BED-Vol. 20, 1991 Advances in Bioengineering
ASME 1991

EFFECT OF THE NON-NEWTONIAN VISCOSITY OF BLOOD ON STEADY AND PULSATILE FLOW IN STENOSED ARTERIES

Rupak K. Banerjee, Young I. Cho, Ph.D.
Department of Mechanical Engineering and Mechanics
Drexel University Philadelphia, PA 19104

and Kenneth Kensey, M.D.
Kensey Nash Corporation, Exton, PA 19341

ABSTRACT

The effect of non-Newtonian viscosity of blood on the steady and pulsatile flow in a straight rabbit femoral artery with various degrees of stenosis is investigated numerically using a finite element technique for the peak Reynolds numbers of 65 and 100. The Cross model is used to take into account the non-Newtonian viscosity of blood at various shear rates. The infinite- and zero- shear-rate viscosities are considered to be 0.0345 and 0.56 poise respectively. A physiological pulsatile flow wave form observed in a rabbit artery with a heart rate of 222 beats/min is used in the current numerical simulation. For steady Newtonian and non-Newtonian flows, the percentage of area constriction due to stenosis is varied from 43% to 81%, whereas for pulsatile flows the study is performed in stenosis with the area constriction ratio of 65%. The magnitude of flow separation and reattachment and the variation of shear stress due to the stenosis are investigated.

NOMENCLATURE

$\dot{\gamma}$	:	shear rate
η	:	non-Newtonian viscosity
η_0, η_∞	:	zero-shear-rate viscosity and infinite-shear-rate viscosity
λ	:	characteristic time, see Table 1 and 2
τ	:	shear stress

INTRODUCTION

Experimental work to understand the effect of non-Newtonian rheology on flows in a mild axisymmetric stenosis has been carried out by Forrester and Young [1]. According to their findings, the length of the separated flow region of blood is smaller than the one for water. Analytical and experimental investigations by Barnes et al. [2] show the non-Newtonian effects of blood on a pulsatile flow in a large arterial vessel flow. In their analysis, a viscoelastic model similar to an Oldroyd model [3] is used. They have concluded that the non-Newtonian effect may not be important in physiological pulsatile flows.

Moravec and Liepsch [4, 5] have concluded that the influence of the viscoelastic characteristics of non-Newtonian fluids upon flow patterns distal to bifurcations is so significant for diameters exceeding 1 mm that it is necessary to consider the non-Newtonian flow behavior. Nakamura and Sawada [6] have conducted a numerical study on the flow of a non-Newtonian fluid through an axisymmetric stenosis. Their results indicate that the non-Newtonian effects weaken the distortion of the flow pattern, pressure distribution and the wall shear stress associated with the stenosis. Perktold [7] has calculated a 3-dimensional pulsatile blood flow in a 90 degree arterial branch taking into account the effect of the non-Newtonian viscosity of blood using the Casson model. However, a comparison with Newtonian viscosity is not provided so that it is difficult to determine the effect of non-Newtonian viscosity. Steffan et al. [8] have conducted a similar numerical simulation in stenotic vessels using a simplified Cross model. They have reported that the general flow structure is not influenced by the non-Newtonian flow effects.

Cho and Kensey [9] reported that the effect of the non-Newtonian viscosity of blood is found to be significant at a flow of the Reynolds number of 100 or less. An experimental investigation by Ojha et. al. [10], using photochromatic tracer methods for a pulsatile flow , has shown spatial and temporal variations of shear stress particularly near the reattachment point [10]. According to Cho and Kensey [11], the instantaneous wall shear stress data indicate that the non-Newtonian characteristics of blood viscosity help a separated flow in the distal region of the stenosis to recover rapidly, thus making flow relatively stable.

The objective of the current investigation was to examine the effect of the non-Newtonian viscosity of blood on the flow in stenosed artery with various degrees of stenosis.

METHODS

In the present investigation, the Cross model [12] has been used to represent the blood viscosity whose model constants are obtained by curve-fitting of available blood viscosity data in the literature (Fig.1).

$$\eta = \eta_\infty + (\eta_0 - \eta_\infty)\frac{1}{1 + (\lambda\dot{\gamma})^m} \qquad (1)$$

where the characteristic time, λ, and the exponent, m, of blood are determined to be 1.007 sec and 1.028, respectively [9].

