

### 3.D Analysis of Blood Flow in Consecutive Stenoses of Coronary Artery

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#### ABSTRACT

Blood flow in the coronary artery with consecutive stenoses has been modeled numerically in the present study and its applications to blood diseases have been inspected. Considering the importance of the artery stenosis, the distributions of the wall shear stress (WSS) in the consecutive stenoses of the artery have been numerically and theoretically investigated in this paper. Angiography results in different studies show that there usually are several stenoses along the coronary artery; accordingly, the analysis of the behavior of the blood flow around the consecutive stenoses with 40, 50, 60 and 75 percents has been focused in this study. This paper presents a comparative study of the different non-Newtonian viscosity models effects. Besides, the Newtonian model has been compared with Casson, Carreau, Powerlaw and non-Newtonian Powerlaw models in different percentages and distances of the stenoses.

Large differences are found in the WSS magnitude distributions in the arteries with different percentage stenoses, leading to the conclusion that it is not possible to make generalizations based on the study of a single percentage of the stenosis. There is a strong correlation between WSS and critical times of the cardiac cycle and a weaker correlation between the distances of the stenoses and extremes of WSS. Also, this research shows that Casson model is the most reliable approach for the blood viscosity in transient flow.

**KEYWORDS:** non-Newtonian model, wall shear stress (WSS), coronary artery, consecutive stenoses.

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#### Nomenclature

P	poise
$\mu$	blood viscosity
V	velocity
$\rho$	density
t	time
$\rho \vec{g}$	gravity force
$\vec{F}$	external body force
$\tau$	stress tensor
WSS	wall shear stress
$r_0$	radius of the artery
$\delta$	radius of the stenosis section
$z_0$	half of the stenosis lengths
$I_l$	local non-Newtonian importance factor
$\mu_\infty$	Newtonian viscosity
m	meter
s	second

#### INTRODUCTION

Common behavior of the blood flow is altered and went out from the regular state in the face of stenosis. High values of the stress around the stenosis point areas that cause the serious destruction of the artery wall and extensive alteration in hemodynamic behavior is produced due to the strong flows. Therefore, this disease can be prevented by the determination of the effective causes in the stenosis formation or its intensification through the arteries. Sudden changes in the geometry or flow velocity of the arteries, stenosis intensity, distances between two stenoses and number of stenoses can be mentioned as the most important causes.

Atherosclerosis is an artery disease in which plugs formation and subsequently artery wall thickness increasing cause the constriction of the artery; therefore, with decreasing the blood flow to the below organs, these organs probably will be damaged seriously or partly. High levels of plugs formation and artery stenosis make

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serious problems which need for some special clinical operations. Bypass-Grafting operations, angioplasty with balloon and stent implant are the most important clinical operations that are used to remove artery stenosis [1, 2].

WSS distributions in four different coronary arteries in transient and steady state flows were investigated by Janston and his colleagues during 2004- 2005 [3, 4]. Liu has considered the effects of the Reynolds number on flow model for right coronary artery with single stenosis [5]. Generally, the properties of the blood are similar to the Newtonian fluids in high strains (above  $100S^{-1}$ ) [6]. Jeisent's study has shown the effects of the various artery geometries and the models of the blood viscosity on the distribution of the WSS [7]. Gerlek and his colleagues in 2002 have studied the behavior of the blood flow in the arteries with more than one stenosis [8]. Researches show that artery stenosis turn to a serious problem if it exceeds 50% [8, 9]. Also, according to angiography results, there are several stenoses along the coronary artery [3, 9]. Therefore, in this study, the analysis of the behavior of the blood flow around the consecutive stenoses with 40, 50, 60 and 75 percents is investigated. The model of 40% is chosen to consider the state of below critical point, and the models of 50%, 60% and 75% are selected to consider the states of critical point and above critical point. The blood flow is supposed as a laminar, incompressible, unsteady, and 3 dimensional flow; furthermore, the velocity is considered pulsatile (Fig. 1) [4]. This study looks at blood flow in Newtonian and four different non-Newtonian models, which have been considered the most common models for blood flow in various studies [3, 4, 7 and 10]. Newtonian and four non-Newtonian models of Casson, Carreau, Powerlaw and non-Newtonian Powerlaw are used to study the blood flow at coronary artery.

