

EFFECT OF TIME PERIOD OF PULSATION ON THE FLOW BEHAVIOR THROUGH AN ECCENTRIC STENOSED ARTERY

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ABSTRACT

A numerical simulation is carried out to investigate the effect of time period of pulsation on the flow behavior through an arterial stenosis. A semicircular eccentric stenosis is considered which is more relevant in cardiovascular system rather than the symmetric one. The degree of stenosis has been considered as 50%. The pulsatile flow has been represented by a sinusoidal wave form. The Reynolds number is varied from 200 to 970 during the pulsation while the Womersley number has been varying from 7.1 to 10.1 corresponding to the variation of time period from 0.3s to 0.7s. Results showed significant variation of flow behavior due to the variation of pulse periods. Moreover vortex rings are developed asymmetrically and the post stenotic areas are severely affected by the vortex rings. In addition, a significant variation of wall shear stress has been observed at different time periods.

Keywords: Pulsatile Flow, Stenosis, Wall Shear Stress.

1. INTRODUCTION

Narrowing of an arterial lumen (stenosis), tends to occur in regions of disturbed flow and low wall shear stress. Once plaque develops and encroaches into the lumen, further flow disturbances are established. Hemodynamically significant stenoses experience an increased shear stress in the entrance region. In the post stenotic region, the flow decelerates and tends to become unstable with separation, recirculation and generally for the more severe constrictions, transition to turbulence which influences flow resistance, shear stress, pressure, mass transport from the blood to the vessel wall. Low shear stress have been associated with adverse changes in the endothelium including apoptosis, diminished nitric oxide synthesis and increased expression of adhesion molecules and chemotactic factors concluded that regions that are prone to atherosclerosis, such as bends and bifurcations. The fluid dynamical behavior of blood flow through constricted channel has been studied for the last many years. Ahmed and Giddens [1], Ohja et al. [10] experimentally visualized the post stenotic behavior of pulsatile flow through stenoses. Beyond these there are many computational researches on the investigation of pulsatile flow in stenotic geometries [2, 4, 5, 7, 9-12]. However most of the previous researches were based on the assumption of symmetric stenoses in the artery. In addition these researches considered the simple pulsation flow. In the present study, a numerical simulation has been carried out to investigate the effect of pulse period on the flow behavior flow through an eccentric arterial stenosis.

2. NUMERICAL METHODS

In general the blood is a non-Newtonian fluid. In present study, it is assumed to be Newtonian, homogeneous, and incompressible. This is because it is considered large arteries with radii of the order of 1.0 mm, where the velocity and shear rate are high. The apparent viscosity is nearly a constant in large arteries with diameter (~ 5 mm) and therefore the non-Newtonian effects can be neglected. The governing equations are the 2D Navier Stokes equations of motion and mass continuity. Further, since in the present study, the Reynolds number is below 1000, the flow can be modeled by laminar modeling which is equivalent to turbulence modeling [9].

The governing equations are discretized by the weak formulation of finite element method (FEM). The computational domain is subdivided into finite number of elements. The elements used are the isoparametric triangular and the discretization is shown in Fig. 1. For the time integration, second order accurate implicit backward difference formulation is used.

3. MODEL ARTERY AND COMPUTATIONAL CONDITIONS

A simple model of the planar artery with one-sided semicircular stenosis is considered in the present study as shown in fig 1 (a). The Height or diameter of the artery H is 8mm. The degree of stenosis is defined as, $s = (H - h)/H \times 100\%$. The domain extends $8H$ and $16H$ upstream (L_u) and down stream (L_d) from the centre of the stenosis, respectively. The viscosity of the blood (μ) is taken as 3.5×10^{-3} Pa.s with a mass density (ρ) of 1060

