

Response of a Composite Stenosis for Two Layered Blood Flow Through Arteries

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ABSTRACT:

The present work deals with the blood flow through a composite stenosis assuming that flowing blood is represented by a two layered model consisting of a core region of suspension of all the erythrocyte assumed to be a particle-fluid suspension (i.e., a suspension of all erythrocyte in plasma) surrounded by a peripheral layer of plasma (Newtonian fluid). The expression for the flow characteristics, namely, the impedance, the wall shear stress and the shear stress at the stenosis throat has been derived. Discussions are made from a physiological point of view with the help of graph.

Keywords: *Stenosis, haematocrit, plasma, peripheral layer, core region.*

INTRODUCTION

There has been growing interest in studying blood rheology and blood flow. Dintenfass (1977) reported that rheologic and fluid dynamic properties of blood and its flow play important role in the fundamental understanding, diagnosis and treatment of many cardiovascular, cerebrovascular and arterial disease. Circulatory disorders are known to be responsible for over seventy-five percent of all deaths and stenosis is one of the frequently occurring cardiovascular diseases. The narrowing of anybody passage, tube or orifice in a living mammal is known as stenosis or arteriosclerosis Young (1979). It is an abnormal and unnatural growth that develops at various locations of the cardiovascular systems under diseased conditions and occasionally results in to serious consequences (cerebral strokes, myocardial infarction, angina pectoris, cardiac arrests, etc.). Probably the deposits of the cholesterol, fatty substances, cellular waste products, calcium and fibrin in the inner lining of the of an artery, etc. are responsible for the frequently occurring disease. Irrespective of the cause, it is well known that once stenosis has developed, it brings about the significant changes in the flow field. In the region of narrowing arterial constriction, the flow accelerates and consequently the velocity gradient near the wall region is steeper due to the increased core velocity resulting in relatively large shear stress on the wall even for a mild stenosis. The knowledge that the hemodynamic factors play an important role in the genesis and proliferation of the disease has attracted the investigators including Mann et al. (1938), Young (1979), Shukla et al. (1980), Sarkar and Jayaraman (1998), Pralhad and Schultz (2004), Liu et al. (2004), Srivastava et al. (2012), Ponalagusamy (2007), Mekheimer and El-Kot (2008), Joshi et al. (2009), Singh et al. (2010), Medhavi et al. (2012), Srivastav et al. (2013, 2014a, 2014b, 2015, 2016) and many others.

It has been observed that the whole blood, being predominantly a suspension of erythrocytes in plasma, behaves as a non-Newtonian fluid at low shear rates in microvessel, Whitmore (1963).

Bugliarello and Sevilla (1970), Cokelet (1972) and Thurston

(1989) have shown experimentally that for blood flowing through small vessels, there is cell-free plasma (Newtonian viscous fluid) layer and a core region of suspension of all the erythrocytes. Haynes (1960) presented a two-fluid model of blood flow consisting of a core region of suspension of all the erythrocytes as a homogeneous Newtonian viscous fluid and a cell-free plasma layer as a Newtonian fluid of constant viscosity (equal to the viscosity of water). Skalak (1972) concluded that an accurate description of the blood flow in small vessels requires the consideration of erythrocytes as discrete particles. An examination of viscometric data (Bugliarello et al., 1965; Chein et al., 1965; Rand et al., 1964) suggests that non-Newtonian behavior of blood increases rapidly, when haematocrit rises above 20%, possibly reaching a maximum at between 40-70%. Srivastava and Srivastava (1983) observed that the individuality of red cells (of diameter $8 \mu m$) is significant in such a large vessels with diameter up to hundred cells diameter and concluded that blood can be suitably represented by a macroscopic two-phase model (i.e., a suspension of red cells in plasma) in small vessels (of diameter $\leq 2400 \mu m$). A survey of the literature on arteriosclerotic development indicates that the studies in the literature have been conducted mainly for single symmetric and non-symmetric stenosis. The stenosis may develop in series (multiple stenoses) or may be of irregular shapes or overlapping or of composite in nature. Chakravarty and Mandal (1994) studied the effects of an overlapping stenosis on arterial flow problem of blood.