Then, in order to calculate viscosity in the flow field locally, the local shear rate, $\dot{\gamma}$, is calculated from velocity gradient data through the second invariant of the rate of strain tensor, $II_{\dot{\gamma}}$, as follows:

$$\dot{\gamma} = \sqrt{\frac{1}{2}\Pi} = \sqrt{\frac{1}{2}\left[\sum_i \sum_j \dot{\gamma}_{ij}\,\dot{\gamma}_{ji}\right]} \qquad (2)$$

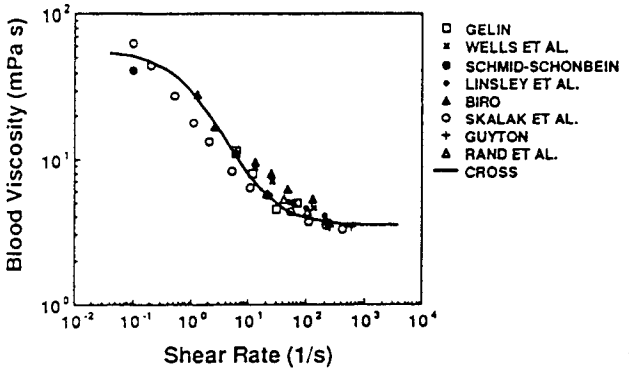

FIG. 1 Blood viscosity vs. shear rate. The percentage of hematocrit is in the range of 33 - 45. Symbols represent viscosity data measured experimentally and the solid line represents the predictions from the Cross model.

After the local viscosity is determined from the Cross model, Eq. (1), the local shear stress, τ, is calculated using the apparent viscosity, η.

$$\tau = \eta \dot{\gamma} \qquad (3)$$

The geometry of a rabbit femoral artery with stenosis is shown in Fig. 2, which has been used for the current numerical simulation. The regular geometry is chosen to validate the numerical data with analytical, experimental and in-vivo data obtained from the actual stenosis introduced in the rabbit femoral artery. Only the numerical results have been discussed here.

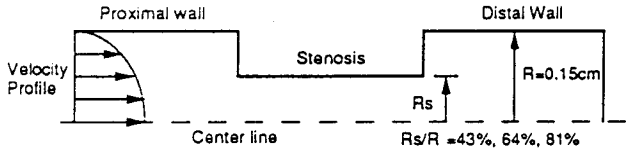

FIG. 2 Lumen radius vs. axial distance along the lumen for a rabbit femoral artery.

Galerkin formulation is applied to discretize the continuity and momentum equations. Of note is that pressure term is removed from the momentum equation using the penalty formulation. Thus, a matrix equation representing discrete analog of the original equations for an individual fluid element is constructed and is solved using standard solution procedures. The generic finite-element computer code FIDAP is used to formulate and solve the matrix equation on the IBM-3090. The numerical simulation of a pulsatile flow requires a time integration method. In particular, the use of the pressure penalty method requires that an implicit time integration scheme be used for the solution of such an unsteady problem. The time integration scheme used in the current study is the second order trapezoidal method with a fixed time step of $\Delta t = 0.00005$ s. At each time step, for spatial integration, the number of iteration steps is limited to ten with a combination of successive substitution, followed by the Newton-Raphson iteration.

RESULTS AND DISCUSSIONS

1) Steady Flows

The magnitude of flow separation and recirculation length, and the variation of shear stress due to stenosis are investigated. The recirculation length is defined as the distance measured from the distal end of the stenosis to the location where the direction of shear stress changes from a negative to a positive value.

Figures 3A, 3B, and 3C show the local wall shear stresses calculated for the steady state flow in axisymmetric vessels with 43%, 64%, and 81% area constrictions, respectively. As the Reynolds number increases from 65 to 100, the wall shear stress at

