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Three Dimensional Large Eddy Simulation of Blood Flow and Deformation in an Elastic Constricted Artery

Xi Gu, Guan Heng Yeoh, Victoria Timchenko

***Abstract*—**In the current work, a three-dimensional geometry of a 75% stenosed blood vessel is analyzed. Large eddy simulation (LES) with the help of a dynamic subgrid scale Smagorinsky model is applied to model the turbulent pulsatile flow. The geometry, the transmural pressure and the properties of the blood and the elastic boundary were based on clinical measurement data. For the flexible wall model, a thin solid region is constructed around the 75% stenosed blood vessel. The deformation of this solid region was modelled as a deforming boundary to reduce the computational cost of the solid model. Fluid-structure interaction is realized via a two-way coupling between the blood flow modelled via LES and the deforming vessel. The information of the flow pressure and the wall motion was exchanged continually during the cycle by an arbitrary Lagrangian-Eulerian method. The boundary condition of current time step depended on previous solutions. The fluctuation of the velocity in the post-stenotic region was analyzed in the study. The axial velocity at normalized position Z=0.5 shows a negative value near the vessel wall. The displacement of the elastic boundary was concerned in this study. In particular, the wall displacement at the systole and the diastole were compared. The negative displacement at the stenosis indicates a collapse at the maximum velocity and the deceleration phase.

***Keywords*—**Large Eddy Simulation, Fluid Structural Interaction, Constricted Artery, Computational Fluid Dynamics.

I. Introduction

A PULSATILE flow through an idealized constricted artery vessel was considered in this simulation, since such phenomenon commonly occurred in an arterial disease called atherosclerosis. Flow through stenosis vessel is created by high velocity spurt because of the constricted section. Even if the upstream flow of the stenosis is laminar generally, flow in the post-stenotic region can be more disordered or even turbulent depending on the flow status and the condition of the stenosis [1]. The altered flow structures may lead to a further impact on the development of the disease and arterial malformation [1]. Highly constricted artery results in stenosis flow transients, transition from laminar to turbulent flow downstream of the stenotic region. Ojha et al. [2] applied photochromic tracer method to observe the transition to turbulence was triggered before peak flow through the breakdown of waves. This experiment also measured the wall shear stress of a 75% constricted pipe flow, which fluctuated quite intensely during the vortex generation phase of the flow cycle. Doppler velocimeter was applied to obtain the post-stenotic blood velocity with 75% to 99% stenosis by [3].

Although experiment observations provided the general understanding and information about pulsatile flows in stenosis vessels, more specific and more accurate results of different constricted geometries can hardly be obtained because of the limitations of experiment equipment. There have been many studies where computational fluid dynamics (CFD) techniques have been applied to better understand the flow phenomena downstream of the stenotic region [4]-[6]. Among these numerical modellings, flows were considered as laminar because of the mildly stenosed geometry and low Reynolds number (Re). However, moderate constrictions (50%-80%) showed more significant influence on flows in experiments because of the transition to turbulence caused by high Reynolds number. According to the experiment of [2], the Reynolds number of the flow in a moderate constricted vessel had a range from around 200 to higher than 2000. Because of this wide range of Reynolds number, a suitable simulation method should be selected to model the turbulence. Applying large eddy simulation to analyze large scales of the flow field can lead to a better fidelity than other methodologies such as Reynolds-averaged Navier–Stokes (RANS) approaches. RANS models the smallest (and most expensive [7]) scales of the solution as well, instead of resolving small scales as direct numerical simulation (DNS) does. This result in a high computational cost for the applications of engineering systems, which may have a complicated geometry or flow structures, such as turbulent jets, attainable using supercomputers, landing gear, vehicles, and pumps. However, direct numerical simulation (DNS), which analyses every scale of the solution, can cause an extremely high cost for almost all systems with complicated geometry or flow structures.

