Spatial velocity distributions in pulse-wave propagation based on fluid–structure interaction

Fan He · Lu Hua · Li-jian Gao

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Abstract In this paper, spatial velocity distributions in pulse-wave propagation based on a fluid–structure interaction model are presented. The investigation is performed using the assumption of laminar flow and a linear-elastic wall. The fluid–structure interaction scheme is constructed using the finite element method. The results show that velocity distributions embody an obvious time delay in an elastic tube model. Further, the fully developed flow is delayed and the velocity values are increased in comparison with a rigid tube model. The increase in the wall thickness makes the time delay between the velocity peaks of different sites smaller while the time delay between the velocity minima is unchanged. Similarly, the time delay between the velocity minima is more easily found when decreasing the internal radius. The model gives valid results for spatial velocity distributions, which provide important information for wave propagation.

Keywords Pulse-wave propagation · Flow distribution · Fluid–structure interaction · Finite element method

1 Introduction

Wave propagation is an important aspect of arterial blood flow. The changes of pulse-wave velocity are regarded as signals of disease and these can be measured by medical devices. The complex pattern of wave propagation in large arteries may be the most important determinant of arterial hemodynamics. Understanding wave phenomena and developing the proper tools for analyzing pressure and flow waves can help researchers and clinicians

F. He (✉)

Department of Mechanics, School of Science, Beijing University of Civil Engineering and Architecture, Beijing, 100044, China
e-mail: hefan@bucea.edu.cn

L. Hua · L. Gao

Key Laboratory of Clinical Trial Research in Cardiovascular Drugs, Ministry of Health, Fuwai Hospital, National Center for Cardiovascular Diseases, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, 100037, China

obtain deeper insight into the functioning of the cardiovascular system and eventually develop better diagnostic tools and possibly better therapies [1]. Wave behavior in arteries has been studied using different methods such as impedance [2], characteristics methods [3], and so on. The constructed models using these methods belong to one-dimensional (1D) models. Although 1D models can provide clinically relevant information, they cannot yield characteristics, such as the wall shear stress field or the vorticity field [4]. Undoubtedly, 3D models have more advantages in this respect. Recently, we developed an axisymmetrical model to investigate wave propagation using fluid–structure interaction. Our final aim is to construct a 3D model. Wave propagation and wall shear stress (WSS) distributions have been discussed in our prior papers. The focus of this paper is to present spatial velocity distributions in wave propagation. These distributions can provide detailed knowledge of flow pattern in wave propagation. A thorough understanding of blood dynamics in human vessels is of great interest, since the local flow behavior of blood is certainly implicated in the formation of atherosclerotic plaques and of phenomena such as thrombogenesis, atherogenesis, endothelial damage, intimal thickening, and hyperplasia [5]. Local hemodynamic temporal and spatial flow patterns are important for understanding the mechanisms leading to various complications in cardiovascular function.

As we know, local flow-field details can only be obtained from 3D models. Local blood-flow characteristics play a determinant role in the development and progression of vascular diseases [6–8]. In this paper, we present spatial velocity distributions in wave propagation based on a fluid–structure interaction model to better understand and analyze local biological and physiological processes when waves propagate.

2 Methods

2.1 Computational model

A three-segment model was designed to investigate wave propagation. The model geometry was a straight tube of diameter 20 mm. In the model, the wall whose thickness was 2 mm in the middle was elastic while the walls at the two ends were rigid (Fig. 1). The length of each segment was 300 mm. The effects of vessel length on wave propagation and the entrance effects were avoided. In Fig. 1, S1, S2, and S3 denote the different sites away from the inlet, whose distances are 100, 500, and 800 mm, respectively.

The proposed model in Fig. 1 is a baseline model when performing numerical comparisons, which is called the elastic tube model. The rigid tube model is obtained when the rigid tube takes the place of the elastic tube in the middle.

2.2 Governing equations

The viscous flow was assumed to be laminar, Newtonian, and incompressible. For the fluid model, the governing equations were the Navier–Stokes equations:

$$\rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial r} + \frac{v}{r} \frac{\partial u}{\partial \theta} - \frac{v^2}{r} + w \frac{\partial u}{\partial z} \right) = -\frac{\partial p}{\partial r} + \mu \left(\frac{\partial^2 u}{\partial r^2} + \frac{1}{r^2} \frac{\partial^2 u}{\partial \theta^2} + \frac{\partial^2 u}{\partial z^2} + \frac{1}{r} \frac{\partial u}{\partial r} - \frac{u}{r^2} - \frac{2}{r^2} \frac{\partial v}{\partial \theta} \right) \quad (1)$$

$$\rho \left(\frac{\partial v}{\partial t} + u \frac{\partial v}{\partial r} + \frac{v}{r} \frac{\partial v}{\partial \theta} + \frac{uv}{r} + w \frac{\partial v}{\partial z} \right) = -\frac{1}{r} \frac{\partial p}{\partial \theta} + \mu \left(\frac{\partial^2 v}{\partial r^2} + \frac{1}{r^2} \frac{\partial^2 v}{\partial \theta^2} + \frac{\partial^2 v}{\partial z^2} + \frac{1}{r} \frac{\partial v}{\partial r} - \frac{v}{r^2} + \frac{2}{r^2} \frac{\partial u}{\partial \theta} \right) \quad (2)$$