For analyzing and simulating, commercially available software package (Gambit & Fluent) has been employed, and as described previously (Corney et al.,200 1,2004; Johnston et al.,2004 ),coronary artery meshes were reconstructed from biplane angiograms, from which the centreline and radius of the artery along the centerline were extracted. The mesh was completed by creating a mesh of the entrance plane and extruding this mesh along the centerline taking into account variations in radius. The number of computational grid cells for each simulation is about 50,000. The boundary walls have been considered constant; moreover, inlet flow and outlet pressure have been supposed as a developed flow and zero respectively.

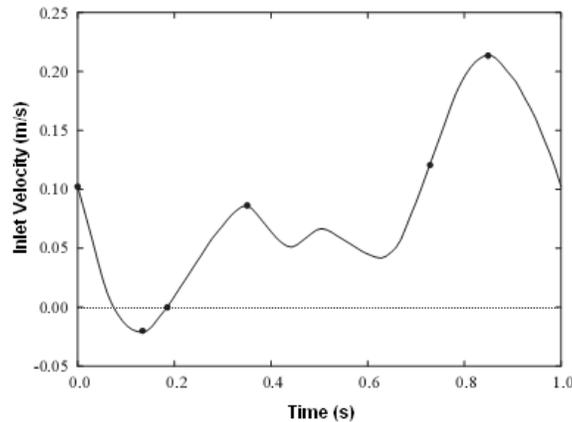


Fig. 1: The inlet velocity boundary condition as a function of time [4]

**Simulation equations**

The blood flow in the right coronary artery is modeled by Nover-Stocks equation [4]:

$$\rho \left( \frac{\partial V}{\partial t} + V \cdot \nabla V \right) = -\nabla \tau - \nabla P \tag{1}$$

The continuity equation for an incompressible flow is expressed below [4]:

$$\nabla \cdot V = 0 \tag{2}$$

Application of the Nover-Stocks equation in this form permits to use each non-Newtonian model for modeling. The momentum equations for fluid are modeled below [10]:

$$\frac{\partial}{\partial t} (\rho \vec{V}) + \nabla \cdot (\rho \vec{V} \cdot \vec{V}) = -\nabla P + \nabla \cdot (\vec{\tau}) + \rho \vec{g} + \vec{F} \tag{3}$$

Where  $V$  ,  $t$  ,  $P$  ,  $\rho$  ,  $\tau$  ,  $\rho \vec{g}$  and  $\vec{F}$  are the blood flow velocity, time, pressure, density, stress tensor, gravity force, and external body force respectively. Then stress tensor is obtained from this formula [10]:

$$\vec{\tau} = \mu[(\vec{\nabla}V + \nabla\vec{V}^T) - \frac{2}{3}\nabla\cdot\vec{V}I] \quad (4)$$

Where  $\mu$  shows molecular viscosity and I shows unit tensor.

Considering the effect of different non-Newtonian models on the distribution of WSS in coronary arteries is one of the most important purposes in this paper. Therefore, the distribution of the WSS in Newtonian model is compared with four non-Newtonian models of Carreau, Casson, Power Low, and non-Newtonian Power Low. The details of these five models are presented in Table 1 [3, 4, 7, 10].

Table 1. Model of blood viscosity  $\mu$  given in Poise (1 P=0.1 Pa.s) as a function of strain