According to [8], in Large Eddy Simulation (LES) merely the large scales, since the sub-grid scales (SGS) are modelled while the energy-containing scales are solved. It has already been proved that Large Eddy Simulation, which lies between Reynolds-average Navier-Stokes (RANS) and direct numerical simulation (DNS), is an excellent approach for

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turbulence flow simulations, including modelling a transition to turbulent pulsatile flow [9]-[11]. For a low Reynolds number flow, direct numerical simulation can be applied to solve it; however, Large Eddy Simulation can be applied to solve not only low but also high Reynolds number flows. Additionally, it costs less grid densities and time than DNS, because the smallest scales do not need to be resolved in large eddy simulations. Nevertheless, few applications of LES were found in most of earlier biomechanical investigations. In the research studied by Paul et al., a Large Eddy Simulation (LES) with the dynamic sub-grid scales (SGS) model was utilized to learn a three-dimensional pulsatile blood flow model in a constricted artery [8]. This was a new way to understand the transition to turbulent flow of a pulsatile model in a constricted blood vessel by applying LES methodology. According to the result of this study, the sub-grid scale model has an excellent performance on the resolved scale. As a consequence, this proved again that applying LES in biological flow is important. Large Eddy Simulation can be a more accurate approach to model transition to turbulent pulsatile blood flow [10]-[12].

There have been many studies where computational fluid dynamics (CFD) techniques have been applied to better understand the flow phenomena downstream of the stenotic region [8], [13]. In principle, there is always an interaction between fluid flowing and its surrounding. The vessels deform because of the force from the flow, and the fluid part is influenced by the displacement of the vessel wall. Nevertheless, there have been very few studies on the dynamics of post-stenotic blood flow coupled with a highly constricted deforming artery vessel. The mechanical properties of the arteries usually have an obvious impact on blood flows, especially in a constricted blood vessel. These mechanical properties were studied in many investigations about the relationship between the diameter and the pressure [14]. Normally, the wall of a blood vessel consists of three layers, which shows an elastic property [5]. This elastic property leads to a deformation of the blood vessel and a displacement of the vessel wall, which cause an interaction with the blood flow. In order to obtain a better and clearer understanding of the biological flow in a constricted blood vessel, numerous researches have investigated the stress and stiffness of arteries. Particularly, cross-sectional stress distributions were investigated [15]. In this study, Cheng et al. also illustrated that to high stress regions, the histology of cracking sites conformed quite well. It has also been demonstrated that in a two-dimensional atherosclerotic vessel wall, a large circumferential tensile stress was concentrated around the plaque edges [16]. Additionally, from clinical discoveries, fibrous cap fissuring usually happened around the plaque shoulder in the weak structure [17]-[19]. It can be seen from these observations that mechanical stress of blood vessels might increase the possibility of plaque rupture.

