumed to be rigid, and blood–wall interactions in the artery zone are employed. The wall thickness is set to 2 mm for the inlet length. Stenosis severity is commonly defined as [16].

$$S_0 = \frac{R_0 - R_{\min}}{R_0} \times 100\% \quad (1)$$

where R_0 is the radius of the uniform part of the tube and $S_0 = 50\%$.

For the numerical simulation of interacting pulsatile blood flow and arterial wall motion, partial differential equations plus associated boundary conditions have to be solved. Navier–Stokes equations are used as the governing equations:

Fig. 1 Geometrical model

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + ((\mathbf{u} - \mathbf{u}_m) \cdot \nabla) \mathbf{u} \right) = -\nabla p + \nabla \cdot \mathbf{T} \quad (2)$$

$$\nabla \cdot \mathbf{u} = 0 \quad (3)$$

where $\mathbf{u}$ is the fluid velocity vector, $\mathbf{u}_m$ is the fluid mesh velocity, p is the fluid pressure, $\partial/\partial t$ derivative with mesh points fixed, ρ is the fluid density, and $\mathbf{T}$ is the stress tensor. The fluid is modeled to have a density $\rho = 1050 \text{ kg/m}^3$.

$$\mathbf{T} = 2\eta(\dot{\gamma})\mathbf{D} \quad (4)$$

$$\mathbf{D} = \frac{1}{2}(\nabla \mathbf{u} + \nabla \mathbf{u}^T) \quad (5)$$

where η represents the viscosity of the blood and $\dot{\gamma}$ is the shear rate. For a non-Newtonian fluid, η is a function of $\dot{\gamma}$, while for a Newtonian fluid, η is a constant and independent of $\dot{\gamma}$.

Since blood demonstrates non-Newtonian behavior, blood flow in the fluid domain is modeled to be non-Newtonian. In the present study, the Carreau–Yasuda shear thinning model [17] was used:

$$\frac{\eta - \eta_\infty}{\eta_0 - \eta_\infty} = [1 + (\lambda \dot{\gamma})^a]^{(n-1)/a} \quad (6)$$

The parameters in Eq. (6) are written as $\eta_\infty = 2.2 \times 10^{-3} \text{ Pa}\cdot\text{s}$, $\eta_0 = 22 \times 10^{-3} \text{ Pa}\cdot\text{s}$, $\lambda = 0.110 \text{ s}$, $a = 0.644$, $n = 0.392$ [18].

Usually, microcirculation is considered as a porous medium. In this study, we set the porosity of the microcirculation to $\phi = 0.5$ [19]. The following empirical equation is adopted to compute the permeability, which is determined by the porosity ϕ :

$$k = \frac{d^2 \phi^3}{180(1-\phi)^2} \quad (7)$$

where d is the diameter of the microvessels in microcirculation; here, $d = 8 \text{ }\mu\text{m}$.

To simplify the calculation, we assume that the arterial wall is an isotropic, incompressible and linear-elastic material. We address it using a simple model because we only want to find a methodology of the seepage condition application for numerical simulation. The methodology

may be used for a more complex model in the future. Thus, the linear-elastic constitutive equations are used as the governing equations:

$$\rho_w \frac{\partial^2 d_i}{\partial t^2} = \frac{\partial \sigma_{ij}}{\partial x_j} + F_i \quad (8)$$

where d_i and σ_{ij} are the components of the displacement and stress tensor, ρ_w is the material density of the wall, and F_i is the component of the body force acting on the solid:

$$\sigma = D\varepsilon \quad (9)$$

where D is the matrix of elastic constants, which are determined from the Young's modulus and Poisson's ratio.

In this simulation, the arterial wall is modeled to have a density $\rho_w = 1120 \text{ kg/m}^3$ and a Young's modulus $E = 5 \text{ MPa}$ [20]. The arterial wall is nearly incompressible, with a Poisson's ratio of 0.499.

A time-dependent velocity profile (Fig. 2) is assigned at the inlet. At the same time, a seepage outlet boundary condition is specified. The period of the flow waveform is 0.8 s. The initial conditions in the whole field are assumed to be zero [21, 22].

We set that the ends of the blood vessel are fixed in all degrees of freedom. No-slip boundary conditions are imposed at the tube wall.

$$(u, v, w)|_{\Omega} = \frac{\partial \chi}{\partial t} \quad (10)$$

Here, the subscript Ω stands for the tube wall and $\chi = (r, \theta, z)$ are the position vectors of the deformed tube wall.

The structural problem and the transient behavior of the fluid domain are solved by ANSYS, a finite-element-based program. The finite element method changes partial differential equations to a system of algebraic equations, which yield approximate values of the unknowns at a discrete number of points over the domain. To solve the problem, it subdivides a large system into smaller, simpler parts that are called finite elements. The simple equations

Fig. 2 Inlet velocity

that model these finite elements are then assembled into a larger system of equations that model the entire problem. During the calculation, corresponding physical quantities, such as fluid forces, solid displacements, and velocities, transfer across fluid–structure interfaces until a convergence criterion (10^{-4}) is reached for each time step. A constant time step of 0.01 s is used in this study. The fluid equations are spatially discretized using a high-resolution advection scheme. The transient discretization scheme is second-order backward difference. The arbitrary Lagrangian–Eulerian algorithm provides an effective way in sequentially solving the equations that treat the fluid problem, the structural problem and the remeshing problem by a staggered approach. The proper mesh is obtained when the results become independent of mesh density and are used to produce numerically accurate results. Three cardiac cycles are required because it is found that the solution attains the periodic steady state after two cycles of calculations. The third cycle is extracted and used for the proceeding analysis.