Keeping these in view, in this paper an effort is made to study the effects of composite stenosis on the flow characteristics taking into account that flowing blood is to be represented, as two-layered model consisting of a core region of suspension of all the erythrocyte assumed to be a particle-fluid suspension (i.e., a suspension of all erythrocyte in plasma) surrounded by a peripheral layer of plasma (Newtonian fluid) in arteries. The artery length is considered large enough as compared to its radius so that the entrance, end and special wall effects can be neglected.

2. Formulation of the problem

Consider the axisymmetric flow of blood in an artery of circular cross-section of radius R with an axisymmetric composite stenosis. Assuming that the flowing blood is represented by a two-layered suspension model consisting of a central layer of suspension of all the erythrocytes (assumed to be a suspension of red cells in plasma) of radius R_1 and a peripheral layer of plasma (a Newtonian viscous fluid) of thickness $(R - R_1)$. The stenosis geometry and the shape of the central layer, assumed to be manifested in the arterial segment are described in Fig.1 as

$$\frac{R(z), R_1(z)}{R_0} = (1, \alpha) - \frac{2(\delta, \delta_1)}{R_0 L_0} (z-d); \quad d \leq z \leq d + L_0/2, \quad (1)$$

$$= (1, \alpha) - \frac{(\delta, \delta_1)}{2R_0} \left\{ 1 + \cos \frac{2\pi}{L_0} (z-d-L_0/2) \right\}; \quad d + L_0/2 \leq z \leq d + L_0 \quad (2)$$

$$= (1, \alpha); \quad \text{otherwise,} \quad (3)$$

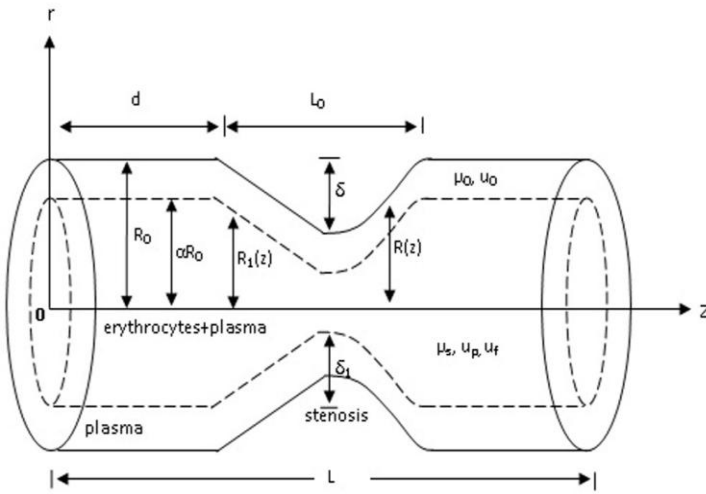


Fig.1 The geometry of a composite stenosis.

where R_0 is the radius of the arterial segment in the non-stenotic region, $R(z)$ is the radius of the stenosed portion; L is tube length, L_0 is the stenosis length and d indicates the location of the stenosis, α is the ratio of the central core radius of the tube radius in the unobstructed region and (δ, δ_1) are the maximum height of stenosis and bulging of the interface at $z=d+L_0/2$.

The equations describing a two-layered suspension blood flow (Srivastava, 2007) in the case of a mild stenosis with additional conditions; $\delta/R_0 \ll 1$, $\text{Re}(2\delta/R_0) \ll 1$ and $2R_0/L_0 \sim O(1)$, are given as

$$(1-C) \frac{dp}{dz} = (1-C) \frac{\mu_s(C)}{r} \frac{\partial}{\partial r} \left(r \frac{\partial}{\partial r} \right) u_f + CS (u_p - u_f), \quad 0 \leq r \leq R_1, \quad (4)$$

$$C \frac{dp}{dz} = CS (u_f - u_p), \quad 0 \leq r \leq R_1, \quad (5)$$

$$\frac{dp}{dz} = \frac{\mu_0}{r} \frac{\partial}{\partial r} \left(r \frac{\partial}{\partial r} \right) u_o, \quad R_1 \leq r \leq R \quad (6)$$