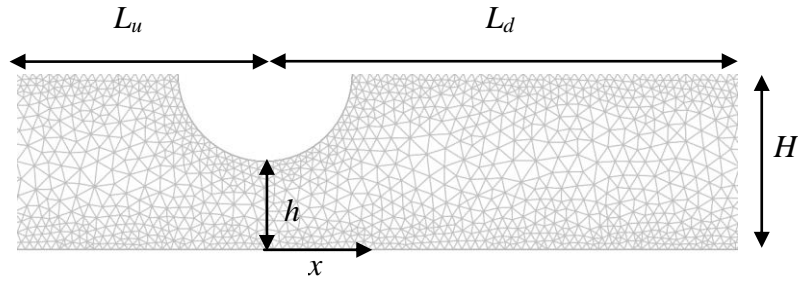


Fig 1. Model eccentric artery with 50% stenosis

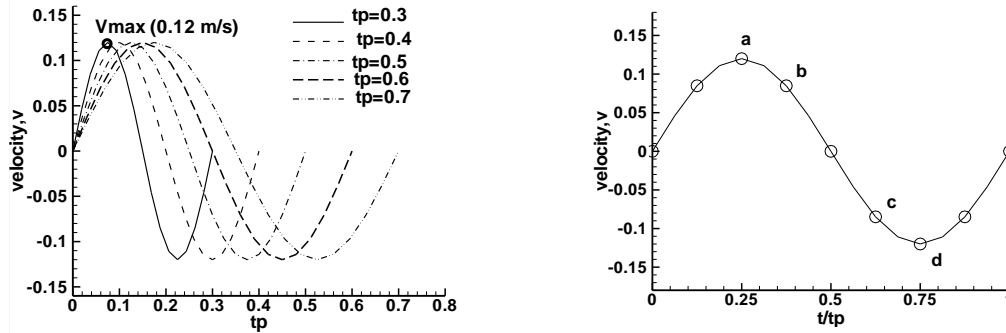


Fig 2. (a) Pulsatile inflow at different time periods; (b) sinusoidal wave form

kg/m^3 . At the inlet boundary the input velocity expression is $u = u_0 \sin(2\pi t/t_p)$ where, t_p = pulse period, $u_0 = 0.12$ m/s (corresponding to Reynolds number 290). The Womersley parameter ($\alpha = \frac{H}{2} \times \sqrt{2\pi\rho/t_p\mu}$) varies from 7.1 to 10.1 as time period varies from 0.7s to 0.3s. The corresponding pulsatile flows are shown in Fig. 2(a). Observations have been taken at the points of maximum acceleration (a), maximum velocity (b), maximum deceleration (c) and minimum velocity (d) as shown in Fig. 2(b). All the arterial walls are considered to be no-slip.

3. RESULTS AND DISCUSSION

Figure 3 shows the instantaneous streamlines of the flow through an eccentric arterial stenosis at five different pulse periods for 50% stenosis. A, B, C, D, E, and F represents the flow behavior corresponding to $t_p = 0.3\text{s}, 0.4\text{s}, 0.5\text{s}, 0.6\text{s},$ and 0.7s , respectively. At phase *a* (Fig. 2(b)) when the flow velocity becomes maximum, reduction of flow acceleration causes the vortices to be larger for longer time periods at the upper wall. Furthermore, the separated shear layer reattaches to the upper wall after a certain distance. This reattachment causes to form vortex at the lower wall. At next phase, *b* (Fig. 2(b)), of maximum deceleration vortices (elliptic) are found to enhance their major axes length with the increase of time period because, for higher time period the Womersley parameter reduces that results in convective shear layer instability. The vortices formed are elliptic in nature and with the increase of time period the distance between the centre of vortex and stenosis becomes dominant. Lift of streamlines from the lower wall is observed for the stenosis. Again at phase, *c* (Fig. 2(b)) of maximum acceleration, for the negative half cycle, the instability of streamlines is observed for increased time period. The lower wall is also observed to be affected as time period increases. At phase *d*, (Fig. 2

(b)), of maximum velocity the flow direction changes and the size of the vortices formed near the upper wall is found to be increased with time period. The lifting of streamlines is also observed near lower wall, with time periods as the shear layer generated near the lip of stenosis becomes longer for higher time period and tries to reattach with the upper wall.