Fluid-structural interaction is a recently applied methodology to learn the interaction between fluid flow and its surrounding. This interaction cannot be neglected in blood flow [20]. Although it was originally applied to solve aeroelasticity problems, it has increasingly been used in biomechanical simulations, including blood flow simulations. This is considered as an important progress in research on blood flows, since it lead to a better understanding of the structural change of both the blood flow and the blood vessel. Applying Fluid-Structure Interaction into the simulation enhanced the cross-sectional area of the flow in the non-stenosed regions, leading to a decrease of the velocity profile and enlarged the flow recirculation effects [21]. More specifically, the wall shear stress (WSS) was reduced by applying FSI simulation. Additionally, the maximum stress appeared at the shoulder of the constricted part [21]. Oscuii et al. applied FSI modelling to learn the interaction between the blood flow and the elastic blood vessel [22]. The results demonstrated that fluid-structural interaction modelling with pressure boundary conditions supplies an appropriate simulation of haemodynamic shear stress, as well as wall circumferential stress. The existence of a constricted part in a blood vessel has been assumed to cause a very low pressure of an area, where the most serious obstruction is, especially in a high constricted blood vessel. Tang et al. pointed out that the blood vessel is possible to collapse in this part and totally suspense the flow of blood [23]. They analyzed a Fluid Structural Interaction simulation on several of constricted blood vessels and studied the wall deformation, flow structures and stress distribution. In their studies, it is demonstrated that a decrease of pressure appears near the constricted zone. However, due to the wall thickness of that region, the blood vessel is strong enough to avoid collapse. Ku supposed that besides of this kind of collapse, the obvious change of pressure could lead to an acceleration of the fatigue near the stenosed region. As a consequence, the possibility of plaque rupture would be enhanced [24]. Lee and Xu comprehensively investigated a blood flow through a stenosis vessel with sharp transitions [5]. The geometry of this study is based on a previous experiment by [2]. A linear elastic vessel wall model and a Newtonian behaved fluid, which has similar properties to Ohja's experiment [2], were applied in [5]. Commercially available software, which has a coupling methodology depends on geometric displacements, was applied to carry out this simulation. It is found that the result of their study is similar to the result of the previous experiment. Because of this, the techniques they applied, including the fluid-structural interaction, were proved to be appropriate. It is observed from their results that the artery expansion caused by internal pressure lead to the decrease of the flow velocity and a maximum stress around the shoulders of the constricted zone. Although in this simulation the wall was time-dependent but the motion of the moving boundary was predicted by the wall model. This simulation method means the interaction between flow and wall were only combined in single time step, which is not continuous. Initially, the investigation studied by Chakravaty and Mandal validated the supposed methodology of simulating the interaction between the fluid flow and the wall deformation [25]. After that, Mandal established a non-Newtonian fluid model based on Generalized Power Law. The other simulation

method of this model is similar to the previous one. Although this model demonstrated more reasonable fluid characteristics, in terms of velocity, wall shear stress (WSS) and pressure, comparing to the previous investigation, the deformation of the wall depended on governing equations instead of material properties [26].

In the current work, a three-dimensional geometry of a 75% stenosed blood vessel is analyzed. Large eddy simulation (LES) with the help of a dynamic subgrid scale Smagorinsky model is applied to model the turbulent pulsatile flow. The flow rate ranges between 2 ml/s and 10 ml/s for a period of 0.25 s was applied to simulate the arterial systole. The results of a similar previous simulation were validated against the experimental data of [2]. For the flexible wall model, a thin solid region is constructed around the 75% stenosed blood vessel. The deformation of this solid region was simplified as a moving boundary with properties of an abdominal aorta. Fluid-structure interaction is realized via a two-way coupling between the blood flow modelled via LES and the deforming vessel. The results of this simulation provide insights to more realistic understanding of arterial flow and deformation.

II. Numerical Method

A. Model Description

The problem considered was pulsatile flow through a constricted artery vessel. The constricted part was 75% reduction of cross sectional area. The centerline velocities of three mesh models (0.49 million, 1 million and 1.9 million) during three flow cycles were compared. The difference of the centerline velocity between the coarse and fine mesh is around 1.6%. The difference between the fine mesh and the finer mesh is 0.6%. The deference between the first cycle and the second cycle was around 6.3%. The deference between the second cycle and the third cycle was smaller than 0.1%. Therefore, results of 1 million elements' model were obtained from the second flow cycle to ensure the accuracy with relatively less computational cost. The flow was supposed as an incompressible Newtonian flow, and the turbulence was simulated by Large Eddy Simulation (LES). Since the artery vessel was assumed to be incompressible, the wall model was simplified as a moving boundary, with a linear circumferential elastic modulus.