where r is the radial coordinate measured normal to the artery axis and (Re, p) denotes (the tube Reynolds number, pressure), (u_f, u_p) are the axial velocity of (fluid, particle) phases in the core region ($0 \leq r \leq R_1$), (μ_0, u_o) are (viscosity, axial velocity) of fluid (plasma) in the peripheral region ($R_1 \leq r \leq R$), $\mu_s(C) \cong \mu_s$ is the suspension viscosity (apparent or effective viscosity) in the core region, C denotes the constant volume fraction density of the particles (called haematocrit), S is the drag coefficient of interaction exerted by one phase on the other and the subscripts f and p denote the quantities associated with the plasma (fluid) and erythrocyte (particle) phases, respectively. The limitations and the usefulness of the present theoretical model are discussed briefly in Srivastava (2007). The expressions for the viscosity of suspension, μ_s and the drag coefficient of interaction, S for the present study are selected (Srivastava 2015) as

$$\mu_s(C) \cong \mu_s = \frac{\mu_o}{1 - qC}, \quad (7)$$

$$q = 0.07 \exp [2.49C + \left(\frac{1107}{T}\right) \exp (-1.69C)], \quad (8)$$

$$S = 4.5 \left(\mu_o/a_o^2 \right) \frac{4 + 3[8C - 3C^2]^{1/2} + 3C}{(2 - 3C)^2}, \quad (9)$$

where T is measured in absolute scale of the temperature (K), μ_o is the constant plasma viscosity and a_o is the radius of an erythrocyte. The empirical relation for the suspension viscosity suggested by Charm and Kurland (1974) is found to be reasonable accurate up to $C=0.6$ (60% haematocrit).

The boundary conditions are the standard no slip conditions of velocities and the shear stresses at the tube wall and the interface are stated as

$$u_o = 0, \quad \text{at } r=R, \quad (10)$$

$$u_o = u_f \quad \text{and} \quad \tau_p = \tau_f, \quad \text{at } r=R_1, \quad (11)$$

$$\frac{\partial u_f}{\partial r} = \frac{\partial u_p}{\partial r} = 0, \quad \text{at } r=0, \quad (12)$$

where $\tau_p = \mu_o \partial u_o / \partial r$ and $\tau_f = (1-C) \mu_s \partial u_f / \partial r$ are the shear stresses of the peripheral and central layers, respectively.

3. Analysis

The expressions for velocities, u_o , u_f and u_p obtained as the solutions of Eqs. (4)-(6), subject to the boundary conditions Eqs. (10)-(12), are given as

$$u_o = -\frac{R_0^2}{4\mu_0} \frac{dp}{dz} \left\{ \left(\frac{R}{R_0} \right)^2 - \left(\frac{r}{R_0} \right)^2 \right\}, \quad (13)$$

$$u_f = -\frac{R_0^2}{4(1-C)\mu_0} \frac{dp}{dz} \left[\mu \left\{ \left(\frac{R_1}{R_0} \right)^2 - \left(\frac{r}{R_0} \right)^2 \right\} + (1-C) \left\{ \left(\frac{R}{R_0} \right)^2 - \left(\frac{r}{R_0} \right)^2 \right\} \right], \quad (14)$$

$$u_p = -\frac{R_o^2}{4(1-C)\mu_0} \frac{dp}{dz} \left[\mu \left\{ \left(\frac{R_1}{R_0} \right)^2 - \left(\frac{r}{R_0} \right)^2 \right\} + (1-C) \left\{ \left(\frac{R}{R_0} \right)^2 - \left(\frac{r}{R_0} \right)^2 \right\} + \frac{4(1-C)\mu_0}{SR_0^2} \right], \quad (15)$$

where $\mu = \mu_0/\mu_s$.

The flow flux, Q is now calculated as

$$Q = 2\pi \left[\int_{R_1}^R ru_o dr + \int_0^{R_1} r \left\{ (1-C)u_f + Cu_p \right\} dr \right]$$

$$Q = -\frac{\pi R_o^4}{8(1-C)\mu_0} \frac{dp}{dz} \left[(1-C) \left\{ \left(\frac{R}{R_0} \right)^4 - \left(\frac{R_1}{R_0} \right)^4 \right\} + \mu \left(\frac{R_1}{R_0} \right)^4 + \beta \left(\frac{R_1}{R_0} \right)^2 \right], \quad (16)$$

With $\beta = 8C(1-C)\mu_0/SR_0^2$, a non-dimensional suspension parameter.

