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# A NUMERICAL STUDY ON THE VARIATION OF STATIC PRESSURES OF BLOOD NEAR THE STENOSIS OF CORONARY ARTERY

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## ABSTRACT

In this paper, the variation of static pressure of blood on the wall and average pressure of blood near the stenosis in a part of coronary artery is studied at different Re and also for different percent stenosis. The two-dimensional steady differential equations for conservation of mass and momentum is solved by finite difference method for Re ranges from 40 to 100 and percent stenosis from 20% to 50%. The effect of Re and percent stenosis on the variation of said static pressures is studied. From the study, it is revealed that for all the cases, higher the Reynolds number, higher is the concerned static pressure. No appreciable variation in the quantum of the said static pressures is noted during the variation of percentage of restriction. For all the cases, a sharp variation in average static pressure and wall pressure has also been noted at the zone of restriction.

**Keywords:** Stenosis, Average pressure, Wall pressure.

## 1. INTRODUCTION

The heart is the human body's hardest working organ. Throughout life, it continuously pumps blood with oxygen and vital nutrients through a network of arteries to all part's of the body's tissues. In order to perform the task of pumping blood to the rest of the body, the heart muscle itself needs a plentiful supply of oxygen-rich blood, which is provided through a network of coronary arteries. These arteries carry oxygen-rich blood to the heart's muscular walls (myocardium).

In the present days, the major causes of death of human being in developed and developing countries are identified as due to the cardiovascular diseases. Among the various types of cardio-vascular diseases, the disease related to coronary arteries (CAD) is considered to be an important one. It harms the myocardium in maintaining the proper functioning of heart.

Coronary artery disease (CAD) is the end result of a complex process called atherosclerosis, which is commonly called hardening of the arteries. Eventually the hardened artery becomes narrower, a condition known as stenosis. It is well known that the inner lining of the normal coronary artery is smooth and free from any blockages and obstructions. When too much cholesterol (lipid) or high level of triglycerides (fatty substances) or both persist in the blood, these fatty substances may begin to build along the inside of the artery walls as fatty streaks (atheroma). The streaks are minimally raised initially and thus do not produce any major blood flow restriction to the myocardium causing no symptoms to feel it. As the hardening process and narrowing continues, the blood flow slows and prevents sufficient oxygen-rich blood from reaching the heart. These narrow and the inelastic arteries not only slow down the blood flow but also become vulnerable to injury and tears.

It is well established that up to even a 50% reduction in radius of the coronary artery, the flow of blood will not be impaired in the myocardium but it will reduce maximum flow capacity which can lead to ischemia-induced chest pain during exertion. This is called chronic stable angina. Radius reduction of more than 75% can significantly reduce resting blood flow, depending upon the degree of collateralization. This can lead to chronic myocardial hypoxia. Therefore, generally, below the 50% reduction in radius of coronary artery, no symptoms can be felt by human being carrying this disease. When the symptom is felt, the reduction of the radius of coronary artery becomes more than 50%, which is detected by angiography. To remove this restriction, angioplasty is advised by the doctors because the disease can not be removed with the help of medicine therapy. Therefore, if any mathematical tool is used to predict the extent of blockage in the coronary artery in initial stage of the disease, the medicine or alternative therapy may be prescribed by the medical practitioners to avoid from going to angiography or angioplasty or bypass surgery.

A number of researchers have worked either experimentally or numerically on different types of arteries. Among them, M. Siouffi et al. [1] have studied the post stenotic velocity flow field corresponding to oscillatory, pulsatile and physiological flow web forms.

