21st Australasian Fluid Mechanics Conference
Adelaide, Australia
10-13 December 2018

Effect of Degree of Stenosis on the Pulsatile Flow Pressure Drop in a Coronary Artery

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Abstract

Artery blockage due to plaque formation affects the performance of the heart considerably. These plaques are formed inside an artery and forced the heart to work harder to feed the vessels and organs with oxygenated blood. In this study, the pressure drop of a pulsatile flow is calculated in diseased coronary arteries with different degrees of stenosis and for blood with different levels of blood viscosity. Pressure drop is much more significant at the severe degrees of stenosis (more than 60%) compared to the mild and moderate cases. The effect of changes in the level of blood viscosity on the pressure drop is more significant at early stages of atherosclerosis (mild degrees of stenosis). The comparison of the pressures measured before and after stenoses with the ones for healthy arteries is recommended to be used to estimate the severity of vessel constriction, which can be helpful in early detection of atherosclerosis via a non-invasive diagnostic procedure.

Introduction

Cardiovascular diseases are considered as one of the main causes of death worldwide. Among these diseases, atherosclerosis caused by plaque deposition on the arterial wall, known as stenosis, leads to the obstruction of the blood flow. These plaques, comprised of lipid, calcium, bad cholesterol (LDL), and also a specific type of white blood cells (macrophage cell), can occupy almost the entire cross section of the artery and cause difficulty for the heart to provide oxygenated blood to the various organs. The power which is required by the heart to supply the organs with a uniform blood flow rate can be determined by the pressure drop throughout the artery. The degree of stenosis, representing the area reduction percentage of a normal artery because of the plaque deposition, directly influences the amount of pressure drop. Higher pressure drop means higher heart muscle work rate to provide the same blood flow to the various organs.

Pressure drop is increased in the presence of flow separation and transitional flow after the stenosis. The pressure drop can also be changed due to the presence of the pulsatile flow introducing flow acceleration and deceleration during a cardiac cycle which is neglected in the steady flow assumption. Hence, it is necessary to study the pulsatile blood flow inside the blood vessels to observe the pressure drop and flow patterns before and after the constriction [9]. Although blood flow in the human arterial system is predominantly laminar, transition to turbulence can also be observed because of the fluctuations happening at different arterial locations. The transitional flow causes a higher velocity gradient and consequently higher values of wall shear stress. Higher shear stress leads to a higher pressure drop inside the artery due to the stenosis. One of the susceptible locations for laminar transitional flow is near the stenotic regions. Flow at and after the stenosis will become transitional even at lower flow velocities compared to a healthy artery. Furthermore, stenosis in the artery results in changes in the blood flow patterns. The flow patterns in models of a normal internal carotid artery (ICA) as well as two diseased ICAs with 50% and 70% degrees of stenosis were studied by Kefayati and Poepping [7]. Blood flow was uniform in both internal and external carotid arteries (both left and right sides of bifurcation) for the healthy artery. Reversed flow patterns were also observed along the inner wall of both diseased ICA models.

To observe the laminar flow transition in a channel, Khair et al. [8] performed direct numerical simulation (DNS) modelling to investigate the pulsatile flow in a constricted rectangular channel. The peak value of turbulent kinetic energy (TKE), representing the transition to turbulence point, is located after the constricted section for all cases. For higher values of Reynolds number and higher degrees of stenosis, the transition location moved closer to the constricted location. After reaching the transitional point, the TKE value gradually reduces which indicates that the flow is re-laminarised.

Blood in large arteries (diameter > 0.5 mm) is considered as a Newtonian fluid where the Reynolds number is high [11]. Furthermore, since the blood behaves as a Newtonian fluid at shear rates greater than 100 s^{-1} [6] and considering that the shear rates applied to the coronary arteries are larger than 100 s^{-1} [14], blood behaviour in the coronary arteries is considered as Newtonian fluid. The blood viscosity can be interpreted as the resistance of blood to flow through the arteries. The higher the viscosity, the more difficult it is for the blood to pass through and consequently the heart needs to work harder to provide the same flow rate compared to the normal viscosity blood case. Blood consists of different components including red (about 45 volume percentage) and white (about 0.7 volume percentage) blood cells and platelets (about 54.3 volume percentage). Whole blood viscosity is mostly dependent on the red blood cells since the white blood cells and platelets do not have a great influence on the blood viscosity [12]. Hematocrit is a volume percentage of red blood cells in the blood. The change in the level of hematocrit causes the change in the blood viscosity. It has been hypothesised that increasing the levels of blood viscosity may lead to an increase in the cardiovascular diseases such as atherosclerosis and thrombogenesis related diseases [10]. Furthermore, low levels of hematocrit, i.e. anemia, may cause some serious symptoms such as fatigue, shortness of breath and fast heartbeat. Thus, it is of great importance to study the effect of blood viscosity on the pressure drop through a diseased coronary artery.