Following now report of (Srivastava, 2007) and using the fact that the total flux is equal to the sum of the fluxes across the two regions (peripheral and core), one determines the relations: $R_1 = \alpha R$ and $\delta_1 = \alpha \delta$. In view of these relations, the pressure drop, Δp ($= p$ at $z = 0$, $-p$ at $z = L$) across the stenosis between the sections $z = 0$ and $z = L$, is calculated from Equ. (16) as

$$\Delta p = \int_0^L \left(-\frac{dp}{dz} \right) dz,$$

$$\Delta p = \frac{8(1-C)\mu_0 Q}{\pi R_o^4} \psi, \quad (17)$$

where

$$\psi = \int_0^d [\phi(z)]_{R/R_0=1} dz + \int_d^{d+L_0/2} [\phi(z)]_{R/R_0 \text{ from } (1)} dz + \int_{d+L_0/2}^{d+L_0} [\phi(z)]_{R/R_0 \text{ from } (2)} dz + \int_{d+L_0}^L [\phi(z)]_{R/R_0=1} dz$$

$$\phi(z) = \frac{1}{\eta(R/R_0)^4 + \beta\alpha^2(R/R_0)^2}, \quad \eta = (1-C)(1-\alpha^4) + \mu\alpha^4$$

The first, second and fourth integrals in the expression for ψ obtained above are straight forward whereas third integral evaluated numerically using computer programming. Using now the definitions from Srivastava (2015), one obtains the final expressions for the impedance (flow resistance), λ , the wall shear stress, τ_w and the shear stress at the stenosis throat, τ_s are obtained in their non-dimensional form as:

$$\lambda = (1-C) \left[\frac{1-L_0/L}{\eta + \beta\alpha^2} - \frac{L_0/L}{2\beta\alpha^2(\delta/R_0)} \left\{ \frac{\sqrt{\eta}}{\alpha\sqrt{\beta}} \tan^{-1} \left(\frac{\alpha\sqrt{\eta\beta}(\delta/R_0)}{\alpha^2\beta + \eta(1-\delta/R_0)} \right) - \frac{\delta/R_0}{(1-\delta/R_0)} \right\} + \frac{1}{L} \left[\int_{d+L_0/2}^{d+L_0} [\phi(z)]_{R/R_0 \text{ from } (2)} dz \right] \right], \quad (18)$$

$$\tau_w = \frac{(1-C)}{\eta(R/R_0)^3 + \beta\alpha^2(R/R_0)}, \quad (19)$$

$$\tau_s = \frac{(1-C)}{\eta(1-\delta/R_0)^3 + \beta\alpha^2(1-\delta/R_0)}, \quad (20)$$

where $\lambda = \bar{\lambda}/\lambda_0$, $(\bar{\tau}_w, \bar{\tau}_s) = (\tau_w, \tau_s)/\tau_0$, $\bar{\lambda} = \Delta p/Q$,

$$\bar{\tau}_w = (-R/2)dp/dz, \quad \bar{\tau}_s = [-(R/2)(dp/dz)]_{R/R_0=(1-\delta/R_0)},$$

$$\lambda_0 = 8\mu_0 L/\pi R_0^4, \quad \tau_0 = 4\mu_0 Q/\pi R_0^3,$$

λ_0 and τ_0 are the impedance and shear stress in a normal (no stenosis) artery for a Newtonian fluid (i.e., $C = 0$), and $(\bar{\lambda}, \bar{\tau}_w, \bar{\tau}_s)$ are (impedance, wall shear stress, shear stress at the stenosis throat) in their dimensional form.