Blood Model	Effective Viscosity $\mu$
Newtonian	$\mu = 0.0345 p$
Carreau	$\mu = \mu_{\infty} + (\mu_0 - \mu_{\infty})[1 + (\lambda\dot{\gamma})^2]^{-\frac{(n-1)}{2}}$ $[\lambda = 3.313, n = 0.3568, \mu_0 = 0.56 p, \mu_{\infty} = 0.0345 p]$
Power Low	$\mu = \mu_0 (\dot{\gamma})^{n-1}$ $[\mu_0 = 0.035, n = 0.6]$
non-Newtonian Power Low	$\mu = \lambda  \dot{\gamma} ^{n-1} \lambda(\dot{\gamma}) = \mu_{\infty} + \Delta\mu \exp[-(1 + \frac{ \dot{\gamma} }{a}) \exp(\frac{-b}{ \dot{\gamma} })]$ $n(\dot{\gamma}) = n_{\infty} - \Delta n \cdot \exp[-(1 + \frac{ \dot{\gamma} }{c}) \exp(\frac{-d}{ \dot{\gamma} })]$ $[\mu_{\infty} = 0.035, n_{\infty} = 1.0, \Delta\mu = 0.25, \Delta n = 0.45, a = 50, b = 3, c = 50, d = 4]$
Cosson	$\mu = [(\eta^2 J_2)^{1/4} + 2^{-1/2} \tau_y^{1/2}]^2 J_2^{-1/2}$ $ \dot{\gamma}  = 2\sqrt{J_2}, \tau_y = 0.1(0.625H)^3, \eta = \eta_0(1-H)^{-2.5}$ with $\eta_0 = 0.012 P$ and $H = 0.37$

The differentials of the WSS expressed the tangential force entering into the artery wall can be obtained by using this equation [10]:

$$WSS = \mu(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i}) \quad (5)$$

**Stenosis geometry modeling**

The stenosis in this paper is modeled as 3-dimensional using the equation 6, and the diameter of the artery is determined 3 mm [5]. Various figures are suggested for modeling of stenosis geometry in different papers. For example, Chacaravarty showed the stenosis geometry as the Fig. 2 in 2000, and Fig. 3 illustrates cosine, smooth and irregular models represented by him [14, 15].

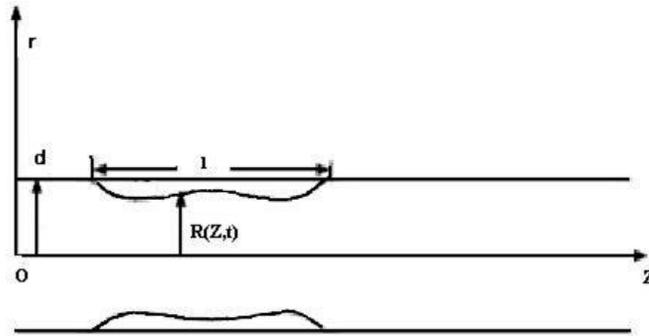


Fig. 2: The assumed geometric model for artery stenosis by Chacravarty in 2000 [14]

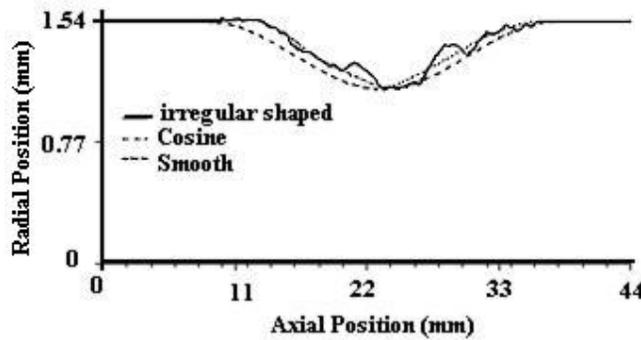


Fig. 3: Three assumed geometric models for artery stenosis by Chacravarty in 2005 [15]

To choose proper stenosis geometry modeling based on real condition, the results of different studies that have investigated the most prone points of coronary artery to form stenosis are used in this paper [11, 12, 13 and 14]. Therefore, the equation 6 introduced modeling by Han Jang is used to modeling the stenoses of coronary artery [16], and the diameter of the artery is considered 3 millimeters.

$$r(z) = \begin{cases} r_0 - \left(\frac{\delta}{2}\right) \left[1 + \cos\left(\frac{\pi z}{z_0}\right)\right] & \text{if } L_0 = 2z_0 \\ r_0 & \text{Otherwise} \end{cases} \quad (6)$$

Where  $r_0$ ,  $\delta$  and  $z_0$  are the radius of the artery in the non-stenotic section, the radius of the stenosis section and the half of the stenosis length respectively.

