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RESEARCH ARTICLE

MATEHEMATICAL MODELING IN TWO PHASES PULMONARY BLOOD FLOW THROUGH ARTERIOLES IN LUNGS WITH SPECIAL REFERENCES CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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ABSTRACT

In the present paper, we will discuss the pulmonary blood flow in Lung. Pandey and Upadhyay have considered the blood flow of two phase, one of which is that of red blood cells and other is plasma. They have also used Herschel- Bulkley non- Newtonian model in purpose of bio fluid mechanical set-up .We have known that as the velocity of blood flow decrease successively because of the fact that arterioles, venules and veins are far enough from the heart. We have collected clinical data in case of COPD for Hematocrit v/s Blood pressure. The graphical presentation for particular parametric value in much closed to the clinical observation. The overall presentation is in tensorial form and solution technique adapted is analytical as well as numerical.

Key words: Herschel Bulkely, pulmonary, arterioles, parametric value, overall presentation.

INTRODUCTION

Blood is mixed fluid. Mainly there are two in blood. The first is plasma, while the other phase is that of blood cells are enclosed a semi-permeable membrane density is greater than of plasma. These blood cells are uniformly distributed in plasma. Thus, blood can be considered as a homogenous mixture of two phases **(**Jain 1961). This work will focus on two phases pulmonary blood flow in Lungs with special reference to COPD. Mishra and Pandey 2003 have suggested that blood flow in vessels is a peristaltic transport system because they thought blood is having two layers of fluid while in the peripheral reasons of vessels blood flow is a newtonian phenomena. They also observed that flow rate of blood in single layer stress is more than two layer flow while the peripheral layer is more viscous than the core layer. A lot of work is available, but Pandey and Upadhyay (1999) discussed some phenomena is two phase blood flow gave an idea on the two phase pulmonary blood flow in lungs with pulmonary disease COPD. the whole work of Pandey and Upadhyay is in whole circulatory system but our work will focus on pulmonary circulatory system.

The lungs are the main organ of the lower respiratory tract and responsible for the exchange of oxygen, which is necessary for cellular processes, for carbon dioxide, a cellular waste product. Atmospheric air, which contains mostly nitrogen and oxygen, is inhaled through the upper respiratory system into the lungs. The oxygen passes into the circulation and carbon dioxide is exhaled out from the lungs through the mouth and nose along with the nitrogen and other gases the body does not use. This is the process known as Breathing. Oxygen is necessary for cellular processes, but the gas exchange that occurs in the lungs is also important for maintaining blood pH. The exchange occurs due to differences in the pressure in the lungs, atmosphere and blood. Exhalation occurs passively**,** whereas inhalation requires energy (active process). Chronic obstructive pulmonary disease (COPD) is a common condition, the prevalence of which is increasing in developed countries, as is the mortality rate.[3] In most countries, primary care clinicians diagnose and treat the majority of patients with chronic respiratory diseases, as exemplified by the UK and the Netherlands where approximately 85% of patients with asthma and COPD are managed almost entirely by general practitioners and primary care nurses (Van Schayck *et al*., 2006).

In the early stages the disease can be non-symptomatic, although episodes with coughing and sputum production may occur. Other than during the earliest stages of the disease, COPD has a great impact on health care systems and causes increasing costs to society due to absence from work, doctor office visits, medication, and hospital admissions.[3] COPD is usually diagnosed in the later stages when significant lung function has already been lost or, in extreme cases, patients are not diagnosed until they are hospitalised for an acute exacerbation.[5] The diagnosis of COPD in developing countries is even more difficult as these countries often lack the technical resources and expensive equipment for objective diagnosis. There are many barriers to early diagnosis. One is the delay due to the patient's gradual adaptation to decreasing lung function. Another barrier for early diagnosis of COPD is the 'doctor's/health care delay' – for example, poor access to spirometers or limited use of spirometers in primary care.

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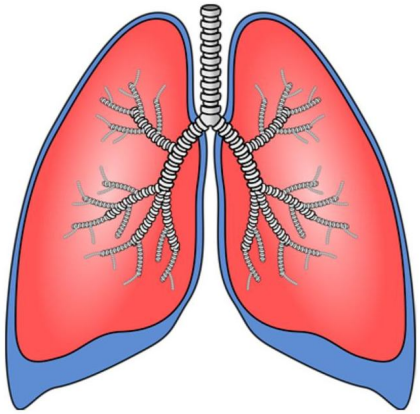


MATHEMATICAL MODELING

Number of workers as Mishra and Pandey 2003, Misra and Ghosh 2004 and Liformaggia and others have worked on blood transport and have given their views. So taking the example up these works I have taking these problem just to express the transportation and flow of blood in vessels. Similarly Mishra and ghosh in 2004 gave their views using the oscillatory flow in elastic vessels their study pertains to the case where the motion is oscillatory. So the velocity distribution in the small vessels. So the velocity at any radial station is more than that of uniform vessels. In pulmonary circulation the blood flow is quite different then the circulation in cylindrical duped and for which several reasons have been observed.

