Computational Hemodynamics Analysis in Large Blood Vessels: Effects of Hematocrit Variation on the Flow Stability

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Abstract: Understanding the effects of blood viscosity variation plays a very crucial role in hemodynamics, thrombosis and inflammation and could provide useful information for diagnostics and therapy of (cardio) vascular disease. Blood viscosity, which arises from frictional interactions between all major blood constituents, i.e. plasma, plasma proteins and red blood cells, constitutes blood inherent resistance to flow in the blood vessel. Because red blood cells (RBCs) are the main constituent of the cellular phase of blood, white blood cells and platelets normally do not have a great influence on whole blood viscosity. When blood flows through a vessel, it tends to separate in two different phases. In direct contact with the wall a low viscosity phase exists, which is deficient in cells and rich in plasma and acts as a lubricant for the blood transport. In the central core region of the vessel a high viscosity phase exists, which depends on the hematocrit. In this poster, the nature and stability of blood flow in a large artery is investigated numerically using a spectral collocation technique with expansions in Chebyshev polynomials. The study reveals that a rise in hematocrit concentration in the central core region of a large artery has a stabilizing effect on the flow.

1.1 Model Assumptions

For the development of mathematical model (as illustrated in fig. 1 below), the following assumptions are made:

(i) In the large artery, blood is assumed to be an incompressible Newtonian fluid.

(ii) Due to the presence of plasma layer near the vessel wall, the local viscosity in this peripheral region would be close to that of the plasma and it would be lower than the viscosity in the central core region which depends on the hematocrit.

(iii) A two-dimensional flow problem is considered.

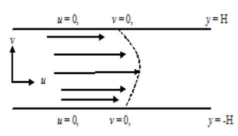

Fig. 1. Geometry of the problem

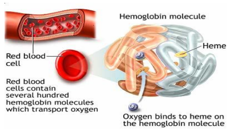

Fig. 2. Red blood cells in the artery

1.2 Blood Viscosity

Blood viscosity, which arises from frictional interactions between all major blood constituents, i.e. plasma, plasma proteins and red blood cells, constitutes blood inherent resistance to flow in the blood vessel. Because red blood cells (RBCs) are the main constituent of the cellular phase of blood, white blood cells and platelets normally do not have a great influence on whole blood viscosity.

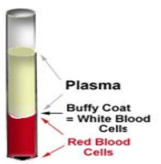

Fig. 3. Blood composition

1.3 Arterial Blood Viscosity Model

When blood flows through a vessel, it tends to separate in two different phases near the vessel wall, where shear rate is highest. In direct contact with the wall a low viscosity phase exists, which is deficient in cells and rich in plasma and acts as a lubricant for the blood transport. This effect leads to a steep rise in velocity near the wall. This is in contrast to the situation in the core region of the vessel, where shear rate is low, RBC aggregation is predominant and therefore local viscosity rises, which reduces the differences in velocity in adjacent central fluid layers. Based on the transverse variation in the hematocrit ratio within the blood vessel, the blood viscosity function μ is modeled as;

$$\mu(y) = e^{\beta(1-y^2)}$$

where β is the viscosity variation hematocrit parameter.

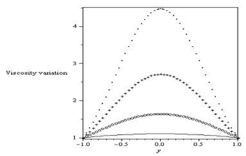

Fig. 4. Arterial blood viscosity variation, ____β=0.1, ooooβ=0.5, ++++β=1, ...β=1.5

1.4 Model Equations

The governing equations of continuity and momentum for axially symmetric flow of blood through an artery in dimensionless form under the above mentioned assumption are