Fig. 1 Model geometry

$$\rho \left(\frac{\partial w}{\partial t} + u \frac{\partial w}{\partial r} + \frac{v}{r} \frac{\partial w}{\partial \theta} + w \frac{\partial w}{\partial z} \right) = -\frac{\partial p}{\partial z} + \mu \left(\frac{\partial^2 w}{\partial r^2} + \frac{1}{r^2} \frac{\partial^2 w}{\partial \theta^2} + \frac{\partial^2 w}{\partial z^2} + \frac{1}{r} \frac{\partial w}{\partial r} \right) \quad (3)$$

$$\frac{\partial u}{\partial r} + \frac{1}{r} \frac{\partial v}{\partial \theta} + \frac{\partial w}{\partial z} + \frac{u}{r} = 0 \quad (4)$$

where u , v , w are the radial, circumferential, and axial velocity components, respectively. The fluid was modeled to have a density $\rho = 1,050 \text{ kg/m}^3$ and a dynamic viscosity $\mu = 0.0035 \text{ Pa}\cdot\text{s}$.

At the inlet, we set a time-dependent velocity profile, as shown in Fig. 2. The period of the flow waveform was 0.8 s. Then, the average Reynolds number varied by about 2100, while based on the period of the flow pulse, and the Womersley number was equal to 15.3. A zero normal traction was applied at the outlet. The initial conditions in the whole field were assumed to be zero [9, 10].

For computational convenience, the wall was assumed to be linear-elastic, isotropic, and incompressible. Thus, for the wall model, the governing equation was as follows:

$$\varepsilon_{ij} = \frac{1+\nu}{E} \sigma_{ij} - \frac{\nu}{E} \sigma_{kk} \delta_{ij}. \quad (5)$$

The tube wall was modeled to have a Young's modulus $E = 5 \text{ MPa}$ [11] and a Poisson's ratio $\nu = 0.499$.

Fig. 2 The velocity at the inlet

We set that one end of the blood vessel was entirely fixed and the other end was free. For the boundary conditions at the tube wall, we assume that no slipping takes place and that the fluid and wall move together:

$$(u, v, w)|_{\Omega} = \frac{\partial \chi}{\partial t}. \quad (6)$$

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

2.3 Numerical method

The fluid–structure interaction scheme was constructed using the finite element method [12]. The finite element formulation based on the Galerkin method was employed. It was an implicit procedure in time. The arbitrary Lagrangian–Eulerian (ALE) formulation was used. The unstructured meshes were composed of hexahedral eight-node elements in the wall domain and of tetrahedral four-node elements in the fluid domain. There were 9,000 elements and 12,240 nodes in the wall domain and 59,370 elements and 12,953 nodes in the fluid domain. The grid independence of the solution scheme was tested and assessed. The results showed that a further increase of the mesh size did not significantly change the final results.

We calculated three cycles with a time interval of 0.01 s to obtain convergent and stable solutions [13, 14]. The convergence was assumed to have been achieved when the relative tolerance for all degrees of freedom was set to 0.0001. The simulation time for one fluid–wall interaction case based on three consecutive unsteady flow cycles employing 300 time iterations was approximately 50 CPU hours.

3 Results

The elastic tube model in Section 2.1 was performed numerically. Furthermore, the rigid tube model and other models, including an increase of elastic wall thickness and a decrease of internal radius, are introduced for evaluating the effects of different parameters on wave propagation. The velocity distributions in three sites (S1, S2, and S3 in Fig. 1) are presented at different times (t_1, t_2, t_3, t_4 in Fig. 2).