They have measured two-dimensional velocity in a 75% severity stenosis using a pulse Doppler ultrasonic velocimeter. It is concluded by the authors that beyond the influence of the flow parameters such as the Reynolds number and the frequency parameter, velocity field highly depends on the flow waveform particularly downstream from the stenosis. T. Shipkowit et al. [2] have numerically predicted the three dimensional flow through a rigid model of the human abdominal aorta comprising of iliac and renal arteries using Navier-Stokes equations for an incompressible Newtonian fluid. They have introduced a purely axial velocity profile (parabolic) at the entrance of the model. G. R. Zendehbudi and M. S. Moayeri [3] have made a numerical solution for a physiological pulsatile flow as well as for an equivalent simple pulsatile flow through an axisymmetric rigid stenosed artery (61% area reduction) considering flow as laminar. A. Kirpalani et al. [4] have developed a technique to construct a rigid flow model from a cast of a human right coronary artery to characterize the velocity and wall shear stress patterns using a laser photometric method. They have used steady flow conditions at Reynolds number of 500 and 1000 as well as unsteady flow with womersley parameter 1.82 and Reynolds number 750. D. Tang et al. [5] have quantified the compressive conditions of arteries with high-grade stenoses, due to negative transmural pressure caused by high velocity flow passing through the stenoses. They have indicated that severe stenoses cause critical flow condition such as negative pressure and high and low shear stresses, which may be directly related to artery collapse. J. S. Stroud et al. [6] have investigated the blood flow in the presence of significant plaque deposits and evaluated the influence of factors such as stenosis morphology and surface irregularity. They have solved the equations of motion using a finite volume technique for three dimensional, unsteady flows in the stenotic vessels for an incompressible, Newtonian fluid. M. S. Moayeri and G. R. Zendehbudi [7] have made a model of pulsatile blood (Newtonian fluid) flow by using measured values of the flow rates and pressure for the canine femoral artery. They have concluded that deformability of the wall causes an increase in the time average of pressure drop. N. C. Chesler and O. C. Enyinna [8] have quantified the effect of hemodynamic pressure, flow and wave form perturbation on the deposition of protein sized particles in porcine carotid arteries.

Although many investigators have studied numerically and experimentally the flow through several arteries but the study of the variation of static pressures near the stenosis for coronary artery has not been attempted so far. Hence in the present work, an attempt has been made to study the variation of average static pressure of blood and static pressure of blood on the wall near the stenosis in a part of the coronary artery for Re ranges from 40 to 100 and also for different percent stenosis ranges from 20% to 50% respectively. From the study, it has been revealed that for all the cases, higher the Reynolds number, higher is the concerned static pressure. No appreciable variation in the quantum of the said static pressures has been noted during the variation of percentage of restriction for a particular value of Reynolds number. For all the cases, a sharp variation in average static pressure and wall pressure has also been noted at the zone of restriction.

## 2. MATHEMATICAL FORMULATION

### 2.1 Governing Equations

A schematic diagram of the computational domain is illustrated in Figure 1. The flow under consideration is assumed to be steady, two-dimensional and laminar. The fluid, i.e. blood, is considered to be incompressible and obeys Newton's law of viscosity. The following dimensionless variables are defined to obtain the governing conservation equations in the non-dimensional form;

Lengths: $x^* = x/W \quad , \quad y^* = y/W \quad ,$
$L_i^* = L_i/W \ , \ L_{ex}^* = L_{ex}/W$

Velocities: $u^* = u/V_1 \ , \ v^* = v/V_1$

Pressure: $p^* = \left(p + \rho g y\right)/\rho V_1^2$

With the help of these variables, the mass and momentum conservation equations are written as follows,

$$\frac{\partial u^*}{\partial x^*} + \frac{\partial v^*}{\partial y^*} = 0 \tag{1}$$

$$\frac{\partial\left(u^* u^*\right)}{\partial x^*} + \frac{\partial\left(v^* u^*\right)}{\partial y^*} = -\frac{\partial p^*}{\partial x^*} + \frac{1}{\mathrm{Re}}\left[\frac{\partial}{\partial x^*}\left(\frac{\partial u^*}{\partial x^*}\right) + \frac{\partial}{\partial y^*}\left(\frac{\partial u^*}{\partial y^*}\right)\right] \tag{2}$$

$$\frac{\partial\left(u^* v^*\right)}{\partial x^*} + \frac{\partial\left(v^* v^*\right)}{\partial y^*} = -\frac{\partial p^*}{\partial y^*} + \frac{1}{\mathrm{Re}}\left[\frac{\partial}{\partial x^*}\left(\frac{\partial v^*}{\partial x^*}\right) + \frac{\partial}{\partial y^*}\left(\frac{\partial v^*}{\partial y^*}\right)\right] \tag{3}$$

where, the flow Reynolds number, $\mathrm{Re} = \rho V_1 W/\mu$.