In this study, the blood flow-pressure drop relation across a diseased coronary artery with different degrees of stenosis and different levels of blood hematocrit are investigated. An experimental setup was designed and built to measure the flow rate and pressure drop before and after the diseased part of the coronary artery. Furthermore, a realistic physiological pulsatile flow profile is applied to mimic the blood flow inside the coronary arteries.

Experimental Methodology

The schematic diagram of a diseased coronary artery is presented in Figure 1. The diseased coronary artery is modelled as a rigid asymmetric spherical constriction inside a rigid pipe. Different degrees of stenosis including 11.2%, 39.1%, 56.5%, 65.4%, and 74.2% cross section area reduction are considered to address different amounts of plaque deposition at susceptible locations of coronary arteries. The inside diameter of the model,

D, is 6.1 mm corresponding to almost double the diameter of coronary arteries. Since the flow inside the artery is pulsatile, a 10-D length is considered both before and after stenosis part to account for the beginning and end effects.

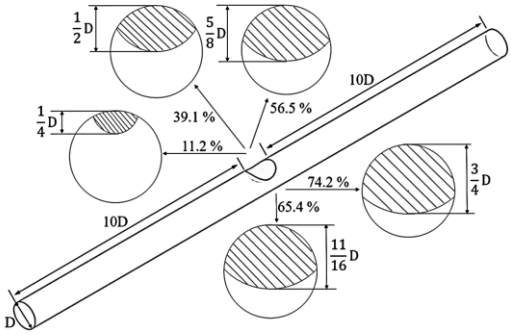


Figure 1. Schematic diagram of 3D asymmetric diseased coronary artery with different degrees of stenosis shown with the percentage of degrees of stenosis.

Figure 2 displays the experimental rig and its components: (1) programmable centrifugal pump (RS pro direct coupling centrifugal pump) and its controller (RS pro process pump controller for brushless micro-pump pump), (2) flow sensor (Omega, FLR1000), (3) 3D-printed settling chamber, (4) pressure sensor (UNIK 5000 silicon pressure sensor), (5) 3D printed diseased artery model (Zortrax M300), (6) valve, (7) water reservoir, and (8) computer, power supply and DAQ card (NI USB-6211). It is worth mentioning that a long acrylic pipe (about 2 m) is inserted before the test section and after the settling chamber to allow the pulsatile flow inside the system to become fully developed.

Three different working fluids were chosen for this experiment; one of them matching the typical blood viscosity (3.2 mPa.s), the second representing the low hematocrit related diseases (with viscosity about half of the typical blood viscosity, 1.8 mPa.s) and another one to account for high levels of hematocrit (with viscosity about double of the typical blood viscosity, 6.4 mPa.s). In order to obtain fluids with the above-mentioned viscosities, a mixture of distilled water and glycerine was employed based on the classic Grunberg and Nissan [4] liquid mixture equation

$$\ln \mu_{lmix} = \sum_{i=1}^{N} x_i \ln \mu_{li} \qquad (1)$$

where μ_{lmix} is the viscosity of the liquid mixture, μ_{li} is the viscosity of the pure fluid component and x_i is the mole fraction of component i in the liquid mixture. The three working fluids were made from distilled water and glycerine portions according to equation (1). The viscosity of the final mixture was then determined by testing the samples in an AntonPaar MCR 301 Rheometer. Accuracy of the measured viscosities of the mixture was $\pm 1.5\%$. All experiments and viscosity calculations were performed at room temperature (23°C). The blood viscosity in the body temperature (37°C) is modelled with the glycerol-water mixture at the room temperature.

The pulsatile waveform, representing the blood flow inside the coronary artery, is shown in Figure 3 [5]. This waveform, as an inlet velocity boundary condition, is modelled using the first 4 Fourier modes of Fast Fourier Transform (FFT) which accurately represents the original waveform. The input voltage of the pump is controlled using a data acquisition card to simulate the physiological velocity waveform of the coronary artery as represented in Figure 3. A slight difference can be observed between the required inlet profile signal and the one the pump creates. The maximum deviation between the achieved signal and the original inlet profile signal is 1.6% for the low hematocrit blood model, 1.8% for the blood mimicking fluid and 2.3% for the high level of hematocrit fluid model.