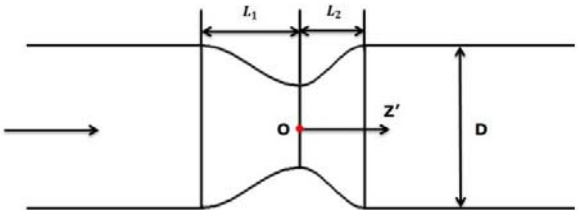

Fig. 1 The geometry of 75% axisymmetric stenosis used in the simulation, where L_1=3 mm, L_2=2 mm and D=5 mm. The normalized distance from the center of stenosis is given by Z=Z'/D

In this simulation, the internal diameter and the thickness of the artery are 5 mm and 0.5 mm. Fig. 1 illustrates the geometry of 75% constricted area, which was formed by the reduction of cross sectional area. O is the origin. X axes is the axes of the artery. D is the diameter of normal region. Z indicates the normalized distance from the origin. (Z=Z'/D) In the axial direction, the radius was reduced from the left side based on a sine curve equation and was increased to the right side based on another sine curve equation which has a higher frequency. The minimum diameter is 2.5 mm (50% reduction of diameter and 75% reduction of cross sectional area. The inlet of the pulsatile flow is in the left side.

B. Flow Modelling

The fluid was assumed to be Newtonian fluid which was also incompressible. The density of the fluid was $1060\ kg/m^3$, and the viscosity was $0.0035\ N \cdot s/m^2$. The inlet flow rate is shown in Fig. 2. The systole was simulated as a pulsatile flow stage and the diastole was simulated as a steady flow stage. Based on the Handbook of Biomaterial Properties, the volumetric flow range of an abdominal aorta is from 2 ml/s to 10 ml/s. The period of the systole was 0.25 seconds (1/3 of the whole cycle [27]) and the period of the diastole was 0.5 seconds. The whole cycle was 0.75 seconds, which indicate a heart rate of 80 beats per minute. A constant pressure 10000 Pascal (75 mmHg) was applied at the outlet. Two cycles were calculated in this simulation.

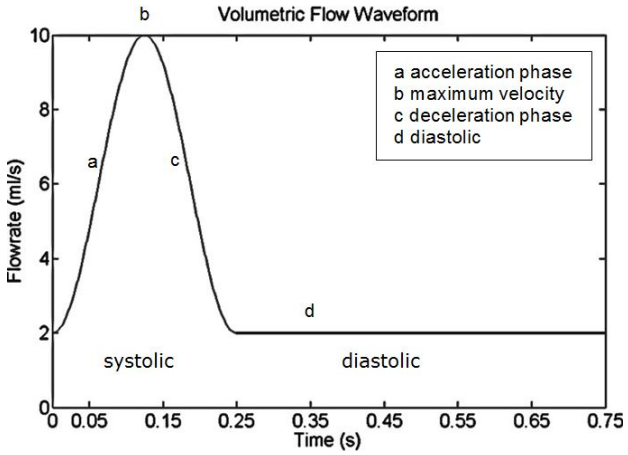

Fig. 2 The volumetric flow waveform

The maximum Re was around 2300. According to the previous studies, the acceleration caused by the stenosis generated reversed flows and transition to turbulence in the post-stenotic regions therefore Large Eddy Simulation (LES) was applied to analyze the turbulence.

For the incompressible Newtonian fluid, the governing equations of Large Eddy Simulation are three dimensional, time-dependent filtered Favre-Averaged Navier-Stokes equations, which are shown in Cartesian coordinates as follows:

LES explicitly solves for the large-scale turbulent eddies while the small-scale eddies are modelled by means of a SGS model via a filtering procedure. Defining an arbitrary filtered variable to be $\bar{\varphi}(x_j, t)$ and adopting Favre-averaging:

$$\tilde{\varphi}(x_j,t) = \frac{\overline{\rho\varphi(x_j,t)}}{\bar{\rho}} \quad (1)$$

where $\bar{\rho}$ is the filtered density, the equations governing the conservation of mass and momentum flow may be written as;

$$\frac{\partial \bar{\rho}}{\partial t} + \frac{\partial(\bar{\rho}\tilde{u}_j)}{\partial x_j} = 0 \quad (2)$$