The percentage of stenosis is defined by below equation:

$$S \% = \frac{(d - 2 \times \delta)}{d} \times 100 \quad (7)$$

Fig. 4 illustrates the geometry of the equation 6 in R-Z coordination:

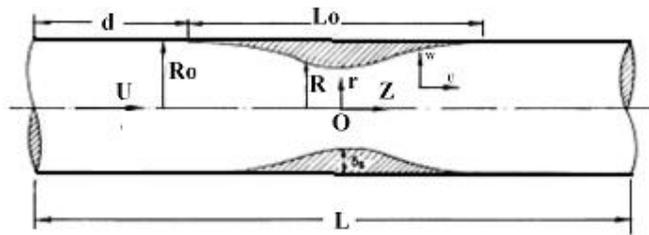


Fig. 4: The assumed geometry model that used by Hun Jung in

2004, used in this paper [5]

Considering some researches that show the left branch of the coronary artery is the most prone point to stenosis formation, the value of artery diameter in this paper is selected based on the left branch of the coronary artery [15].

Boundary conditions and defined properties for this geometry are below:

- a) Blood is fluid contained plasma 55% and blood cells 45% approximately. These ingredients have different densities (plasma density is  $1025 (Kg / m^3)$  and density of blood cells is  $1125 (Kg / m^3)$ ). In this study density of blood is considered  $1060 (Kg / m^3)$ .
- b) The boundaries have not any motion. Then, the velocity on the walls is supposed zero.
- c) Inlet flow is supposed developed and outlet pressure is considered zero.

## RESULTS AND DISCUSSION

According to prior experimental investigations and angiography results of this disease, it has observed that there are usually more than one stenosis along the coronary artery [3, 9]; however, a few studies has been done in the field of coronary diseases investigation with several stenoses. Therefore, in this study, it has been tried to inquire the effects of two stenoses on blood flow.

### 1. Wall shear stress (WSS) distribution

Sudden changes in the geometry or flow velocity of the artery, stenosis percentage, the distance of the stenoses than together, and the number of stenoses can be mentioned as the most important effective factors on WSS. Therefore, in each part of this paper, all the factors are considered constant, with the exception of a special factor to study its effect on blood flow and WSS, and the common values are devoted for constant considered factors.

#### 1.1. Time- dependent effect

Fig. 6 shows the WSS in the critical time of 0.85s for Carreau model with 3mm stenosis distance and 50% stenosis. The values of WSS in critical times of the cardiac cycle were regulated in Table 2. According to the results of this table, the highest values of the WSS in second stenosis are smaller than first one at all six critical times of the cardiac cycle. In other words, the first stenosis is more critical than second one in the all cardiac cycle. This can be explained that the central flow velocity of the second stenosis is greater than one in the first stenosis as Fig. 5, and considering this point that the mass flow through the first and the second stenoses is the same, the velocity near the wall in the second stenosis would be smaller than the first one. That is, the velocity changes related to the radius of the artery in second stenosis have smaller values, and due to the direct relation between the shear stress and the velocity changes, we observe the smaller values of WSS for the second stenosis.

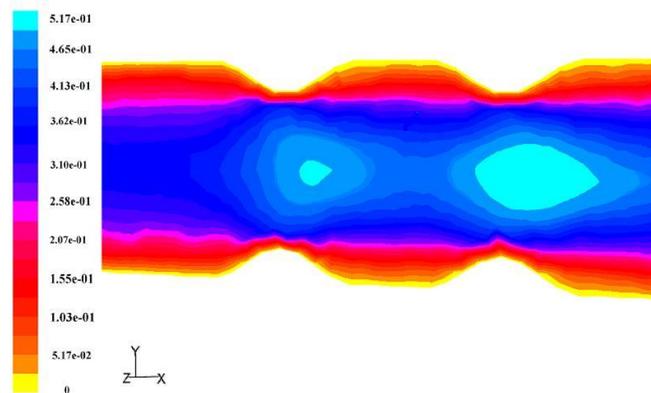


Fig. 5: Velocity at two consecutive stenoses for Carreau model, 50% stenosis and 3mm stenoses distance

Table 2: Maximum of WSS at first and second stenoses in critical times.