- The blood is on important and ideal fluid which is a mixture of plasma and blood cells white flowing through different vessels it changes its shapes & size. Veerendra.
- In aorta and pulmonary arteries the blood flows due to unusual high Reynolds's number of flow as 5000 to 10000 Veerendra.
- Looking to the physical nature of blood it does not behave like a newtonion flow it because hematocrits are there.
- For mathematical modeling of blood flow it is not proper because of some reasons that why general fluid equationsis applied.

As we know that principal of conservations and momentum which is applicable to hemodynamics, hence the equation of motion based on this principle has been obtained in tensorial from. Finally, the equations of coutinuity, motion have been derived for two phase blood flows which are usually used in different two phased models of blood flows of this dissertation.

Review of Literature

In year 2009 – Van Hirtum; Cisonni J; pelorson, X suggested that the position of flow separation along a constriction is important to model fluid – structure interavtion phenomena in the upper is airways such as phonation and obstructive sleep apnea. In particular, grometrical and flow features determining the influence of viscosity are varied. boundry layer solutions and simulations with the two-dimensional Navier Stokes equatios result in an accurate quantitative prediction of flow separation. The assumption applied in quasi –one-dimensional flow descriptions does not accurately predict flow separation (Van Hirtum *et al*., 2009).

In year 2010 - proshin, A.P.; Solodyannikov, yu. V. consider a formulation of the problem of parametric identification from measurements of periodic motion. And also consider the method's practical application using the example of factor analysis of the origin of arterial hypertension and also applications in medicine and sports, including the noninvasive monitoring of the level of blood hemoglobin and some types of stimulants (Proshin *et al*., 2012).

In year 2011 - Mustapha, Norzieha; Mandal, prashanta K.; Abdullah Ilyani ; Amin, Norsrahaida ; Hayat, tasawar focus that the numerical investigations of the generalized Newtonian blood flow through a couple of irregular arterial stenoses. The Marker and Cell (MAC) methoddeveloped for the governing unsteady generalized Newtonian equations in staggered grid for viscuss incompressible flow in the cylindrical polar co-ordinates system. The presented computation show that in comparison to the corresponding Newtonian model the generalized Newtonian fluid experiences higher pressure drop, lower peak wall shear stress and smaller separation region (Mustapha *et al*., 2011).

In year 2011 - Crosett simone; Konataxakis, Dimitrios; Stergiopulos, Nikolaos; Quarternoni, Alfio suggested that the numerical tools to simulate blood flow in the codiovascular system are constantly developing due to the great clinical interest and to scientific advance in mathematical models and computational power. The present work aims to address and validate niw algorithms to efficiently predict the hemodynamics in large arteries. The results of the simulations are in agreement with the physiological data in terms of wall shear, wall displacement, pressure, waveforms and velocities (Crosett simone *et al*., 2011).

Equation of continuity

The flow of blood is affected by the presence of blood cells. This effect is directly proportional to the volume occupied by blood cells. Let the volume portion covered by blood cells in unit volume be X ,this X is replaced by$1/100$, where H is the Hematocrit the volume percentage of blood cells. Then the volume portion covered by the plasma will be $(1-X)$. If mass ratio of cells to plasma is r then clearly

$$r = \frac{X\rho_c}{(1-X)\rho_p} \qquad (1)$$

where ρ_c and ρ_p are densities of blood cells and plasma respectively. Usually this mass ratio is not a constant. Even then this may be supposed to constant in present context. Hence according to the principle of conservation of mass in pulmonary circulatory system, equation of continuity for two phase are as follow (Kanpur 1992).

$$\frac{\partial(X\rho_c)}{\partial t} + (X\rho_c V^i)_{,i} = 0 \qquad \ldots\ldots (2)$$

and $$\frac{\partial(1-X)\rho_p}{\partial t} + ((1-X)\rho_c V^i)_{,j} = 0 \qquad \ldots..(3)$$

Where V is the common velocity of two phase blood cells and plasma. Again $(X\rho_c V^i)_{,j}$ is co-variant derivative of $(X\rho_c V^i)$ with respect toX^i. In the same way $((1-X)\rho_p V^i)_{,i}$ is the co-variant derivative of $((1-X)\rho_p V^i)$ with respect to X^i .