$$\frac{\partial(\bar{\rho}\tilde{u}_i)}{\partial t} + \frac{\partial(\bar{\rho}\tilde{u}_i\tilde{u}_j)}{\partial x_j} = -\frac{\partial \bar{P}}{\partial x_i} + \frac{\partial \tilde{\sigma}_{ij}}{\partial x_j} + \frac{\partial M_{ij}}{\partial x_j} \quad (3)$$

where $\tilde{u}_i$ is the filtered velocity vector and $\bar{P}$ is the filtered pressure. The filtered stress tensor $\tilde{\sigma}_{ij}$ based on Stokes' hypothesis in (3) can be written as;

$$\tilde{\sigma}_{ij} = \mu\left(\frac{\partial \tilde{u}_i}{\partial x_j} + \frac{\partial \tilde{u}_j}{\partial x_i}\right) - \frac{2}{3}\mu\frac{\partial \tilde{u}_k}{\partial x_k}\delta_{ij} \quad (4)$$

where μ is the dynamic viscosity.

Fig. 3 The 1,024,250 hexahedral cells of the simulation mesh

Fig. 3 shows 1 million hexahedral cells of the computational fluid dynamics (CFD) model. The quadrilateral faces of cross-sectional area were nearly 4100 faces. Both of the pre- and the post-stenotic vessel were 50 mm long to fully establish the flow before the stenosis and to obtain sufficient flow feature of the downstream region. The time step size of the solution was 10^{-4} s.

C. Wall Modelling

Wall modelling was only applied in the elastic wall model, which analyzed the Fluid-Structural Interaction. The artery vessel was assumed to be incompressible and was simplified as a moving boundary, with a circumferential elastic modulus. The Young's Modulus, the diameter and the wall thickness of the wall model referred to an abdominal aorta. According to Handbook of Biomaterial Properties, the circumferential pressure-strain modules are 0.07 MPa to 0.15 MPa. This circumferential incremental elastic modulus was from human arterial walls of young adults (≤35 years) at a transmural pressure of 100 mmHg [28]. In this simulation the elastic modulus was 0.1 MPa. The Poisson ratio was 0.5. The wall thickness of non-constricted vessel was 0.5 mm, while the maximum wall thickness of the constricted region was 1.75 mm. The motion of the moving boundary depended on the pressure increment, which is shown as:

$$\Delta R = \frac{\Delta P_i R}{E_p} \quad (5)$$

where ΔP_i is the transmural pressure increment, R is the diameter of the vessel, ΔR is the change in the diameter due to ΔP_i. E_p is the circumferential pressure-strain modulus measured at 100 mmHg transmural pressure [28].

D. Fluid Structural Interaction

Fig. 4 illustrates the process of the Fluid-Structural Interaction in the elastic wall model. The information of the flow pressure and the wall motion was exchanged continually throughout the cycle. The boundary condition of current time step depended on previous solutions. The Fluid Structural Interaction was achieved by a user-defined function (UDF). UDF is a C function that can be dynamically loaded with the ANSYS FLUENT solver [29]. A mesh motion function was applied to obtain the pressure on the fluid-solid interface, calculate the displacement of the moving boundary and update the position of each node based on the deflection due to fluid-structural interaction. To ensure that the moving boundary was not updated until the flow with the previous boundary conditions reached the convergent level, the moving boundary of the FSI interface was updated every 5×10^{-3} s. By applying user-defined function, only one solver, ANSYS FLUENT, was employed to achieve fluid-structural interaction.