Critical times of cardiac cycle	0.01	0.13	0.18	0.35	0.72	0.85
WSS(Pa) at the first stenosis	8.26	2.17	0.532	7.24	11.03	25.7
WSS(Pa) at the second stenosis	5.68	1.10	0.127	4.74	7.16	15.4

Secondly, from this part, an impressive result that clarifies the effect of the consecutive stenoses together can be obtained. As it has explained above, the first stenosis makes the flow stream tend to the center of the artery, and this results in the decreasing of WSS in the second stenosis. Hence, it can be obtained that a light stenosis (low percentage stenosis) before intense stenosis (high percentage stenosis) makes low values of WSS compared to the lack of light stenosis. Therefore, existence a low percentage stenosis before high percentage stenosis decreases the risk of this disease.

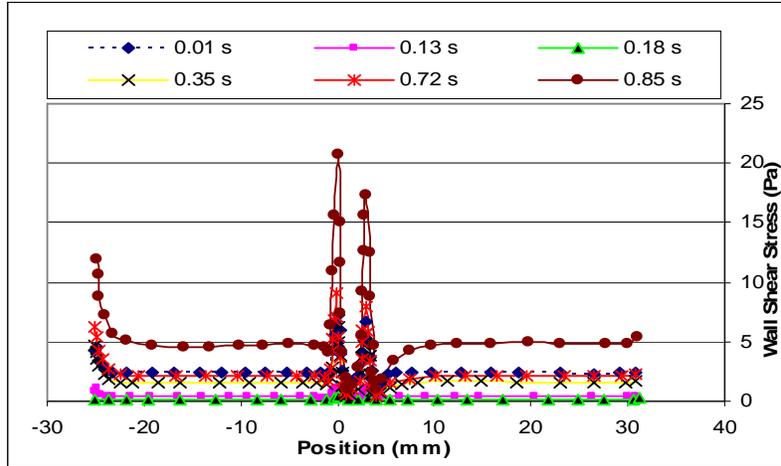


Fig. 6: WSS at critical times for Carreau model, 50% stenosis and 3mm stenoses distance

Thirdly, the maximum amount of WSS has a direct relation with inlet velocity of the artery. As it can be seen in Fig. 6, the maximum values of WSS vary according to inlet velocity of the artery. Considering the pulsatile graph (Fig. 1), at high velocities, the maximum value of the WSS increases; on the contrary, in some points with low inlet velocity, the maximum WSS decreases.

The maximum value of WSS in the stenosis becomes 8.26 while it has the velocity of 0.0847 m/sec at  $t=0.01$  sec. Whereas, with progressing toward  $t=0.13$ s that the inlet velocity gets  $-0.0195$ m/s, the regions of high WSS begin to dissipate and regions with low WSS begin to appear. At  $t=0.18$ s (with velocity of  $-0.0012$ m/s) that the inlet velocity approaches zero, the WSS come near to zero approximately. By progressing towards the times 0.35s, 0.72s and 0.85s with velocities 0.0854m/s, 0.1143m/s and 0.2144m/s, the regions of high WSS appear. The explained process has been showed in Fig. 6, and the high WSS in critical times were illustrated in Table 2.

**1.2. Stenoses distance effect**

The WSS in second stenosis would decrease whatever the stenoses distance has a low value. Fig. 7 shows WSS for Carreau model at  $t=0.85$ s in 50% consecutive stenoses with various stenoses distances.