4. Numerical result and discussion

To have a quantitative estimate of the various parameters involved, computer programs are developed to evaluate the analytical result at a temperature of $37^\circ C$ in an artery of radius $R_0 = 0.1$ cm and for various other parameter values are selected (Young, 1968; Srivastava et al., 2009 a, b) as $d(\text{cm})=0$; $L_0(\text{cm})=1$; $L(\text{cm})=1, 2, 5$; $\delta/R_0=0, 0.05, 0.10, 0.15, 0.20$. Some of the critical results obtained are displayed graphically in Figs. 3-8. In view of the fact that the peripheral layer thickness strongly depends on core suspension viscosity (i.e., on erythrocyte concentration; Bugliarello and Sevilla 1970, Srivastava 2007) we choose $2a_o$ (erythrocyte diameter) = $8 \mu m$, the peripheral layer thickness, $\varepsilon(\mu m) \equiv \varepsilon(C) = 6.18, 4.67, 3.60, 3.12, 2.58, 2.18$ corresponding to the haematocrit, $C = 0.1, 0.2, 0.3, 0.4, 0.5, 0.6$ respectively Haynes (1960). The value of parameter α is then calculated from the relation $\alpha = 1 - \varepsilon/R_0$. It is to note that the present analysis corresponds the case of a two-layered blood flow i.e., $\alpha < 1$.

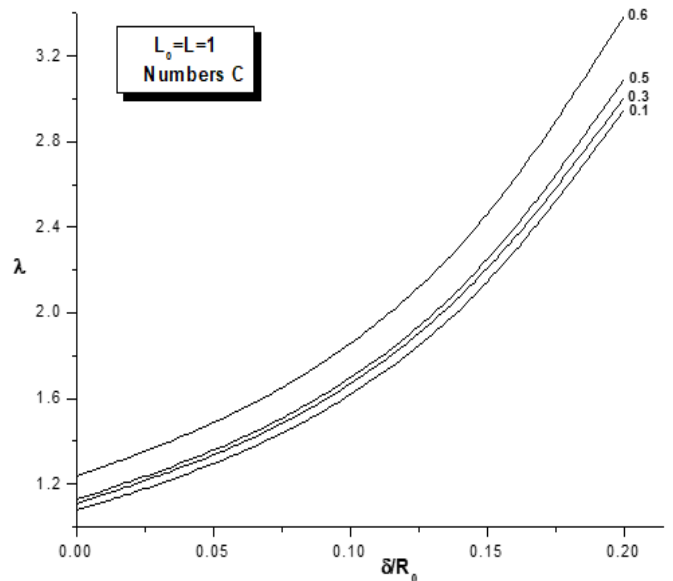


Fig.3 Impedance, λ versus stenosis height, δ/R_0 for different C.

The flow characteristics impedance λ increases with haematocrit, C as well as with the stenosis height, δ/R_0 (Fig. 3).

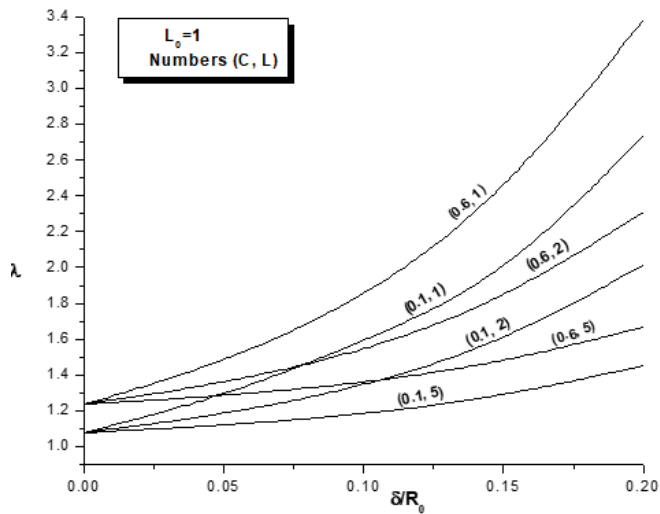


Fig.4 Impedance, λ versus stenosis height δ/R_0 for different C and L .

magnitude of the shear stress, τ_s is not higher than the corresponding magnitude of the impedance, λ for given parameters.