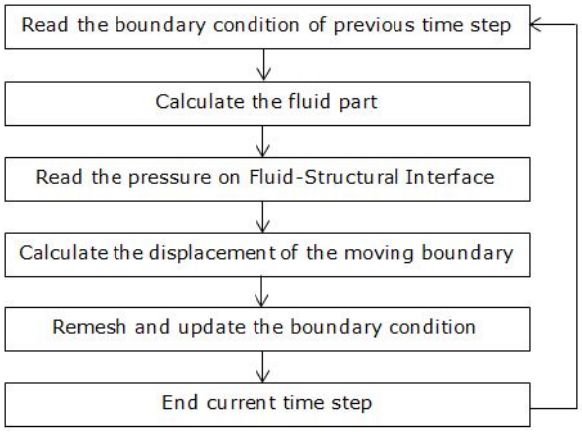

Fig. 4 The scheme of Fluid-Structural Interaction

III. Results

A. Velocity

Figs. 5 and 6 show the comparison of centerline velocity and axial velocity at three normalized distances in post-stenotic region. In the deceleration phase and the diastole, the axial velocity u near the vessel wall at Z=0.5 was negative. This negative velocity indicates reversed flows in the post-stenotic region. It can be predicted that there may be a transition to turbulent flow in this region.

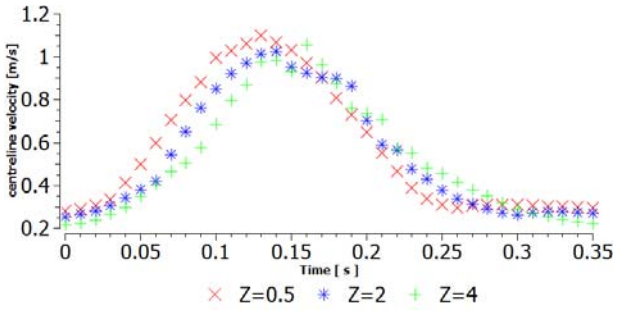

Fig. 5 Centreline axial velocity at normalised post-stenotic distance of Z'=0.5, Z'=2 and Z'=4

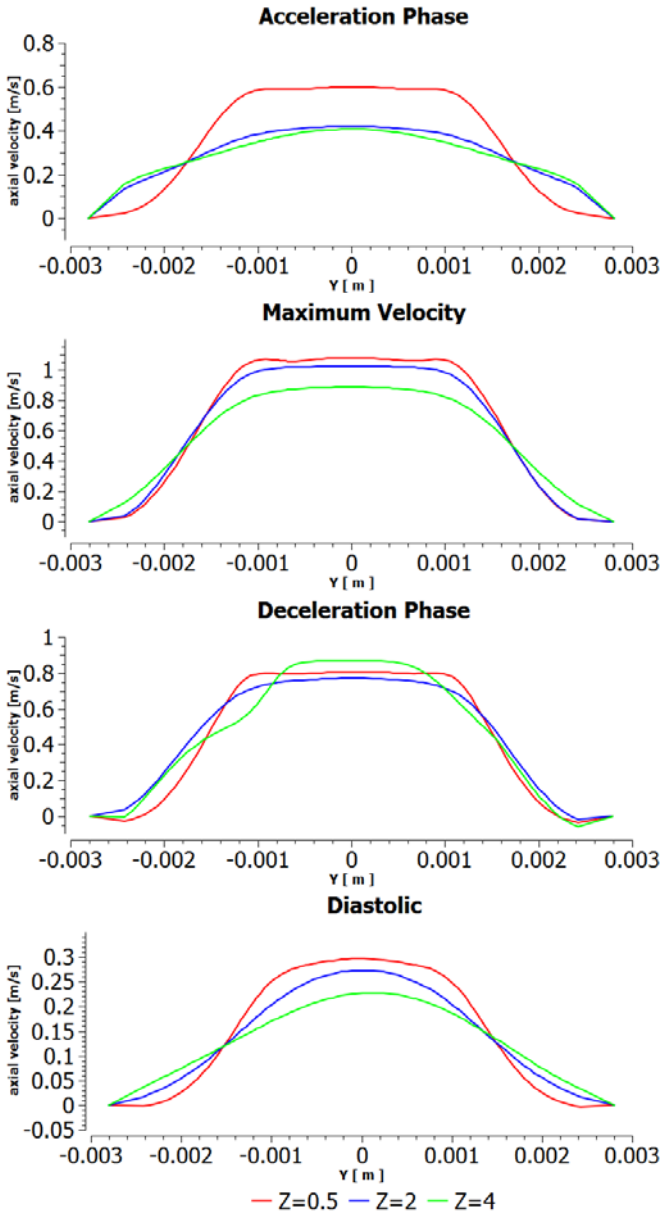

Fig. 6 Axial velocity in systole and diastole

Fig. 7 plots the velocity contours at four phases during the flow cycle. The velocity fluctuated along the artery at deceleration phase because of the reduction of the flow rate and the wall displacement.