3 Results

3.1 Maximum pressures at different sections

The two pressure profiles at sections S1 and S2 are almost identical. However, the pressures at section S1 are much higher than those at section S2, as shown in Fig. 3. The maximum pressure at section S1 is about 2000 Pa while that at section S2 is only approximately 50 Pa. A large pressure drop occurs across the stenosis. In addition, Fig. 3 also shows that the fluctuation range of the pressures progressively falls in the artery zone. Figure 4 depicts the pressure distributions at sections S3 and S4 in the microcirculation zone. Figure 4 shows that there are negative pressures at sections S3 and S4. Furthermore, the absolute values of the pressures at section S4 are higher than those at section S3. The fluctuation range of the pressures progressively rises in the microcirculation zone. According to the results in our previous paper [10], the maximum pressure at section S1 in a normal artery is only 800 Pa. Thus, the maximum pressure at section S1 in the stenotic artery is significantly higher. The change caused by the stenosis suggests that high pressure is closely related with a diseased condition.

Fig. 3 Maximum pressures in the artery zone

Fig. 4 Maximum pressures in the microcirculation zone

3.2 Maximum velocities at different sections

Figure 5 depicts that the velocities at section S2 are higher than those at section S1. It is known that sections S1 and S2 are respectively located at the pre- and post-stenosis regions. Thus, it shows that high flow velocities occur at the downstream of the stenosis. The time-dependent velocities in the artery and microcirculation zones resemble the profile of the inlet velocity. However, the velocities in the microcirculation zone are much lower, as shown in Fig. 6. Due to the extreme low values, the flow in microcirculation is deemed to be steady even if the velocities are time dependent. The change of pulsatile flow to steady flow is completed in capillaries by repelling the pulsations that enter from the larger arteries. Clearly, the flow resistance is increased by the stenosis so that high flow velocities are induced to maintain the necessary blood supply.

3.3 Maximum wall shear stresses at different sections

The maximum wall shear stresses at sections S1 and S2 are almost the same (Fig. 7). The post-stenosis region experiences very high wall shear stresses. Undoubtedly, stenosis leads to the characteristic changes. It is evident that high wall shear stress induces plaque cap rupture. The wall shear stress distributions and their variation trends, as shown in Figs. 7 and 8, are similar to those of the velocities in Figs. 5 and 6. As a bad response, high wall shear stresses caused by the stenosis are not suitable for maintaining the structure of the arterial wall. It may be harmful to the arterial structure. Furthermore, the damaged structure promotes wall shear stress again. Thus, the formation of a terrible circulation between wall shear stress and arterial structure may occur.

4 Discussion

The fluctuation range of blood pressures gradually increases in a normal artery, which is a physiological fact [20, 23]. However, according to the above results, the pressure

Fig. 5 Maximum velocities in the artery zone

distributions in the stenotic artery are greatly different. It is seen that the fluctuation range of the pressures in the stenotic artery progressively falls, contrary to a normal artery where the fluctuation range rises gradually. Evidently, the results violate the physiological fact. The results indicate that microcirculation in the stenotic artery seems to lose an important role in regulating blood pressures, unlike in a normal artery. These microvessels in a normal arterial circulation usually present the greatest resistance to blood flow so that the pressure wave propagation is impeded by micro-arteries and then the reflection is produced. Thus, pulse pressure arises as a consequence of the properties in the arterial circulation. However, it seems that the resistance provided by the stenotic arterial circulation disappears. If the role of microcirculation is assimilated to a sluice gate, the gate is completely open under the diseased condition. According to the logic

Fig. 6 Maximum velocities in the microcirculation zone

Fig. 7 Maximum wall shear stresses in the artery zone

relationship, it is speculated that functional loss of microcirculation will cause stenosis. In addition, the high pressures caused by the stenosis are obviously a disadvantage to arterial tissue. It is destined that hypertension leads to relevant diseases [24]. By this token, it is known that microcirculation significantly affects the function of arterial circulation. In other words, the occurrence of arterial diseases is considerably controlled by microcirculation. The entire negative pressures as shown in Fig. 4 hinder the process of microcirculation and then damage the progress of arterial circulation.

Both the high pressure and the narrowing of blood vessels cause high flow velocity, high shear stress, and low or negative pressure at the throat of the stenosis [1]. Stenosis may create significant flow resistance and a large pressure drop [5, 25]. Our results are found to be in excellent agreement with these facts.

Fig. 8 Maximum wall shear stresses in the microcirculation zone

5 Conclusions

This work deals with a hemodynamic model with a seepage condition and fluid–structure interactions for blood flow in arteries with symmetric stenosis. Flow analysis indicates that (i) the upstream of stenosis experiences very high pressures during the cardiac cycles, (ii) pressure drops much faster as the flow moves into the stenotic region, and (iii) high flow velocities and high shear stresses occur in the post-stenosis region. In summary, such a novel model can provide detailed flow information of blood. The analysis in this study contributes to understanding the relationship between stenosis and the function of microcirculation and the quantitative information provided can inform decision-making regarding stenosis treatment.

Acknowledgements We thank the National Natural Science Foundation of China (81401492) and the Science and Technology Project of Beijing Municipal Commission of Education (KM201510016012) for financially supporting this research. The work is also supported by the Foundation of Research and Innovation Team (21147515602) and the Academic Innovation Team of Beijing University of Civil Engineering and Architecture (X18080).

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

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