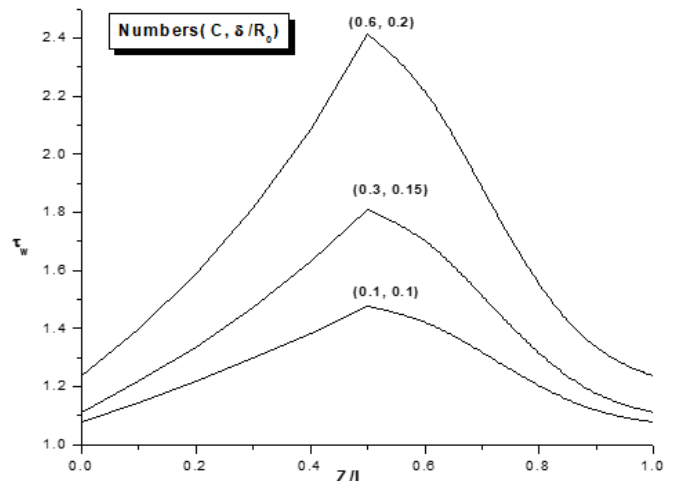


Fig.6 Wall shear stress, τ_w versus Z/L_0 in the stenotic region for different C and δ/R_0 .

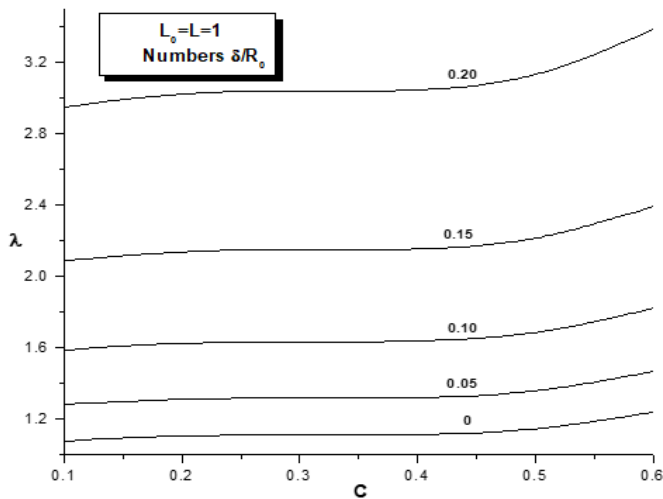


Fig.5 Impedance, λ versus haematocrit, C for different δ/R_0 .

The flow characteristics, impedance, λ decreases with increasing tube length, L which in terms implies that impedance, λ increases with stenosis length, δ/R_0 (Fig. 4).

The impedance, increases steeply with haematocrit, C for any given stenosis height, δ/R_0 (Fig. 5). At any axial distance, the wall shear stress in stenotic region, τ_w increases rapidly in upstream of the stenosis throat and attains its peak magnitude at $Z/L_0 = 0.5$, it then decreases rapidly in downstream of the throat and attains its approached value (i.e., $Z/L_0 = 0$) at the end of the constriction profile located at $Z/L_0 = 1$ (Fig. 6).

The shear stress at the stenosis throat, τ_s increases with the haematocrit, C and stenosis height, δ/R_0 (Figs. 7 and 8). However, the shear stress at the stenosis throat, τ_s assumes lower magnitude in the two-layered analysis than its corresponding magnitude in single-layered analysis for any given haematocrit C . An inspection of Figs. 3-5, reveals that the shear stress at stenosis throat, τ_s possesses the characteristics similar to that of the flow resistance, with respect to any parameter. The

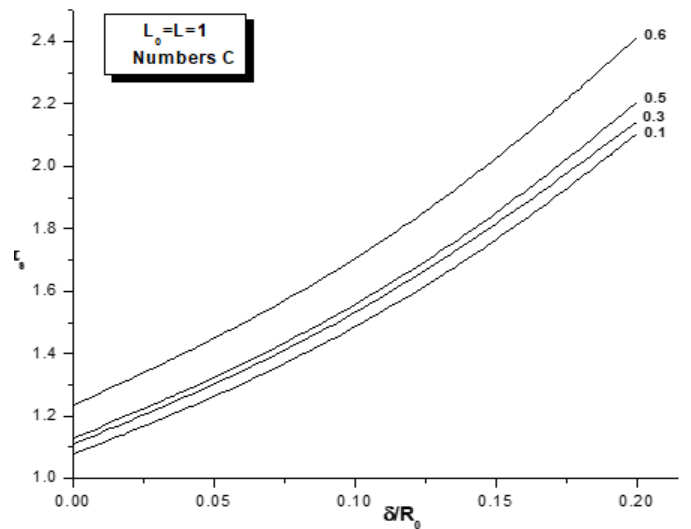


Fig.7 shear stress at stenosis throats, τ_s versus stenosis height δ/R_0 for different C .