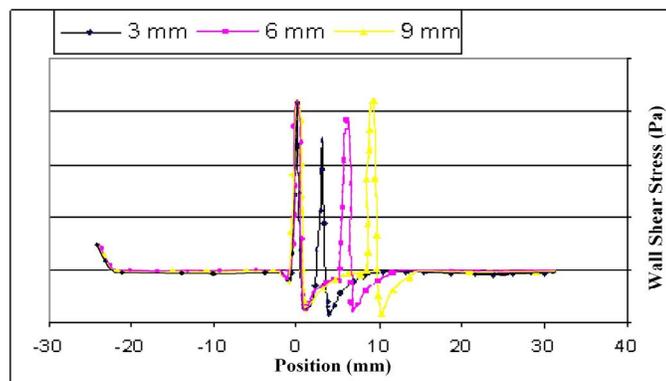


Fig. 7: WSS at various stenoses distances for Carreau model,  $t=0.85$ s and 50% stenosis

In the first stenosis, the high WSSs are same in all cases; however, these values depend on the distance of the stenoses in second stenosis. As mentioned, the first stenosis causes the flow to tend to the center of the artery, and it results in the decreasing of WSS in second stenosis. When the distance of two stenoses decreases, the blood flow does not have enough time to extend along the wall of the artery; therefore, the WSS of the second stenosis goes down, but when the distance of two stenoses increases, the blood flow that has tendency to the center line of the artery returns to the primary mode; consequently, the concentration of the flow disappears, and it causes the WSS increase in comparison with primary situation.

The highest values for WSS in second stenosis for 3mm, 6mm and 9mm stenoses distances are 17.4 Pa, 19.1 Pa, and 21 Pa respectively. These results confirm the above fact.

**13. Intensity of stenosis**

The WSS in stenoses and along the artery is increased by raising the stenosis percentage. Fig. 8 shows the WSS for Carreau model at  $t=0.85s$  in the 3mm stenoses distance with various stenosis percentages 40%, 50%, 60%, and 75%. This diagram shows that the WSS would increase whatever the stenoses percentage has a high value.

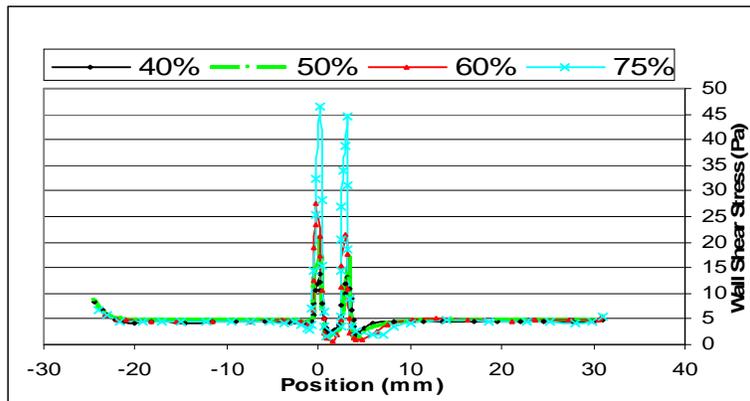


Fig. 8: WSS at various stenoses percentage 40%, 50%, 60% and 75% for Carreau model,  $t=0.85s$  and 3mm stenoses distance.

The increasing trend of WSS by intensity of stenosis has showed in table 3 by determining the critical WSS values in first stenosis at two critical times of 0.13S and 0.85S in different percentages. This table shows the importance of stenosis percentage and different critical times on values of WSS. The maximum value of WSS in the stenosis is getting 20.7 Pa while the percentage of the stenosis is 50%. By increasing the stenosis percentage, the maximum amounts get 24.7 and 46.4 Pa while the stenosis percentages become 60% and 70% respectively. The values of WSS increase more than 9.5 times by growing stenosis from 60% to 75% at critical time of 0.13S. The best conclusion that can be drawn is that the 75% stenosis is critical point and it can be harmful.

Table 3: Maximum of WSS at various percentages of stenoses in the certain point of stenosis

Percentage of stenosis	40%	50%	60%	75%
WSS(Pa) in 0.85 second	13.8	20.7	27.4	46.4
WSS(Pa) in 0.13 second	1.09	1.17	1.38	13.09

According to the mentioned results, there is a direct relation between the maximum value of WSS in the stenosis point and stenosis percentage; similarly, this relation is true along the artery (non-stenosis points), and the WSS along the artery would increase whatever the stenoses percentage has a high value. Table 4 shows the effect of stenosis percentage on WSS of other point of arteries. It can be observed that the changes of WSS are very low; therefore, it might be ignored.