It is recognized that the wall shear stress (WSS) is the primary fluid mechanical properties that affect the biological arterial response [6]. Figure 4 show the instantaneous WSS for 50% stenosis for the upper wall. As this numerical study deals with time dependent pulsatile flow so observations have been taken for four significant points at a, b, c, d. (Fig. 2(b)). In Fig. 4, a line has been shown just above the point $x/H = 0$ which extends ($-0.5 \leq x/H \leq 0.5$), represents the location of stenosis at the upper wall. From Fig. 4 (a) it is observed that at the point of maximum velocity, *a*, the ($t/t_p = 0.25$) downstream is severely affected. With the increase of time period the value of wall shear stress (WSS) increases. It is because of the enhancement of the vorticity with time periods. The vortices (elliptical) become dominant in length along the major axes for longer time periods which results in higher WSS at upper walls. In case of lower wall (Fig. 4(b)), a significant increase in WSS is observed due to the higher strain rate near the stenosis and in the downstream it follows the same trend as found in upper wall. WSS increases with time period at phase *b*, ($t/t_p = 0.375$) the point of maximum deceleration. For upper wall (Fig. 4(a)) WSS increases with decreasing time period whereas for lower wall (Fig. 4 (b)) this behavior is completely reversed. The maximum positive peak of WSS at upper wall is about 2.42 times higher than the lower wall. WSS variation for both walls is observed up to $x/H \approx 4$. At phase *c*, during the time of maximum acceleration at the