Two pressure sensors are used to measure the pressure differences before and after the test section. The pressure transducers have a full scale accuracy of 0.04% and the flow meter has a measurement accuracy of 1%.

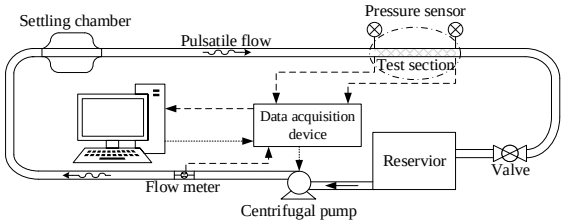
The mean flow rate changes from the resting blood flow rate ($1.1\ ml/s$) to hyperemic flow rate ($3.9\ ml/s$) [3]. Because of the limitation of the centrifugal pump, the resting blood flow condition was slightly higher in this experiment (slightly above $100\ ml/min$).

The Womersley number, a dimensionless parameter governing the pulsatile flow, is defined as

$$\mathrm{Wo} = \frac{D}{2}\sqrt{\frac{2\pi}{T}\frac{\rho}{\mu}} \qquad (2)$$

where T is the a cycle time period and ρ is the fluid density. The blood density is considered to be 1060 kg/m^3 [1]. The Womersley number was set to be 1.9, representing the value for coronary artery [9], for all experiments. In order to keep the Womersley number constant and since viscosity changes for different fluid models, the time period is changed accordingly. For the blood mimicking fluid, the time period is 5.54 s, for the fluid with a lower viscosity ($\mu = 1.8\ mPa.s$) the time period is 9.85 s and finally for the higher viscosity fluid model ($\mu = 6.4\ mPa.s$) the time period becomes 2.77 s.

a)



b)

Figure 2. a) Schematic of the test setup and b) Experimental test setup: (1) centrifugal pump; (2) flow sensor; (3) settling chamber; (4) pressure sensor; (5) 3D-printed test section; (6) valve; (7) water reservoir; (8) computer, power supply and DAQ card.

Results and discussion

The pressure difference before and after the stenosis of a diseased coronary artery model with different degrees of stenosis ranging from 11.2% to 74.2% degrees of stenosis for working fluids with different viscosities is displayed in Figure 4. The mean flow rate varies from about 110 ml/min to 280 ml/min for all cases. A linear relation between the flow rate and pressure drop is observed for all cases which can be explained based on Darcy-Weisbach equation that is valid for laminar pipe flow without any constrictions:

$$\frac{\Delta P}{L} = \frac{128}{\pi} \cdot \frac{\mu \dot{Q}}{D^4} \qquad (3)$$

where ΔP is the pressure drop, L is the pipe length and $\dot{Q}$ is the volumetric flow rate. Based on the Darcy-Weisbach equation, the pressure drop is related to viscous effects which means that the higher the flow rate, the higher the pressure drop (As displayed by black dash line in Figure 4). To account for the

effect of the pulsatile flow regime and constrictions inside the pipe, the following equation was proposed by Young et al. [15]:

$$\Delta P = \frac{K_v \mu}{D} V + \frac{K_t}{2}\left(\frac{A_0}{A_1} - 1\right)^2 \rho |V| V + K_u \rho L \frac{dV}{dt} \qquad (4)$$

where K_v, K_t and K_u are experimentally determined coefficients, A_0 is the area of the pipe with the diameter of D, A_1 is the cross sectional area of the stenosis, and V is the plane average flow velocity of the unobstructed pipe. The first term is almost the same as the Darcy-Weisbach equation which represents the friction loss effect. The second term denotes the pressure drop due to the non-linear changes in the cross section of the pipe because of the stenosis and the last term represents the differential pressure required to accelerate or decelerate the fluid inside the pipe. The difference between steady and pulsatile flow regimes in terms of pressure drop is represented using the last term of equation 4 which is related to the time-dependent nature of pulsatile flow.