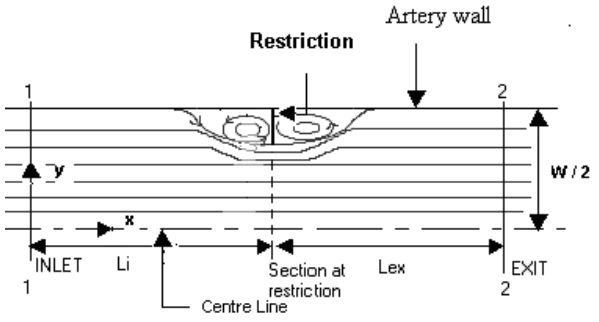


Fig 1. Scematic diagram of the computational domain

### 2.2 Boundary Conditions

Four different types of boundary conditions are applied to the present problem. They are as follows,

1. At the walls: No slip condition is used, i.e., $u^* = 0$, $v^* = 0$.
2. At the inlet: Axial velocity is specified and the transverse velocity is set to zero, i.e., $u^* = specified$, $v^* = 0$. Fully developed flow condition is specified at the inlet, i.e., $u^* = 1.5\left[1 - \left(2y^*\right)^2\right]$.
3. At the exit: Fully developed condition is assumed and hence gradients are set to zero, i.e., $\partial u^* / \partial x^* = 0$, $\partial v^* / \partial x^* = 0$.
4. At the line of symmetry: The normal gradient of the axial velocity and the transverse velocity are set to zero, i.e., $\partial u^* / \partial y^* = 0$, $v^* = 0$.

### 2.3 Numerical Procedure

The partial differential equations (1) – (3) are discretised by a control volume based finite difference method. Power law scheme is used to discretise the convective terms [9]. The discretised equations are solved iteratively by SIMPLE algorithm, using line-by-line ADI method. The convergence of the iterative scheme is achieved when the normalised residuals for mass and momentum equations summed over the entire calculation domain fall below $10^{-8}$.

In the present computation, the flow is assumed to be fully developed at inlet and exit and hence, the inlet and the exit are chosen far away from the restriction. For all the calculations, the inlet length and the exit length are considered to be 50 each. The distribution of grid nodes is non-uniform in both co-ordinate directions allowing higher grid node concentrations in the region close to the wall and restriction of the considered domain.

The grid independence test has been carried out with different grid densities for $\mathrm{Re} = 100$ and PR=50%. In accordance with the grid independence test results, the numerical mesh comprised of 85 × 31 grid nodes in each of the inlet section and exit section in *x* and *y* directions respectively have been considered in the numerical computations during the study.

## 3. RESULTS AND DISCUSSION

The important results of the present study are reported in this section. The parameters during the study are identified as,

(i) Reynolds number, $40 \le \mathrm{Re} \le 100$.

(ii) Percentage of restriction, $20 \le PR \le 50$.

(iii) Inlet velocity distribution – fully developed.

### 3.1 Variation of Average Static Pressure Near the Stenosis

The average static pressure of blood at any section of the concerned coronary artery may be considered to be an important parameter in assessing the extent of growth of stenosis i.e. restriction in that part. Therefore, in this section, an attempt has been made to study the effect of Reynolds number and percentage of stenosis separately on the average static pressure near the stenosed zone. In the present work, the average static pressure at any cross-section is determined by the following expression:

$$p_{av} = \frac{\int p dA}{\int dA} \qquad (4)$$

The effect of different Reynolds number and different percentage of restriction on the variation of the average pressure at the upstream and down stream of the stenosis is shown in Figure 2 and Figure 3 respectively.

Figure 2 shows the variation of average static pressure for three different Reynolds number namely 60, 80, and 100 for a particular value of restriction of 50%.

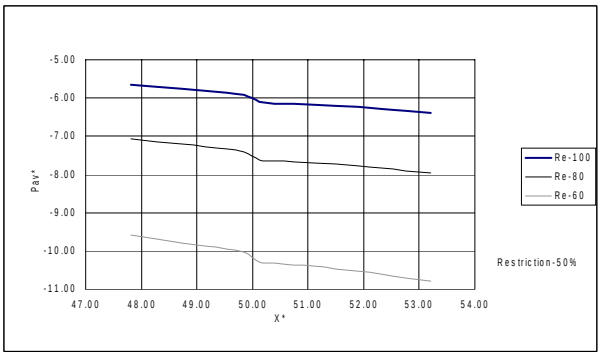


Fig 2. Effect of Reynolds number on the variation of average pressure

It is seen from Figure 2 that for all the Reynolds numbers, there is a sharp variation in average pressure near the stenosis and higher the Reynolds number, higher is the average pressure near the said zone. It is also noted that the quantum of variation in average pressure at that zone decreases with increase in Reynolds number.