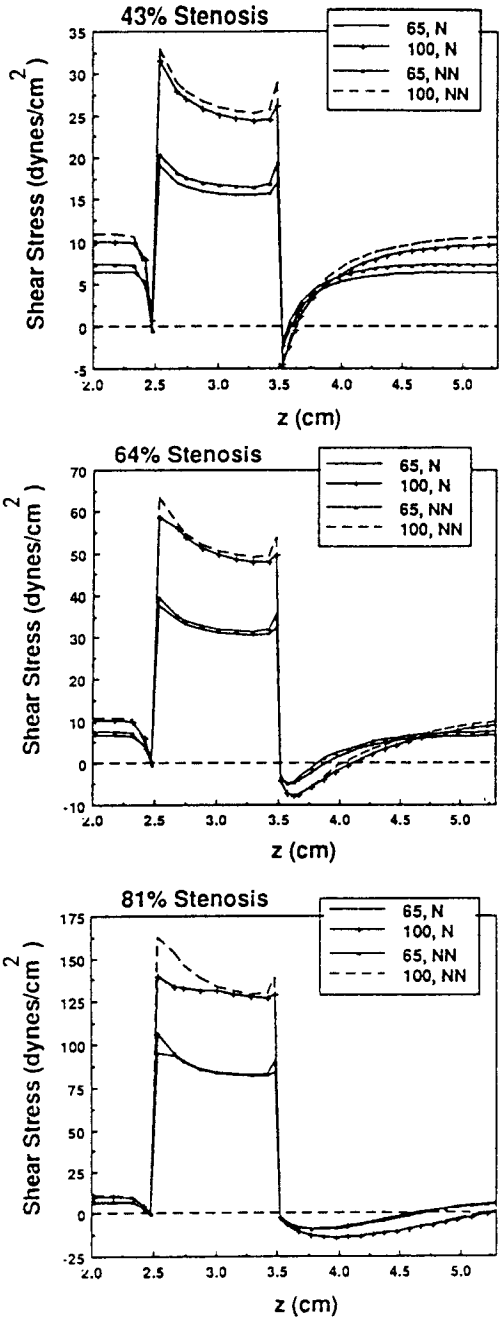

FIG. 3 Wall shear stress distributions at three different stenosis. N represents results with Newtonian viscosity and NN represents those with the non-Newtonian viscosity of blood. 65 and 100 are Reynolds numbers based on the inlet flow conditions.

the stenosed area increases significantly more than the increase in the proximal and non-recirculating distal flow regions of the stenosis. In the recirculation zone the negative value of shear stress becomes more negative. At the 81% stenosis with Newtonian fluid the increase in the peak shear stress in the stenosed region is from 90.4 dyne/cm^2 to 137 dyne/cm^2, whereas in the recirculating zone, the peak negative value changes from -9.37 dyne/cm^2 at an axial distance of 0.38 cm to a value of -14.35 dyne/cm^2 at an axial location of 0.43 cm from the end of stenosis. Compared to the proximal or distal region of the stenosis, the effect of the non-Newtonian viscosity of blood on the wall shear stress, which critically depends on Reynolds

number, is more significant at the start and end of the stenosed region. For blood (a non-Newtonian fluid), the negative value of shear stress in the recirculation zone becomes less negative. At an axial location of 0.53 cm from stenosis and at a Reynolds number of 100, the negative shear stress at the 64% stenosis for blood is -0.56 dyne/cm^2 as compared to a value of -1.41 dyne/cm^2 for the Newtonian fluid.

Figure 4 presents the magnitude of the peak shear stress as a function of the percentage area reduction of the stenosis. In comparison to the Newtonian fluid, the magnitude of a positive peak shear stress at a constant Reynolds number increases for the non-Newtonian fluid. At the stenosis of 81%, the wall shear stress calculated at a Reynolds number of 100 with the Cross model, indicated by "NN", is approximately 163 dyne/cm^2, whereas calculated from the Newtonian model, indicated by "N", it is 134 dyne/cm^2. The effect of the non-Newtonian viscosity of blood on the wall shear stress is found to be more significant at a severe stenosis (81%) than at a mild stenosis (43%).

Figure 5 presents the magnitude of the recirculation length as a function of the percentage area reduction of the stenosis. With the increase in percentage of the stenosis and in the Reynolds number, the recirculation length along the axial direction increases. For the

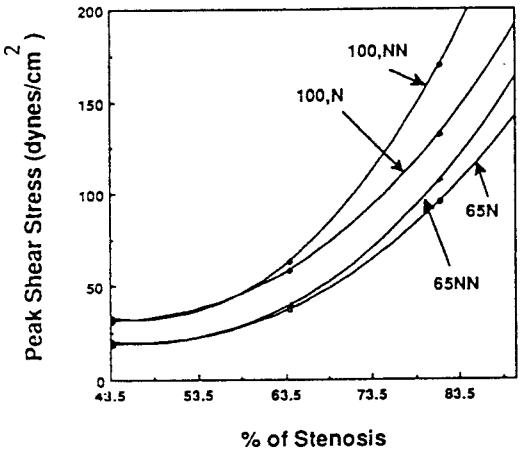

FIG. 4 Peak wall shear stress vs. percentage of stenosis. N represents results with Newtonian viscosity and NN represents those with the non-Newtonian viscosity of blood. 65 and 100 are Reynolds numbers based on the inlet flow conditions.