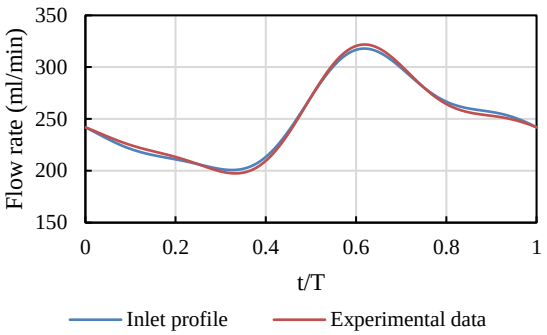


Figure 3. The physiological pulsatile inlet velocity boundary condition [5].

Due to the sudden change in the flow pattern because of the stenosis as well as an increase in the fluid velocity passing the stenosis, the turbulence structures start to grow after the constriction. The velocity fluctuations due to the constriction inside the pipe are intensified with an increase in the cross sectional area covered by the fat plaques in the artery. These turbulent structure cause a more significant pressure drop across the constrictions with higher degrees of stenosis (Figure 4). The Reynolds number range based on the diameter of the artery model and upstream velocity for different fluid viscosity is displayed in Figure 4. The Reynolds number immediately after the stenosis due to a sudden jump in the velocity increases considerably leading to the formation of turbulent structures. A remarkable jump in pressure drop is seen for the case with 74.2% degree of stenosis compared to the others, which was also observed by Young et al. [15] and Flanigan et al. [2]. The results show that an increase in the degree of stenosis to 75% increases the pressure drop dramatically, independent of the blood viscosity. The same trend exists for Figures 4(b) and 4(c) associated with different viscosities. Moreover, the pressure drop calculated from Darcy-Weisbach equation for the straight pipe without any constrictions is considerably lower than the other cases with different degrees of stenosis representing the effect of constriction in the flow path on the pressure drop.

The pressure drop for different degrees of stenosis and viscosities for both resting flow rate (120 ml/min, part a) and hyperemic flow rate (250 ml/min, part b) are displayed in Figure 5. According to Figure 5, the pressure drop increases with an increase in the degree of stenosis. The effect of the degree of stenosis on the pressure drop is more significant as the stenosis degree increases above approximately 60%. According to Figure 5(a), an increase in the degree of stenosis from 56.5% to 74.2% increases the pressure drop from 170 Pa to 342 Pa for $\mu = 6.4\ mPa.s$. Furthermore, for the hyperemic flow rate, the pressure drop increases from 315 Pa to 947 Pa with increasing the degree of stenosis from 56.5% to 74.2% (Figure 5(b)). The observed trend is in agreement with the results reported by Rotman et al. [13] which found that the pressure drop increases exponentially as the degree of stenosis increases in the presence of arterial compliance effect.

Furthermore, for a flow rate of 250 ml/min, the increase of viscosity from 1.8 to 6.4 mPa.s (more than 2 times higher), increases the pressure drop by 50 % for a 74.2% degree of stenosis while the increase of viscosity from 1.8 to 6.4 mPa.s increases pressure drop by more than 3 times for a 11.2% degree of stenosis. The same trend can be seen for the flow rate of 120 ml/min. Hence, apart from the flow rate, the effect of viscosity on pressure drop is more significant at lower degrees of stenosis.

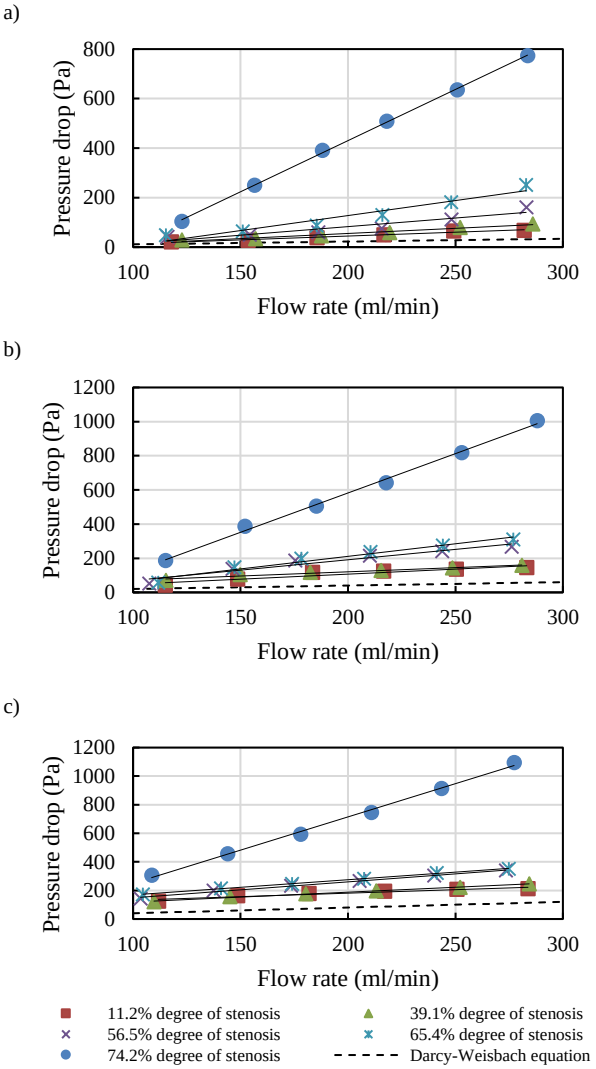
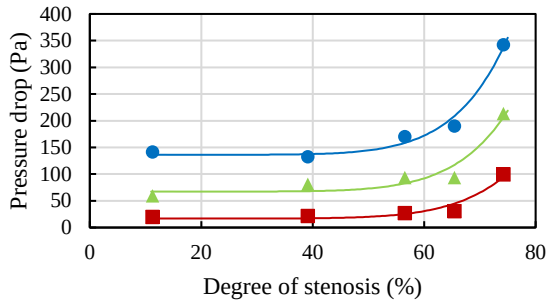