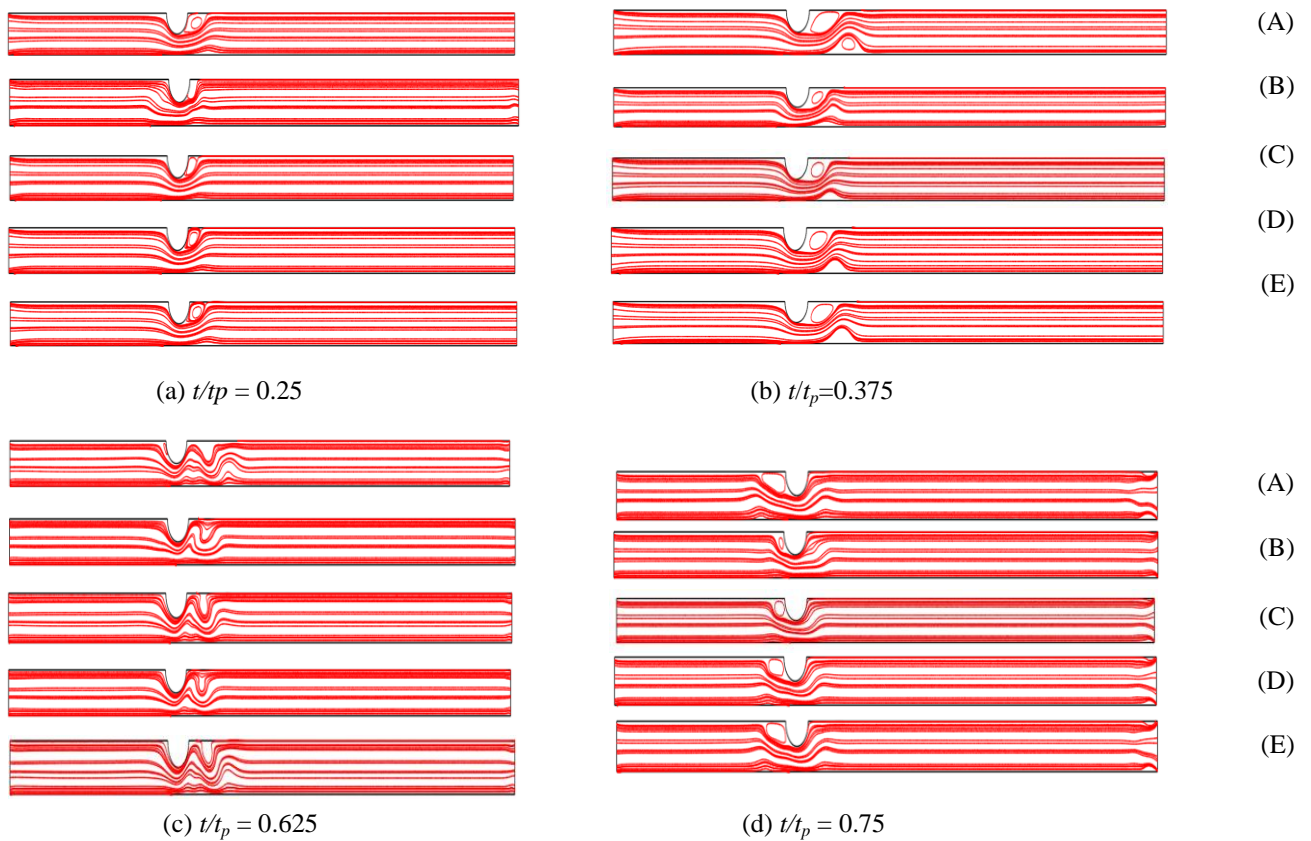
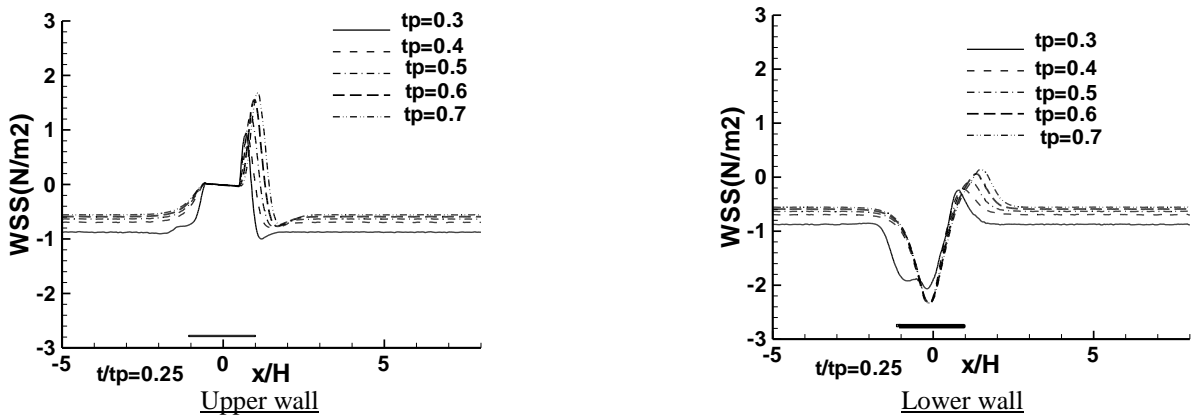


Fig 3. Streamlines plot at different time instants for different pulse periods for 50% eccentric stenosed artery

negative half cycle ($t/t_p = 0.625$) of the sinusoidal wave form, lower wall is significantly affected rather than upper wall as shown in Fig. 4(c). WSS variation follows a remarkable trend. At the downstream region of the lower wall (Fig. 4(c)), WSS increases on both sides of time period 0.5s. So time period 0.5s can be considered as the critical point. But the value of WSS experiences an increasing trend with decreasing time period at downstream of upper wall (Fig. 4(c)). At phase d, the point of maximum velocity for negative peak, ($t/t_p = 0.75$). A significant variation of WSS at upper wall (Fig. 4(d)) is found within $-3 < x/H < 0$. WSS follows the