Figure 3 shows the variations of the same for four different percentage of restriction namely 20%, 30%, 40%, and 50% at a particular Reynolds number of 100.

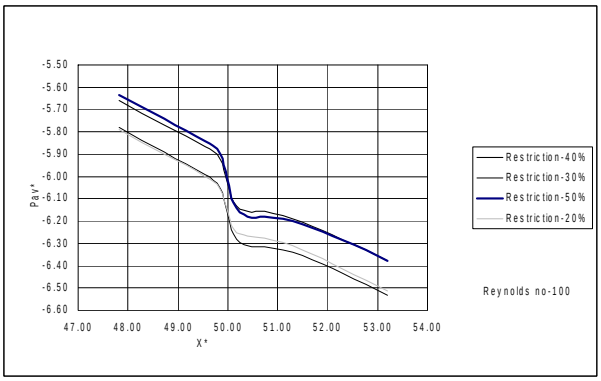


Fig 3. Effect of percentage of restriction on the variation of average pressure

In Figure 3, for all the cases, a sharp drop in average pressure at the restriction is noted. Just after the restriction the wall pressure has increased slightly. It can be attributed due to higher diffusion of kinetic energy of blood due to expansion at the down stream of the restriction. After that average pressure reduces gradually obviously due to friction. No appreciable variation in the magnitude of the average static pressure has been noted during the variation of percentage of restriction for a particular value of Reynolds number. It is also found that for higher percentage of restriction, average pressure is higher at the upstream and is lower at the downstream of the stenosis.

### 3.2 Variation of Wall Pressure Near the Stenosis

The normal and shear stresses of blood near the wall of an artery are considered to be the important factors for the genesis of formation and growth of atherosclerosis on the inner wall of the concerned artery. Since, wall pressure is needed in assessing the normal stress, therefore, in this section, an attempt has been made to study the effect of Reynolds number and percentage of stenosis on the wall pressure near the zone of stenosis. The effect of different Reynolds number and different percentage of restriction on the variation of the wall pressure at the upstream and down stream of the stenosis is shown in Figure 4 and Figure 5 respectively.

Figure 4 shows the variation of wall pressure for three different Reynolds number namely 60, 80, and 100 for a particular value of restriction of 50%.

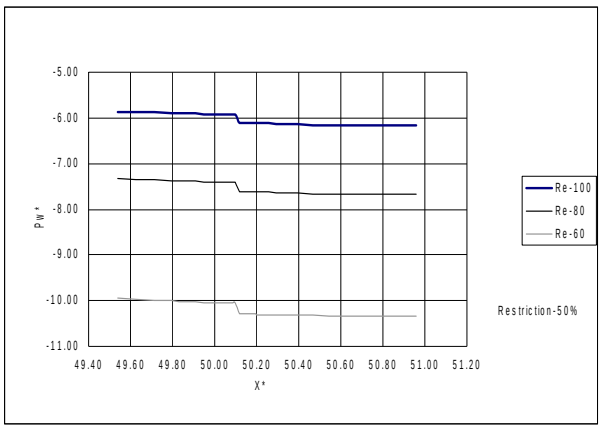


Fig 4. Effect of Reynolds number on the variation of wall pressure

The general trend of the curves of Figure 4 is noted to be more or less same as that of the cases of average static pressure, i.e., for higher Reynolds number, higher is the wall pressure near the said zone and the quantum of variation in wall pressure at that zone decreases with increase in Reynolds number.

Figure 5 shows the variations of wall pressure for four different percentage of restriction namely 20%, 30%, 40%, and 50% at a particular Reynolds number of 100.

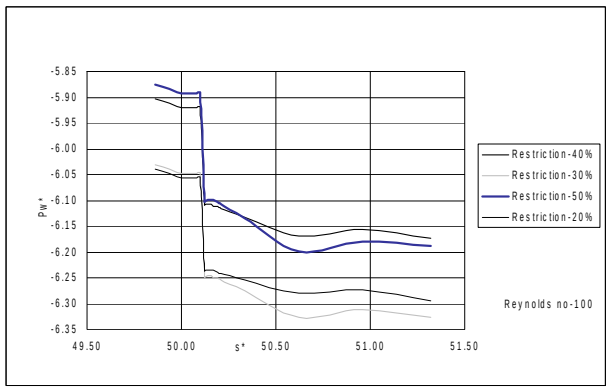


Fig 5. Effect of percentage of restriction on the variation of wall pressure

In this case, a sharp variation wall pressure is noted at the zone of restriction. It is also observed that as the percentage of restriction increases, the variation in the magnitude of the wall pressure increases. Like the cases of average pressures, it is also noted here that wall pressure is higher at the upstream and is lower at the downstream of the stenosis in the case of higher percentage of restriction.