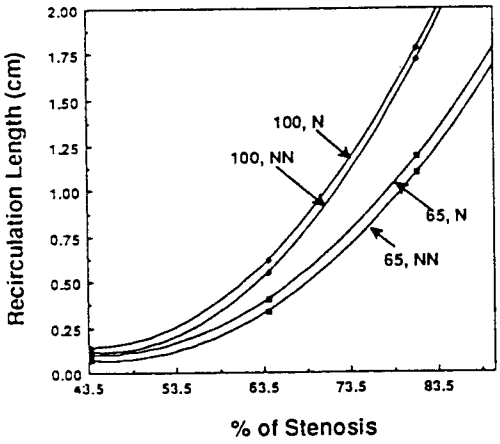

FIG. 5 Recirculation length vs. percentage of stenosis. N represents results with Newtonian viscosity and NN represents those with the non-Newtonian viscosity of blood. 65 and 100 are Reynolds numbers based on the inlet flow conditions.

Newtonian fluid at the 43% stenosis and at a Reynolds number of 100, the recirculation length is 0.64 cm, whereas at the 81% stenosis, it is 1.10 cm. The recirculation length calculated for blood using the Cross model, indicated by "NN", is consistently smaller than that calculated with the Newtonian viscosity, indicated by "N". At the 64% stenosis and at a Reynolds number of 100, the recirculation length for "NN" is 0.55 cm as compared to a value of 0.62 cm for "N".

2) Pulsatile Flows

A normalized pulsatile velocity profile with a cardiac cycle of 0.27s, observed in the rabbit femoral artery, has been shown in Fig. 6. The negative velocity at the end of systole indicates a flow reversal. Shear stress has been studied at six different time steps indicated by A, B, C, D, E and F. The peak Reynolds number is 65 for the current pulsatile flow analysis.

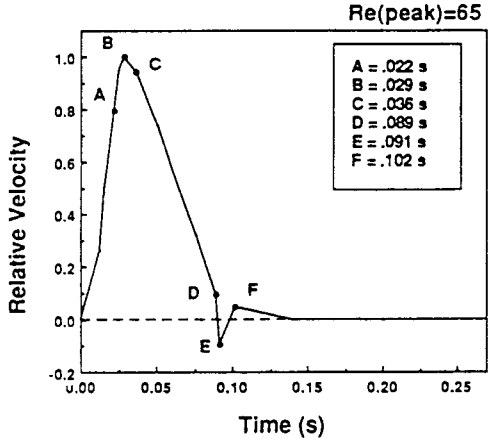

FIG. 6 Relative velocity vs. time observed in the femoral artery of a rabbit. The cardiac cycle is 0.27 s.

Figure 7A represents the wall shear stress plot along the axial direction for time steps A, B, and C which represent the peak velocity region of systole. One of the interesting observations on the shape of pulsatile velocity is that near the peak velocity, a sharp change in velocity gradient takes place, i.e., from positive to zero and then to negative. At the beginning of the heart beat (i.e., acceleration part of systole), the entire region of flow domain is found to have a positive value of shear stress except at the recirculating region (Curve A). While the fluid is in this region, the magnitude of positive shear stress, a typical value being 38.5 dyne/cm^2 at the center of stenosed region, increases until the gradient of velocity is positive. Considering the Curve B, it is observed that the positive value of shear stress, 33.23 dyne/cm^2 at the mid-stenosis, decreases in comparison to Curve A, which is the result of a sharp decrease in the positive gradient of velocity with time. However, for Curve C, the positive shear stress value again increases to a value of 34.71 dyne/cm^2 which may be the cause of the existence of a sharp negative gradient of velocity.

Now considering the negative shear stress in the recirculation region, it is observed that for the accelerating fluid in early systole (Curve A), the magnitude of negative value of shear stress, -1.25 dyne/cm^2 at an axial distance of 0.03 cm from stenosis, is minimum whereas for Curve B the peak negative value of shear stress attains a more negative value of -10.18 dyne/cm^2 at an axial distance of 0.08 cm from stenosis. This is a result of a decrease in positive velocity gradient with time. As the fluid enters the decelerating zone of systole (Curve C), the peak negative value of shear stress becomes less negative, -7.32 dyne/cm^2, at an axial distance of 0.13 cm from stenosis. The recirculating length increases from 0.04 cm (Curve A) to 0.15 cm (Curve B) and then attains a value of 0.21cm (Curve C) as the fluid moves from the accelerating to the decelerating region of systole.