The velocity distributions in the elastic tube model are shown in Fig. 3. Sites S1 and S3 are located at the rigid tubes and the walls are static. However, site S2 is located at the elastic tube and the wall is dynamic, therefore the velocity of the wall is not zero. Due to the wall elasticity at site S2, the wall is distensible and thus the internal radius exceeds 10 mm. It is seen that the wall at site S2 extends to approximately 14.5 mm. In addition, it can be found that there is a significant characteristic in the velocity distributions. At sites S2 and S3, the velocity at time t_2 is higher than that at time t_1 . Meanwhile, the velocity at time t_4 is lower than that at time t_3 . We know that t_1 is the time that the peak velocity occurs while t_3 is the time that the lowest velocity appears in Fig. 2. If there were no delay in propagation, then the velocity at time t_1 should be the highest and the velocity at time t_3 should be the lowest. The velocity distributions at site S1 follow but those at sites S2 and S3 deviate. The reason is that the velocity propagation wastes some time. The findings indicate that an obvious time delay is embodied in the velocity distributions. It appears that our model can capture the time delay, which is an indicator of wave propagation. The distance between two sites divided

Fig. 3 The velocity distributions of the sites (S1-S3) in the elastic tube model

Fig. 4 The velocity distributions of the sites (S1-S3) in the rigid tube model

Fig. 5 The velocity distributions of the sites (S1-S3) with an increase of the wall thickness

Fig. 6 The velocity distributions of the sites (S1-S3) with a decrease of the internal radius

by the time delay is the wave velocity, which is an index for the expansion of an arterial wall.

Figure 4 depicts the velocity distributions at three sites in the rigid tube model. They are almost the same, whether it is the distributions or the values. The results indicate that the flow has been fully developed at site S1. Therefore the velocities at the latter locations do not change. Moreover, the velocity profiles of different cross sections at each time are held constant. Therefore, this implies that there is no time delay. Generally, the velocity distributions are blunt due to the large Womersley number.

The effects of elastic wall thickness were studied by increasing it from 2 to 4 mm. In a thickened vessel, the increase in the wall thickness corresponds to the disease of the arterial wall prior to the disease of the vessel cavity. Compared with the elastic tube model, the velocity distributions are very similar and the velocity values are lower (Fig. 5). Everywhere, the velocity at time t_1 is higher than that at time t_2 , which is a unique difference from the elastic tube model.

The effects of the internal radius were also investigated by reducing it from 10 to 8 mm. The clinical correspondence is stenosis. The velocity distributions at different locations are different (Fig. 6). The velocities of site S2 and S3 at time t_1 and t_2 are very close but the velocity at time t_3 is higher than that at time t_4 .

4 Discussion

The comparison of the velocity distributions in the elastic and rigid tube models illuminates that the elastic tube changes the flow development and distributions. Consequently, the velocity distributions are affected and the fully developed flow is delayed due to the elasticity. The elastic tube enhances the velocity values and induces a time delay in the velocity distributions. Spatial velocity distributions in the elastic tube model contain the characteristics of pulse-wave propagation.

Everywhere, the velocity at time t_1 is higher than that at time t_2 when increasing the wall thickness, which differs from the elastic tube model. However, the velocity at time t_4 is lower than that at time t_3 at sites S2 and S3, which resembles the elastic tube model. The comparative analysis seems that the increase of the wall thickness makes the time delay between the velocity peaks of different sites smaller while the time delay between the velocity minima is unchanged. Correspondingly, the time delay between minima is more stable and easy to catch in this sense, which is likely a good explanation that a velocity wave is quantized by usually measuring the time delay between the minima in clinics and experiments [15]. Compared with the elastic tube model, the decrease of the internal radius makes the velocity distributions different. Similarly, it is easy to recognize the time delay between the velocity bottoms. Certainly, the variation of arterial parameters changes spatial velocity distributions and thus results in changes of the time delay.

The changes of pulse waves are often considered as an indication of cardiovascular diseases in clinics and thus wave propagation analyses are currently proposed as both diagnostic and prognostic tools [16–18]. The forms and values of pulse waves would deviate from those under normal conditions when cardiovascular diseases occurred. The changes of pulse wave velocity indicate changes of the physiological parameters. The changes of these factors, representing actual significations, correlate with the relevant pathologies. For example, the rigid tube could be a good model for the fully calcified condition. The increase of the wall thickness and the decrease of the internal radius may be caused by stenosis. The endovascular repair of the vascular stent may cause a local change of arterial stiffness.

To obtain more specific information about flow physiology during the cardiac cycle, studies must be continued. Higher-order effects, such as anisotropy, nonlinear-elastic properties, and the taper of the arterial wall, can be considered in future studies.

5 Conclusions

Based on a model of wave propagation, spatial velocity distributions have been presented numerically. The results indicate that the velocity distributions embody an obvious time delay in the elastic tube model. The increase in the wall thickness and the decrease of the internal radius can change spatial velocity distributions. The model gives valid results for spatial velocity distributions, which provide important information for wave propagation.

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