## 4. CONCLUSIONS

In the present work, a numerical study on the variation of average static pressure of blood and static pressure of blood on the wall near the stenosis in a part of the coronary artery has been carried out. The effects of important parameters like flow Reynolds number, Re, and percentage of restriction on the variation of said static pressures have been investigated, and this leads to the following important observations:

(l) There is a sharp variation in average pressure and wall pressure near the zone of stenosis.
(2) For higher Reynolds number, higher is the average pressure and wall pressure at the upstream and downstream of the stenosis.
(3) In the case of higher percentage of restriction, the said static pressures are noted to be higher at the upstream and lower at the downstream of the stenosis.

## 5. REFERENCES

1. Siouffi, M., Deplano, V. and Pelissier, R., 1998, “Experimental Analysis of Unsteady Flows Through a Stenosis”, Journal of Biomechanics, 31:11-19.
2. Shipkowit, T., Rodgers, V. G. J., Frazin, L. J. and Chandran, K. B., 1998, “Numerical Study on the Effect of Steady Axial Flow Development in the Human Aorta on Local Sheer Stresses in Abdominal Aortic Branches”, Journal of Biomechanics, 31:995-1007.
3. Zendehbudi, G. R. and Moayeri, M. S., 1999, “Comparison of Physiological and Simple Pulsatile Flow Through Stenosed Arteries’, Journal of Biomechanics, 32:959-965.
4. Kirpalani, A., Park, H., Butany, J., Johnston, K. W. and Ojha, N., 1999, “Velocity and Wall Shear Stress

Patterns in the Human Right Coronary Artery", Journal of Biomechanical Engineering, 121:370-374.

5. Tang, D., Yang, J., Yang, C. and Ku, D. N., 1999, "A Non Linear Axisymmetric Model with Fluid - Wall Interaction for Steady Viscous Flow in Stenotic Elastic Tubes", Journal of Biomechanical engineering, 121:494-501.
6. Stroud, J. S., Berger, S. A. and Saloner, D., 2000, "Influence of Stenosis Morphology on Flow Through Severely Stenotic Vessels: Implications for Plaque Rupture", Journal of Biomechanics, 33:443-455.
7. Moayeri, M. S. and Zendehbudi, G. R., 2003, "Effects of Elastic Property of the Wall on Flow Characteristics Through Arterial Stenoses", Journal of Biomechanics, 36:525-535.
8. Chesler, N. C. and Enyinna, O. C., 2003, "Particle Deposition in Arteries Ex Vivo: Effects of Pressure, Flow and Wave Form", Journal of Biomechanical Engineering, 125:389-394.
9. Patankar, S. V., 1980, *Numerical Heat Transfer and Fluid Flow*, Hemisphere Publication.

## 6. NOMENCLATURE

| Symbol | Meaning | Unit |
|---|---|---|
| A | Area at any section | ($m^2$) |
| $L_i$ | Inlet length(i.e.,length between inlet section and restriction) | (m) |
| $L_{ex}$ | Exit length(i.e., length bet restriction and exit section) Pressure | (m) |
| p | Static pressure | ($N.m^{-2}$) |
| PR | Percentage of restriction | % |
| $p_{av}$ | Average pressure | ($N- m^{-2}$) |
| $p_w$ | Wall pressure | ($N - m^{-2}$) |
| Re | Reynolds Number | - |
| s | Distance along the wall | (m) |
| u | Velocity in x-direction | ($m-s^{-1}$) |
| v | Velocity in y-direction | ($m-s^{-}$) |
| $V_1$ | Average velocity in x-direction at inlet | ($m-s^{-}$) |
| W | Diameter of the artery | (m) |
| x,y | Cartesian co-ordinates | - |
| $\rho$ | Density | ($kg\ m^{-3}$) |
| $\mu$ | Dynamic viscosity | ($kg\ m^{-1}s^{-1}$) |