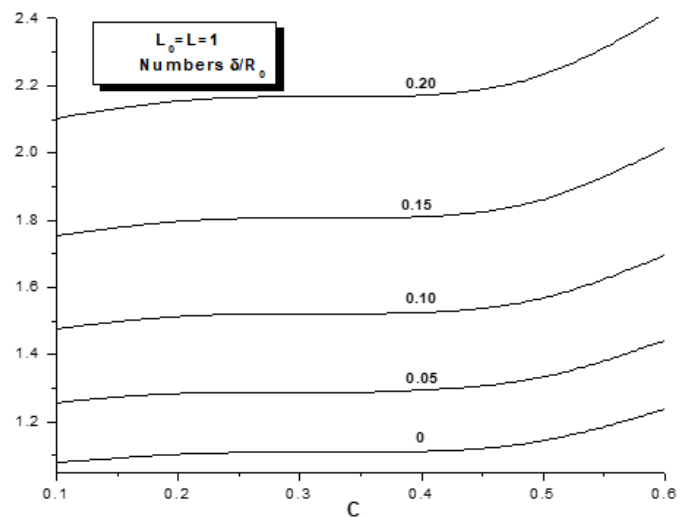


Fig.8 shear stress at stenosis throats, τ_s versus haematocrit, C for different stenosis height, δ/R_0 .

CONCLUSIONS

To observe the effects of composite stenosis on blood flow characteristics taking into account that flowing blood is to be represented as two-layered model has been used to discuss. The blood flow characteristics (the flow resistance, the wall shear stress in the stenotic region and the shear stress at the stenosis throat) increase with the haematocrit as well as with the stenosis size (Length and height). The shear stress at the stenosis throat possesses the characteristics similar to that of the impedance with respect to any parameter.