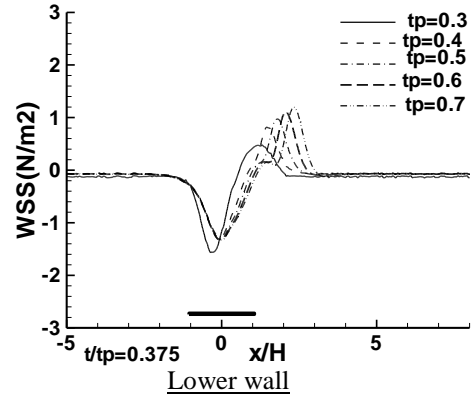
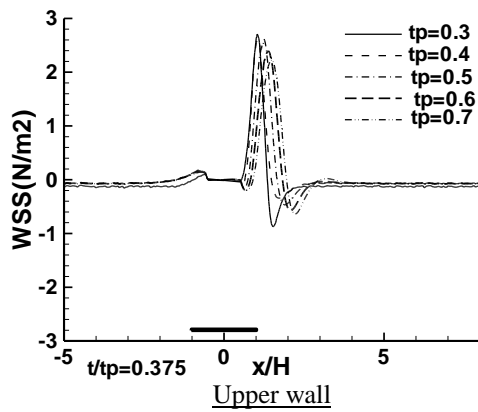
similar trend as the upstream of lower wall shown in Fig. 4(d). WSS at the upper wall decreases up to time period 0.5s and then further increases soon after crossing the value of $t_p = 0.5$ s. In case of lower wall (Fig. 4(d)) the positive peak is found near $x/H = 4.0$ due to the interaction of the particles with the lower wall which results in higher strain rate as well as high WSS. Here WSS enhances with time period for both upstream and downstream.

The time averaged wall shear stress (TAWSS) is evaluated considering one complete sinusoidal waveform for variable time period and the distribution

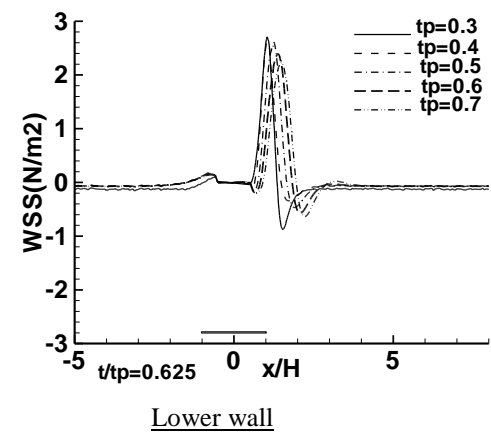
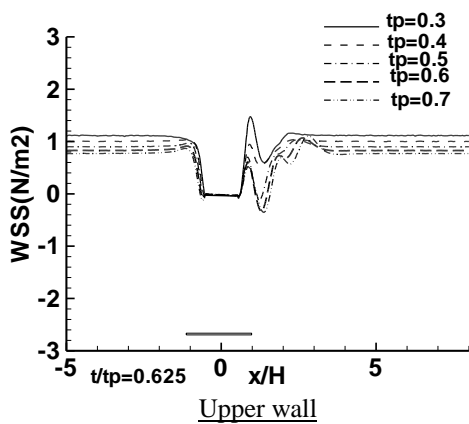


(a)