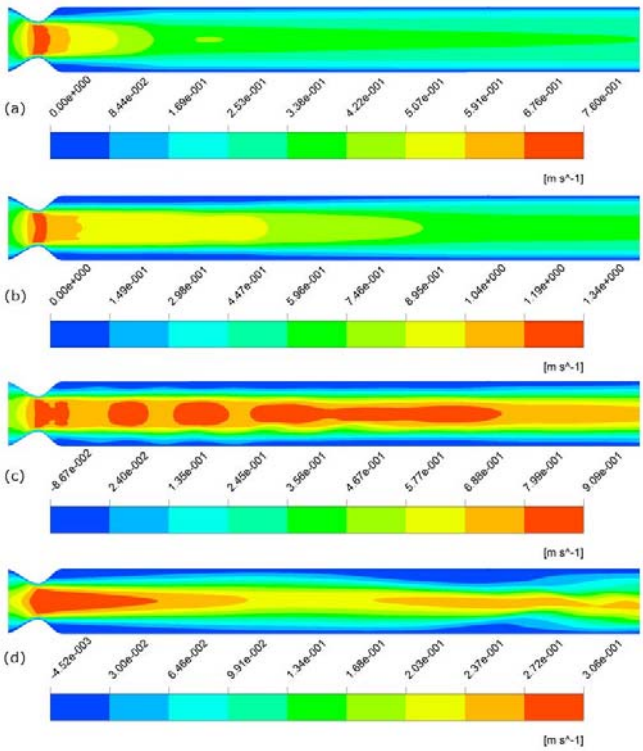

Fig. 7 The velocity contours at four stages shown in Fig. 2

B. Wall Displacement

The wall displacement was compared between the systole and the diastole in Fig. 8. During the acceleration phase, the whole artery expanded. At the maximum velocity, the displacement around the stenosis is smaller than the displacement of diastole, which means this region had a collapse. During the deceleration phase, the radius of the whole artery decreased.

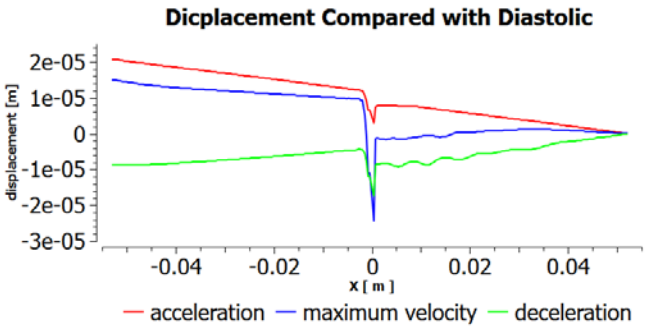

Fig. 8 The wall displacement of systole compared with diastole throughout the artery

IV. Conclusion

In this study, the deformation of this solid region was modelled as a deforming boundary to reduce the computational cost of the solid model. Fluid-structure interaction is realized via a two-way coupling between the blood flow modelled via LES and the deforming vessel. The

information of the flow pressure and the wall motion was exchanged continually during the cycle by an arbitrary lagrangian-eulerian method.

The fluctuation of the velocity in the post-stenotic region was analyzed in the study. The axial velocity at normalized position Z=0.5 shows a negative value near the vessel wall. The displacement of the elastic boundary was concerned in this study. In particular, the wall displacement at the systole and the diastole were compared. The negative displacement at the stenosis indicates a collapse at the maximum velocity and the deceleration phase.

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