REFERENCES

1. Bugliarello, G. and Sevilla, J., Velocity distribution and other characteristics of steady and pulsatile blood flow in fine glass tubes. *Biorheol.* 7, 1970, 85-107.
2. Bugliarello, G., Kapur, C. and Hsiao, G., The profile viscosity and other characteristics of blood flow in non-uniform shear field. *Symp. of Biorheol.* (Ed. Copley, A. L.), Intersci., New York, 1965, pp. 351-370.
3. Charm, S. E. and Kurland, G. S., "Blood Flow and Microcirculation", John Wiley, N. Y., 1974.
4. Chakravarty, S. and Mandal, P. K., Mathematical modelling of blood flow through an overlapping stenosis. *Math. Comput. Model.* 19, 1994, 59-73.
5. Chein, S., Usmani, S., Taylor, H. M., Lundberg, J. L. and Gregerson, M. T., Effects of hematocrit and plasma proteins on human blood rheology at low shear rates. *J. Appl. Physiol.*, 21, 1965, pp. 81-87.
6. Cokelet, G.R., *The Rheology of Human Blood: In Biomechanics.* Prentice-Hall, Englewood Cliffs, New Jersey, 1972.
7. Dintenfass, L, Viscosity factors in hypertensive and cardiovascular diseases. *Cardiovascular Med.*, 2, 1977, 337-363.
8. Haynes, R.H., Physical Basis on Dependence of Blood Viscosity on Tube Radius. *American Journal of Physiology*, 198, 1960, 1193-1205.
9. Joshi, P., Pathak, A. and Joshi, B.K., Two layered model of blood flow through composite stenosed artery. *Applications and Applied Mathematics*, 4(2) , 2009, 343-354.
10. Liu, G.T., Wang, X.J., Ai, B.Q. and Liu, L.G., Numerical study of pulsating flow through a tapered artery with stenosis. *Chin. Journal Phys.*, 42, 2004, 401-409.
11. Medhavi, A, Srivastav, R. K., Ahmad Q. S. and Srivastava, V. P., Two-phase arterial blood flow through a composite stenosis. *e-jst*, 7(4) , 2012, pp. 83-94.
12. Mann, F. C., Herrick, J. F., Essex, H. E. and Blades, E. J., Effects on blood flow of decreasing the lumen of blood vessels. *Surgery* 4, 1938, 249-252.
13. Mekheimer, Kh. S. and El-Kot, Magnetic field and hall currents influences on blood flow through a stenotic arteries. *Applied Mathematics and Mechanics*, 29, 2008, 1-12.
14. Ponalagusamy, R., Blood flow through an artery with mild stenosis: A two layered model, different shapes of stenosis and slip velocity at the wall. *J Appl. Sci.* 7(7), 2007, 1071-1077.
15. Pralhad, R.N. and Schultz, D.H., Modeling of arterial stenosis and its applications to blood diseases. *Math. Biosci.*, 190, 2004, 203-220.
16. Rand, P. W., Lacombe, E., Hunt, H. E. and Austin, W. H., Viscosity of normal blood under normothermic and hypothermic conditions. *J. Appl. Physiol.*, Vol.19, 1964, pp. 117-112.
17. Sarkar, A. and Jayaraman, G., Correction to flow rate-pressure drop in coronary angioplasty: steady streaming effect. *J. Biomech.* 31, 1998, 781-791.
18. Shukla, J. B., Parihar, R. S. and Gupta, S. P., Effects of peripheral layer viscosity on blood flow through the artery with mild stenosis. *Bull. Math. Biol.* 42, 1980, 797-805.
19. Singh, B., Joshi, P. and Joshi, B.K., Blood flow through an artery having radially non-symmetric mild stenosis. *Appl. Math. Sci.* 4(22), 2010, 1065-1072.
20. Skalak, R., *Mechanics of microcirculation: In Biomechanics, Its foundation and Objectives,* Prentice Hall, Englewood Cliffs, 1972.
21. Srivastava, V. P., A theoretical model for blood flow in small vessels. *Applications and Applied Mathematics*, 2, 2007, 51-65.
22. Srivastav R. K., Atul Kumar Agnihotri and Manisha Gupta, "A Mathematical Model for Analysis of Blood Flow in a Stenosed Artery with Permeable Wall" *Asian Journal of Science and Technology*, Vol. 7, Issue 1, 2016, 2230-2236.
23. Srivastav R. K., "Two-Layered Model of Blood Flow Through Arterial Catheterization with non-Symmetric Constriction" *J. of Computation in Biosciences and Engineering*, Vol. 2(2), 2015, 1-8.
24. Srivastav R. K., and V. P. Srivastava, "On Two-Fluid Blood Flow Through Stenosed Artery with Permeable Wall", *Applied Bionics and Biomechanics*, Vol.11 Issue 1-2, 2014a, 39-45.
25. Srivastav R. K., "Mathematical Model of Blood Flow Through A Composite Stenosis In Catheterized Artery With Permeable Wall" *Applications and Applied Mathematics*, Vol. 9, Issue 1, 2014b, 58-74.
26. Srivastav R. K., Qazi Shoeb Ahmad and Abdul Wadood Khan, "Blood Flow Through An Overlapping Stenosis In Catheterized Artery With Permeable Wall" Paper published in an journal: *e-Journal Of Science & Technology*, Vol. 8, Issue 2, 2013, 43-53.
27. Srivastava V.P., Vishnoi R., Medhavi A. and Sinha P., A suspension flow of blood through a bell shaped stenosis. *e-Journal of Science and Technology*, 7(1) , 2012, 97-107.
28. Srivastava, L.M. and Srivastava, V.P., "On two-phase model of pulsatile blood flow with entrance effects", *Biorheology*, 20(6), 1983, 761-777.
29. Thurston, G. B., Plasma release cell-layering theory for blood flow. *Biorheol.* 26, 1989, 199-214.
30. Whitmore, R.L., Hemorheology and hemodynamics. *Biorheology*, 1, 1963, 201-220.
31. Young, D. F., Fluid mechanics of arterial stenosis. *Journal of Biomechanics Eng*, 101, 1979, 157-175..

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