Table4: Maximum values of WSS in different stenosis percentages along the artery (non-stenosis points)

Stenosis percentage	40%	50%	60%	75%
Amount of WSS(Pa) at 0.85 second	4.4	4.5	4.7	5.1
Amount of WSS(Pa) at 0.13 second	0.4	0.42	0.43	1.8

**1.4. Different Models**

In the majority of points from cardiac cycle, especially at high speed points (e.g. at t=0.85 Sec), the blood flow can be considered as a Newtonian flow. In this section, WSS is considered regarding some viscosity models such as Power Law, non-Newtonian Power Law, Carreau, and Casson.

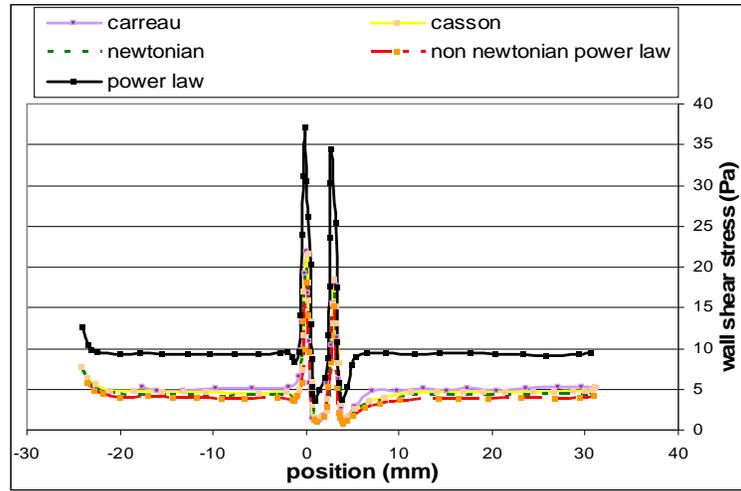


Fig. 9: WSS at 50% stenosis, 0.85 Sec, and 3mm distance in different models

Fig. 9 shows WSS at 50% stenosis, 0.85 Sec and 3mm distance in five models. It seems that the value of WSS in the Power Law model has significant difference with the others. Pondering about the Fig. 9, it can be understood that the value of WSS in Carreau, Casson, and non-Newtonian Power Law have similar results with Newtonian model While Power Law model has unreasonable results.

According to Table.5, the maximum value of WSS in Carreau, Casson, Newtonian and non-Newtonian Power Law are 19.7Pa, 22.5Pa, 18.9Pa, 18.1Pa respectively; however, this value in Power Law is 37.5Pa. Then it can be concluded that the results of Carreau, Casson and non-Newtonian Power Law are acceptable and close to the result of Newtonian model. The other point is that blood modeling with Casson model gives the safest result because it represents the highest value of stress among other logical four models because it gives the biggest value of WSS after Power Low model which is considered as unacceptable model. The other models of Carreau, Newtonian and non-Newtonian after Casson model are recommended respectively simulating blood flow.

Table 5: Maximum amount of WSS at 50% stenosis, 0.85 Sec, and 3mm distance in different models

Viscosity Models	Casson	Carreau	Newtonian	Non Newtonian Power Law	Power Law
WSS (Pa)	22.5	19.7	18.9	18.1	37.5