If we define the uniform density of blood ρ_m as follows

$$\frac{1+r}{\rho_m} = \frac{r}{\rho_c} + \frac{1}{\rho_p} \quad \ldots\ldots\ldots\ldots (4)$$

Then equation (2) and (3) can be combined together as follow,

$$\frac{\partial \rho_m}{\partial t} + (\rho_m V^i)_{,i} = 0 \quad \ldots\ldots\ldots\ldots (5)$$

Equation of motion for blood-flow

The hydro dynamical pressure p between the two phases of blood can be supposed to be uniform because the both phases i.e. blood cells and plasma is always in equilibrium state in blood [12]. Taking viscosity coefficient of blood cells to be η_c and applying the principle of conservation of momentum in pulmonary circulatory system, we get the equation of motion for the phase of blood cells as follows:

$$\frac{\partial v^i}{\partial t} + (X\rho_c V^i) V^i_{,j} = -X p_{,j} g^{ij} + X\eta_c (g^{ij} V^i_{,k})_{,j} \quad \ldots\ldots\ldots (6)$$

Similarly, taking the viscosity coffecient of plasma to be η_p, the equation of motion for plasma will be as follows :

$$(1-X)\rho_p \frac{\partial v^i}{\partial t} + \{(1-X)\rho_p V^i\} V^i_{,j} = -(1-X) p_{,j} g^{ij} + (1-X)\eta_c (g^{ij} V^i_{,k})_{,j} \quad \ldots\ldots (7)$$

Now adding equation (6) and (7) and using relation(4), the equation of motion for blood flow with the both phases will be as follows:

$$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m V^j) V^i_{,j} = - p_{,j} g^{ij} + \eta_m (g^{ij} V^i_{,k})_{,j} \quad \ldots\ldots\ldots (8)$$

Where $\eta_m = X\eta_c + (1-X)\eta_p$ is the viscosity coefficient of blood as a mixture of t

The pumping effect of heart on these vessels is very low. The yield stress derived from this phenomenon increases the blood viscosity ten times. Hence the pumping of the heart on these vessels is relatively low (Van Hirtum *et al.*, 2009). Secondly these vessels are relatively narrow down more rapidly. In this situation, the blood cells line up on the axis to build up rouleaux. Hence a yield stress is produced. Though this yield stress is very small**,** even then the viscosity of blood is increased nearly ten times [47]

That's why the Herschel-Bulkley Law holds good on the two phase blood flow through veins arterioles, venules and whose constitutive equation is as follows:

$T' = \eta_m e^n + T_p \ (T' \geq Tp)$ *and*
$e = 0 (T' < T_p)$

Where T is the yield stress.

When strain rate $e=0(T'<T_p)$ a core region is formed which flows just like a Plug. Let the radius of the plug be r_p. The stress acting on the surface of the plug will be T. Equating the forces acting on the plug, we get [Fig. (1)]

$$P \pi r_p^2 = T_p 2 \pi r_p$$

$$\Rightarrow \quad r_P = \frac{2T_P}{P} \quad \ldots\ldots\ldots\ldots (9)$$

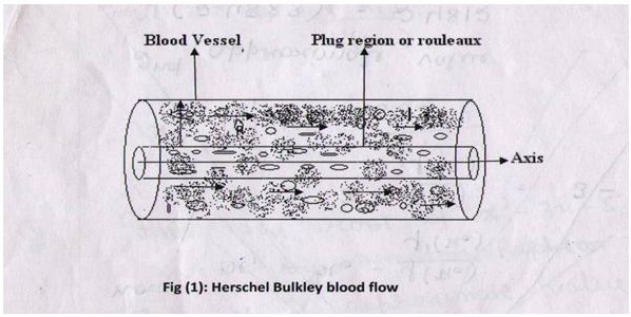


Fig (1): Herschel Bulkley blood flow

The consecutive equation for rest part of blood vessel is

$T' = \eta_m e^n + T_p$
$T' - T_p = \eta_m e^n = T_e$

Where T_e is the effective stress.

Whose generalized form will be as follows:

$$T^{ij} = -pg^{ij} + T^{ij}_e$$

Where the symbols have their usual meanings.

Now we describe the basic equation for Herschel-Bulkley flow as follows:

Equation of continuity-

$$\frac{1}{\sqrt{g}} (\sqrt{g} v^i) = 0$$

The equation of motion -

$$\rho_m \frac{\partial v^i}{\partial t} + \rho_m v^j v^i_{,j} = -T^{ij}_{,j} \quad \ldots\ldots\ldots (10)$$

Where all the symbols have their usual meanings.