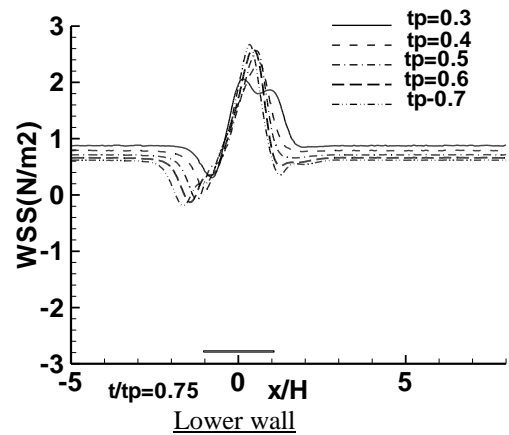
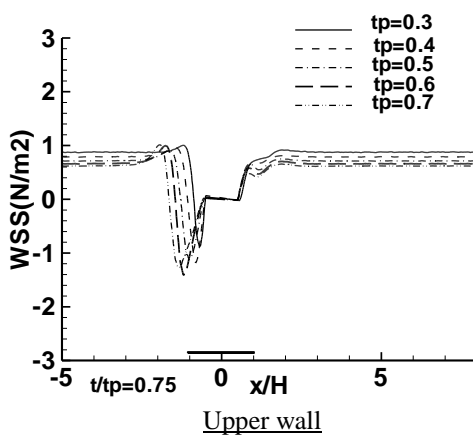
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(b)



(c)



(d)

Fig 4. Wall shear stress (WSS) distribution along the upper and lower wall of the model arterial stenosis for different pulsatile time periods, t_p ; (a) $t/t_p = 0.25$, (b) $t/t_p = 0.375$, (c) $t/t_p = 0.625$, and (d) $t/t_p = 0.75$.

has been shown in Fig. 5. It is found that the peak TAWSS at upper wall is about 2.3 times higher than the lower wall. The fluctuation in TAWSS is found up to $x/H \approx 4$ for both of the walls. The downstream for upper wall shows a trend taking $t_p=0.5$ s as critical time period from which TAWSS starts to increase on both sides of $t_p=0.5$ s. The upstream condition of upper wall is

also same as the downstream condition. On the other hand (in Fig 5 (b)), the upstream condition shows a decrease in TAWSS with decreasing time period at lower wall. Further a significant region of negative TAWSS is found at the lower wall for 50% stenosis. This means that the lower wall is also affected by the upper wall.

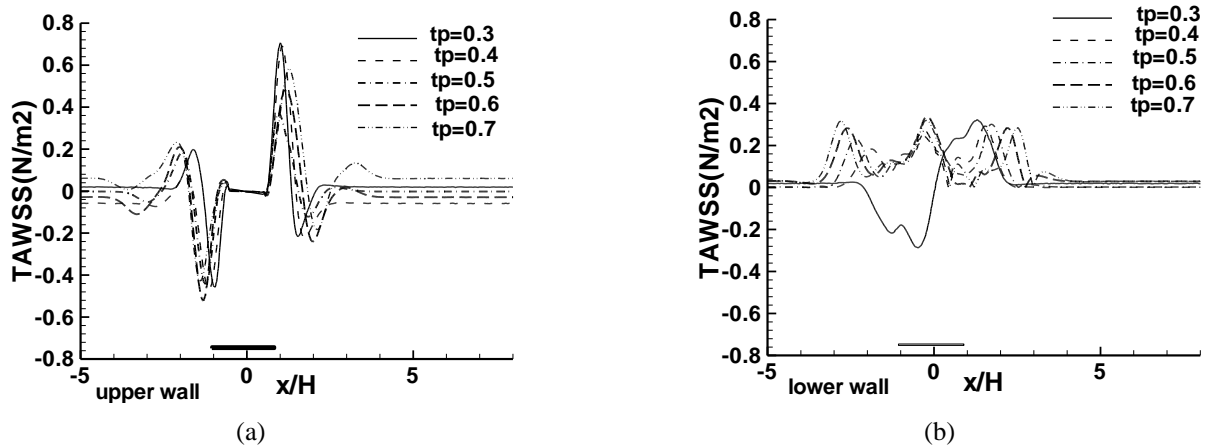


Fig 5. Distribution of time averaged wall shear stress (TAWSS) along the (a) upper wall and (b) lower wall for different time periods, t_p