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0,$$

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} + v\frac{\partial u}{\partial y} = -\frac{\partial P}{\partial x} + \frac{2}{\mathrm{Re}}\frac{\partial}{\partial x}\left(\mu\frac{\partial u}{\partial x}\right) + \frac{1}{\mathrm{Re}}\frac{\partial}{\partial y}\left[\mu\left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x}\right)\right],$$

$$\frac{\partial v}{\partial t} + u\frac{\partial v}{\partial x} + v\frac{\partial v}{\partial y} = -\frac{\partial P}{\partial y} + \frac{2}{\mathrm{Re}}\frac{\partial}{\partial y}\left(\mu\frac{\partial v}{\partial y}\right) + \frac{1}{\mathrm{Re}}\frac{\partial}{\partial x}\left[\mu\left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x}\right)\right],$$

where t is the time, P is pressure, *Re* is the Reynolds number, *x* is the coordinate in the streamwise direction, *y* is the normal coordinate, (*u*, *v*) are the velocity components in the *x* and *y* directions respectively.

1.5 Basic Flow Model

The basic steady state of the arterial blood flow system corresponds to a parallel flow with velocities $u = U(y)$ and $v = 0$. The equation and the boundary conditions describing the basic state are

$$\frac{d}{dy}\left(\mu\frac{dU}{dy}\right) = -G, \quad \frac{dU}{dy}(0) = 0, \quad U(1) = 0.$$

The solution is given by

$$U(y;\beta>0) = \frac{G}{2\beta}\left[1 - e^{\beta(y^2-1)}\right]$$

$$U(y;\beta\to 0) \approx \frac{G}{2}(1-y^2) - \frac{G}{4}(y^2-1)^2\beta - \frac{G}{12}(y^2-1)^3\beta^2 + O(\beta^3)$$

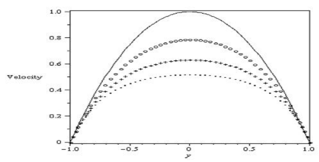

Fig. 5. Velocity profile, G=2; ____β=0, ooooβ=0.5, ++++β=1, ...β=1.5

1.6 Linear Stability Analysis

In the stability analysis, two-dimensional disturbances will be considered which implies that Squire's transformation is applicable. Introducing small disturbances to the basic flow as follows:

$$u(x,y,t) = U(y) + \hat{u}(x,y,t),\ v(x,y,t) = \hat{v}(x,y,t),\ p(x,y,t) = P(x) + \hat{p}(x,y,t),$$

where $\hat{u} = \frac{\partial \psi}{\partial y} = \phi'(y)e^{i\alpha(x-ct)}, \quad \hat{v} = -\frac{\partial \psi}{\partial x} = -i\alpha\phi(y)e^{i\alpha(x-ct)}$ represent the small disturbances.

After substituting the small disturbances into the linearized model equations, we obtain

$$(U-c)(\phi'' - \alpha^2\phi) - U''\phi = \frac{\mu}{i\alpha \mathrm{Re}}(\phi^{iv} - 2\alpha^2\phi'' + \alpha^4\phi) + \frac{2\mu'}{i\alpha \mathrm{Re}}(\phi''' - \alpha^2\phi') + \frac{\mu''}{i\alpha \mathrm{Re}}(\phi'' + \alpha^2\phi),$$

with the boundary conditions;

$$\phi(-1) = \phi'(-1) = 0,$$
$$\phi(1) = \phi'(1) = 0.$$

1.7 Computational Approach

The linear stability equation is solved using the Chebyshev spectral collocation method. Let

$$\phi(y) \approx \phi_N(y_j) = \sum_{k=0}^{N}\tilde{\phi}T_k(y_j), \quad y_j = \cos\frac{\pi j}{N}, \quad j = 0, 1, \ldots, N$$

T_k is defined in terms of Chebysev polynomials. We obtain an $(N+1)\times(N+1)$ algebraic equations which form the generalized eigenvalue problem $E\phi = cB\phi$, where

$$E(m,n) = \begin{cases} 1 & m = n = 0; \\ 0 & m = 0, n = 1, \ldots, N; \\ \sum_{n=0}^{N} D_{0n} & m = 1, n = 0, \ldots, N; \\ \tilde{E}(m,n) & m = 1, \ldots, N-2, n = 0, \ldots, N; \\ \sum_{n=0}^{N} D_{Nn} & m = N-1, n = 0, \ldots, N; \\ 0 & m = N, n = 1, \ldots, N-1; \\ 1 & m = N, n = N; \end{cases}$$