Figure 4. The pressure drop calculated from Darcy-Weisbach equation and measured across different degrees of stenosis in the pipe with flow rate change for different viscosities of a) 1.8 mPa.s ($200 < Re < 600$), b) 3.2 mPa.s ($115 < Re < 350$) and c) 6.4 mPa.s ($60 < Re < 180$).

Figure 6 presents the pressure drop at different flow rates for different levels of hematocrit in the blood for a 74.2% degree of stenosis. An increase in the levels of hematocrit which is equal to the increase in viscosity, increases the pressure drop. This means that the heart needs to work harder to push the blood through the semi-blocked artery for patients suffering from hyper-viscosity syndromes. For example, for the hyperemic flow rate (about 250 ml/min), an increase in viscosity from 1.8 to 6.4 mPa.s increases the pressure drop from about 600 Pa to 950 Pa.

a)



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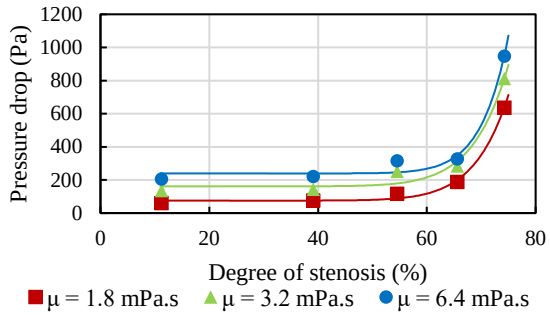

Figure 5. Pressure drop for different degrees of stenosis and viscosities for a) flow rate = 120 ml/min and b) 250 ml/min.

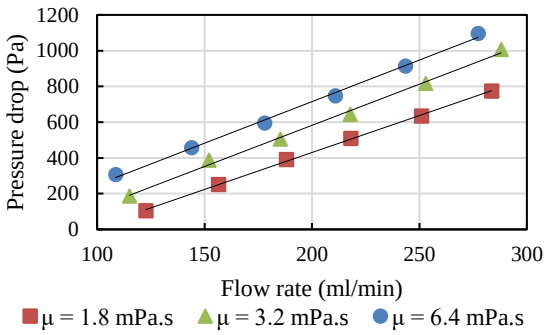

Figure 6. The pressure drop measured across the stenotic pipe with flow rate change for a 74.2% degree of stenosis.

Conclusion

The pressure drop has been measured inside a pipe model representing a coronary artery suffering from atherosclerosis with the change in the level of blood hematocrit. The effect of changes in the level of hematocrit on the pressure drop is more significant at early stages of atherosclerosis. On the other hand, at the more progressive levels of atherosclerosis, the pressure drop changes are more significant compared to the changes that occur due to the hematocrit level in the blood. The results of this study provide a better prediction of pressure drop and blood flow rate which can be applied to investigate heart muscle work rate and the required heart power.

Acknowledgement

Financial support for the project has been provided by the Australian Government Research Training Program and the University of Adelaide Scholarship. The authors would like to acknowledge the School of Mechanical Engineering and the Mechanical Workshop at the University of Adelaide. The authors acknowledge the financial support of the Australian Research Council.

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