4. CONCLUSIONS

The effect of time period of pulsatile flow has been simulated numerically through an eccentric artery of 50% stenosis. The Reynolds number varies from 200 to 970 during the pulsation and Womersley number has been varying from 7.1 to 10.1 corresponding to the variation of time period from 0.3s to 0.7s. A short summary of results are as follows:

- Shear layers and vortex rings are generated asymmetrically in the stenosed artery and distributed in the post stenotic regions and becomes more unstable for increasing time period.
- The hemodynamic parameter such as WSS fluctuates during the pulse period. Mean flow characteristics such as TAWSS shows that both of the walls are affected by shear layer and vortex rings. At $t_p=0.5s$, a critical behavior of TAWSS at the upper wall, along both the upstream and downstream is observed. However, at the upstream of lower wall, a gradual decrease of TAWSS is noticed with decreasing time period. However, downstream shows a critical distribution of TAWSS just opposite to the upper wall.

5. REFERENCES

1. Ahmed, S.A., Giddens, D.P., 1984, "Pulsatile Poststenotic Flow Studies with Laser Doppler Anemometry", *Journal of Biomechanics*, 17: 695-705.
2. Beratlis, N., Balaras, E., Parvinian, B., Kiger, K., 2005, "A Numerical and Experimental Investigation of Transitional Pulsatile Flow in a Stenosed Channel", *Journal of Biomechanical Engineering*, 127: 1147-1157.
3. Berger, S.A., Jou, L.D., 2000, "Flows in Stenotic Vessels", *Annual Review of Fluid Mechics*, 32: 347-382.
4. Buchanan, Kleinstreuer, Jr.C., Comer, J.K., 2000, "Rheological Effects on Pulsatile Hemodynamics in a Stenosed Tube", *Computers and Fluids*, 29: 696-724.
5. Griffith, M.D., Leweke, T., Thompson, M.C., Hourigan, K., 2009, "Pulsatile Flow in Stenotic Geometries: Flow Behaviour and Stability", *Journal of Fluid Mechanics*, 622: 291-320.
6. Ku, D.N., 1997, "Blood Flow in Arteries", *Annual Review of Fluid Mechanics*, 29: 399-434.
7. Long, Q., Xu, X.Y., Ramnarine, K.V., Hoskins, P., 2001, "Numerical Investigation of Physiologically Realistic Pulsatile Flow Through Arterial Stenosis", *Journal of Biomechanics*, 34:1229-1242.
8. Nichols, W.W., O'Rourke, M.F., McDonald's, 1998, *Blood Flow in Arteries: Theoretical, Experiental and Clinical Principles*, Oxford University Press.
9. Nosovitsky, V.A., Hegbusi, O.J., Jiang, J., Stone, P.H., Feldman, C.L., 1997, "Effects of Curvature and Stenosis-like Narrowing on Wall Shear Stress in a Coronary Artery Model with Phasic Flow", *Journal of Computational Biomedical Research*, 30: 61-82.
10. Ohja, M., Cobbold, R.S.C., Johnston, K.W., Hummel R.L., 1989, "Pulsatile Flow Through Constricted Tubes: an Experimental Investigation using Photochromic Tracer Method", *Journal of Fluid Mechanics*, 203: 173-197.
11. Paul, M.C., Molla, M.M., Roditi, G., 2009, "Large-eddy Simulation of Pulsatile Blood Flow", *Medical Engineering and Physics*, 31:153-159.
12. Varghese, S.S., Frankel, S.H., 2003, "Numerical Modeling of Pulsatile Turbulent Flow in Stenotic Vessels", *Journal of Biomechanical Engineering*, 125: 445-460.
13. Womersley, J.R., 1955, "Method for the Calculation of Velocity, Rate of Flow and Viscous Drag in Arteries when the Pressure Gradient is Known", *Journal of Physiology*, 127:553-563.

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