**2. Local non-Newtonian importance factor**

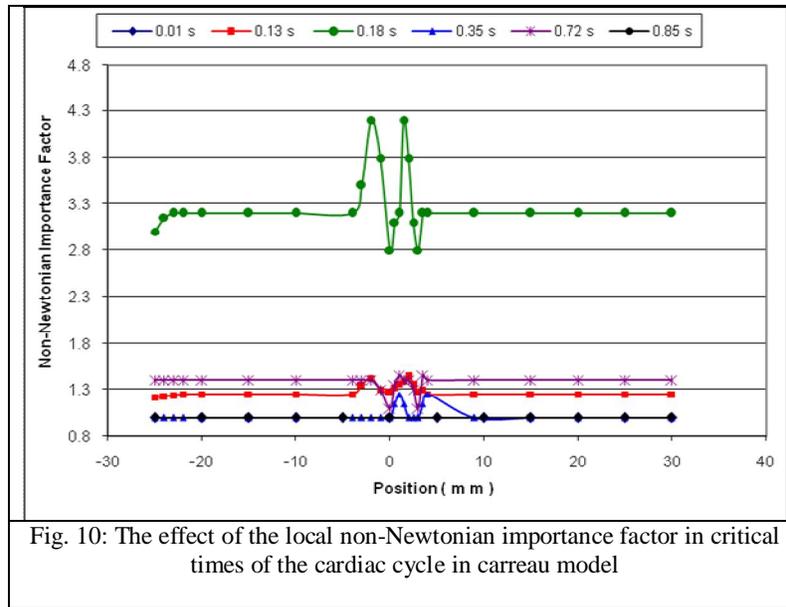
In order to quantify the effect of the remembered non-Newtonian models on the WSS distributions, we now investigate a concept introduced by Ballyk [17]. Local non-Newtonian importance factor ( $I_l$ ) can be obtained by:

$$I_l = \frac{\mu}{\mu_\infty} \tag{8}$$

where  $\mu$  is fluid viscosity at non-Newtonian state and  $\mu_\infty$  is Newtonian viscosity of fluid.

In fact, this factor reveals how much the Newtonian assumption for blood flow in coronary artery is correct. In other words,  $I_l$  shows the amount of difference between the Newtonian model and non-Newtonian models when they are used to simulate blood flow in coronary artery. Fig. 10 shows the effect of this factor at cardiac cycle critical times.

Carreau model is selected as a non-Newtonian model for study in this section. According to this diagram, the non-Newtonian behavior of blood is not important for most times except when the speed approaches to zero values (t=0.05-0.25 Sec); therefore, at these times this factor ( $I_l$ ) becomes effective. The other point is that the values of  $I_l$  tend to 1 in the first and second stenoses where the speed rises strongly.  $I_l$  values during all cardiac cycle is approximately 1 except t=0.18 Sec. Therefore, blood flow in coronary arteries can be considered as a Newtonian flow unless in particular points where speed approaches to zero.



### Conclusion

This paper has analyzed the WSS in different conditions, and the effect of different parameters like time, the stenosis percentage, and the distance between couple stenoses on WSS has been investigated. In this study, it has been concentrated on the analysis of the behavior of the blood flow around the consecutive stenoses with 40, 50, 60 and 75 percents of stenosis. Different viscosity models for blood flow were considered and compared; moreover, local non-Newtonian importance factor ( $I_l$ ) was probed.

At all six critical times of the cardiac cycle, the highest value of the WSS in second stenosis was smaller than first one. Therefore, a mild stenosis (low percentage stenosis) before intense stenosis (high percentage stenosis) reduces the WSS values. The maximum value of WSS has a direct relation with the inlet velocity of the artery. In other words, the maximum values of WSS will increase at high inlet velocity; on the contrary, it will descend at low inlet velocity. This agrees with the conclusion of M. Johnston Barbara [4].

The results showed that the WSS in second stenosis would decrease whatever the stenoses distance has a low value. When the distance of two stenoses decreases, the blood flow does not have enough time to extend along the wall of the artery; therefore, the WSS of the second stenosis becomes low, but when the distance of two stenoses increases, the blood flow that has tended to the center line of the artery has suitable occasion to come back to the primary mode; therefore, the concentration of the flow omits, and it results in increasing the WSS than the former situation.

Comparing the different stenoses percentages showed that the WSS will increase by rising of the stenosis percentage as mentioned in references [4], [10] and [18], and the other point is that the 75% stenosis is a critical stenosis.

Finally, the comparison of the different viscosity models showed it can be concluded that the results of Carreau, Casson and non-Newtonian Power Law are sensible and close to the result of Newtonian model while Power Law model has unreasonable results that similarly presented in [3] and [4]. According to  $I_l$  values, blood flow in coronary arteries can be considered as a Newtonian flow unless in the particular points where speed approaches to zero.

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