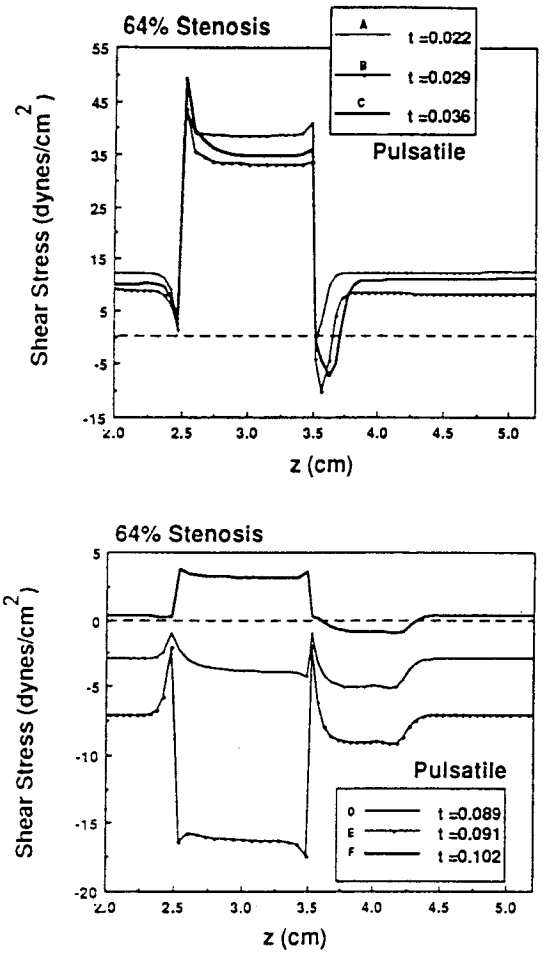

FIG. 7 Wall shear stress distribution for Newtonian viscosity of blood along the axial direction at different time steps of a rabbit cardiac cycle.

Figure 7B represents the wall shear stress plot along the axial direction for time steps D, E, and F which represents low velocities of late systole and entire diastole region. At the late part of systole, the velocity gradient is negative even though the velocity is positive. The deceleration of fluid causes the shear stress at the stenosis to change its direction from positive to negative (Curve D). This trend of changing direction is further evident in the early diastole when both the velocity and its gradient are negative. The maximum value of the negative shear stress is -17.5 dynes/cm^2 (Curve E). However, during mid-diastole where velocity is again positive but less in magnitude as compared to the peak velocity of systole, the shear stress is observed to follow a similar trend as observed during early and mid-systole. The magnitude of shear stress at the stenosis is 3.17 dynes/cm^2 (Curve F) which is comparatively less as compared to a systolic region. Recirculating region is not observed at late systole and early diastole (Curves D and E). However, the maximum recirculating length of 0.86 cm has been obtained at mid-diastole of the cardiac cycle (Curve D). The two peak values of shear stress are -0.92 dynes/cm^2 at an axial location of 0.38 cm from stenosis and -0.99 dynes/cm^2 at a distance of 0.68 cm from stenosis. Note that the late diastole has been considered to be of no flow region.

SUMMARY

The interesting observations are summarized as follows:

i) In a single cardiac cycle there are formations of two recirculating zones, each produced at different time - one generated during systole and the other during diastole. The recirculating zone at systole has a higher shear stress in magnitude than the one at diastole, whereas the recirculating length is greater at diastole than at systole. The repeated generation and shedding of recirculating vortex with time may be responsible for axial progression of stenosis.

ii) The overall change in magnitude and direction of shear stress value in the stenosed region is significantly higher in comparison to the proximal and distal parts of the stenosed artery. The changes in the shear stress value is from +49.4 dynes/cm^2 to -17.5 dynes/cm^2 in the stenosed region whereas it is +11.6 dynes/cm^2 to -6.8 dynes/cm^2 in proximal and distal part of the stenosed artery. Once an initiation of stenosis occurs, the inside region of stenosis always experiences the maximum oscillation of shear stress which may be linked to the progression of stenosis and further damages to the stenosed wall region of the artery.

iii) Along the distal end of stenosis for every cardiac cycle, the recirculating length not only oscillates with time but also moves back and forth along the axial direction. Hence, the recirculating length is a function of both temporal and spatial variation. The oscillation of shear stress with time and space may create increasing damage to the distal arterial wall which is so-called a low-shear region.

iv) The shear stress data indicate that the non-Newtonian characteristics of blood viscosity helps a separated flow to stabilize earlier than a Newtonian fluid. In contrast, the non-Newtonian characteristics of blood viscosity results in the increase in the absolute value of wall shear stress.

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