$$B(m,n) = \begin{cases} 0 & m = 0, 1, N-1, N \quad n = 0, \ldots, N; \\ \tilde{B}(m,n) & m = 2, \ldots, N-2, \quad n = 0, \ldots, N; \end{cases}$$

and

$$\tilde{B} = (D^2 - \alpha^2 I)$$

$$\tilde{E} = U(D^2 - \alpha^2 I) - U'' + \frac{i\mu}{\alpha \mathrm{Re}}(D^4 - 2\alpha^2 D^2 + \alpha^4 I) + \frac{2i\mu'}{\alpha \mathrm{Re}}(D^3 - \alpha^2 D) + \frac{i\mu''}{\alpha \mathrm{Re}}(D^2 + \alpha^2 I)$$

with

$$\tilde{\phi}_0 = 0, \quad \sum_{n=0}^{N} D_{0n}\tilde{\phi}_n = 0, \quad \text{on } y = 1.$$

$$\tilde{\phi}_N = 0, \quad \sum_{n=0}^{N} D_{Nn}\tilde{\phi}_n = 0, \quad \text{on } y = -1.$$

The eigensolutions of the generalized eigenvalue problem obtained numerically are presented as follows;

Tab. 1. Computation showing the convergence of the procedure. (G =2, Re = 10000, α = 1, β = 0)

N	c (wave speed)
20	0.94506189220347 - 0.01411409170062i
40	0.23751460074093 +0.00374110523480i
60	0.23752650030571 +0.00373966231199i
80	0.23752648881692 +0.00373967059842i
100	0.23752648857617 +0.00373967068991i
120	0.23752648844019 +0.00373967128489i
140	0.23752648870166 +0.00373967124014i
160	0.23752648870166 +0.00373967124014i

Tab. 2. Computation showing the eigenvalue of the most unstable mode (G =2, Re =10000, α= 1)

β	c (wave speed)
0.0	0.23752648870166 +0.00373967124014i
0.2	0.22071871898235 - 0.00222898529911i
0.4	0.20622156700088 - 0.00828137142384i
0.6	0.19343094110417 - 0.01422161182373i
0.8	0.18191776169859 - 0.01989289744428i
1.0	0.17137725303345 - 0.02514787924071i
1.2	0.16162042785847 - 0.02983817494878i
1.4	0.50275600099699 - 0.03378659623069i
1.6	0.46347037649715 - 0.03330907160023i
1.8	0.42848389064951 - 0.03273863003689i
2.0	0.39728869392928 - 0.03209421083017i

Tab. 3. Computations showing the critical values at which unstable modes begin to exist (G = 2)

β	α_c	Re_c
0.0	1.02052	5772.2283
0.1	0.98418	8866.9768
0.2	0.95655	13194.8926
0.3	0.93639	19001.9400
0.4	0.92187	26519.6703
0.5	0.91948	36002.2233
0.6	0.90911	47522.8415
1.0	0.91330	118070.0013

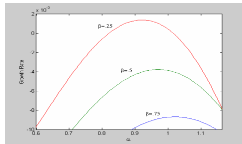

Fig. 6. Disturbance growth rate (αc_i) for Re = 10000

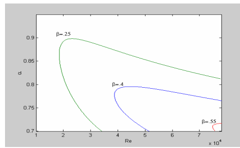

Fig. 7. Marginal stability curve for G = 2.

1.8 Conclusion

Linear stability analysis of variable viscosity arterial blood flow is presented. The resulting eigenvalue problem is solved numerically using Chebyshev spectral collocation method implemented in MATLAB. We obtained accurately the critical Reynolds number *Rec* and the critical wave number *αc* for increasing values of blood viscosity variation parameter. It has been observed that an increase in hematocrit towards the central core region of the artery has a stabilizing effect on the flow. The results obtained are of physiological interest and clinical applications.

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