Analysis

Since the blood vessels are cylindrical, the above governing equations are transformed into cylindrical form. As we know earlier

$x^1 = r, \ x^2 = \theta, x^3 = z$

Matrix of metric tensor in cylindrical co-ordinates is as follows

$$[g^{ij}] = \begin{bmatrix} 1 & 0 & 0 \\ 0 & r^2 & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

While matrix of conjugate metric tensor is as follows:

$$[g_{ij}] = \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1/r^2 & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

Whereas the Christoffel's symbols of 2nd kind as follows:

$$\begin{Bmatrix} 1 \\ 2\ 2 \end{Bmatrix} = -r\begin{Bmatrix} 2 \\ 2\ 1 \end{Bmatrix} = \begin{Bmatrix} 1 \\ 1\ 2 \end{Bmatrix} = \frac{1}{r}$$

remaining others are zero.

Relation between contravarient and physical components of velocity of blood flow will be as follows:

$$\sqrt{g_{11}v^1} = v_r \Rightarrow v_r = v^1$$
$$\sqrt{g_{22}v^2} = v_\theta \Rightarrow v_\theta = rv^2$$
$$\sqrt{g_{33}v^3} = v_z \Rightarrow v_z = v^3$$

Again the physical components of $-p_j g^{ij}$ are $-\sqrt{g_{ii}}\ p_j g^{ij}$ Equation (9) and (10)are transformed into cylindrical form so as solve as power law model to get

$$\frac{dv}{dr} = \left(\frac{Pr}{2\eta_m}\right)^{1/n}$$

Where pressure gradient $\frac{dp}{dz} = P$

$$\frac{dv}{dr} = \left(\frac{P(r-r_p)}{2\eta_m}\right)^{1/n}$$

$$\frac{dv}{dr} = \left(\frac{\frac{1}{2}Pr - \frac{1}{2}Pr_P}{2\eta_m}\right)^{1/n}$$

From equation (9)

$$\frac{dv}{dr} = \left\{\frac{\frac{1}{2}Pr - T_P}{\eta_m}\right\}^{1/n} \quad \ldots\ldots\ldots\ldots (11)$$

Substituting the value of T'_o from (7) into (11)

$$\frac{dv}{dr} = \left(\frac{pr/2 - pr_p/2}{\eta_m}\right)^{1/n}$$

$$\Rightarrow \quad \frac{dv}{dr} = \left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} (r-r_p)^{1/n} \quad \ldots\ldots\ldots\ldots\ldots (12)$$

Integrating above equation (12) under the no slip boundary condition $v = 0$ at $r = R$, so as to get:

$$v = \left(\frac{P}{2\eta_m}\right)^{1/n} \frac{n}{n+1}\left[(R-r_p)^{\frac{1}{n}+1} - (r-r_p)^{\frac{1}{n}+1}\right] \quad \ldots\ldots\ldots (13)$$

Which is the formula of velocity of blood flow in arterioles, venules and veins. Putting $r = r_p$ to get he velocity v_p of plug flow as follows:

$$v_p = \frac{n}{n+1}\left(\frac{P}{2\eta_m}\right)^{1/n}\left(R-r_P\right)^{\frac{1}{n}+1} \quad \ldots\ldots\ldots\ldots (14)$$

Where the value of r_p is taken from (7).

RESULT AND DISCUSSION

Observation: Hametocrit vs blood pressure from an authorized. G.S.V.M. medical college Kanpur by Dr. Avdhesh Kumar
Patient name: shiv kumar
Diagnosis: COPD (Chronic Obstructive Pulmonary Disease)

Date	HB(Hemoglobin)	B.P.(Blood Pressure)	Hematocrit
16-o7-2012	6.7	100/70	20.1
18-07-2012	6.9	120/80	20.7
20-07-2012	6.9	130/80	20.7
24-07-2012	7.0	120/80	21.0
26-07-2012	7.4	110/80	22.2

the flow flux phased blood flow in arterioles, venules and veins is

$$Q = \int_o^{r_p} 2\pi r v_p dr + \int_{r_p}^{R} 2\pi r v dr$$

$$= \int_0^{r_p} 2\pi r \frac{n}{n+1}(p/2\eta_m)^{1/n}(R-r_p)^{1/n+1}dr + \int_0^{r_p} 2\pi r \frac{n}{n+1}(p/2\eta_m)^{1/n}$$

$$[(R-r_p)^{1/n+1} - (r-r_p)^{1/n+1}]dr$$

Using (12) and (13)

$$= \frac{2\pi n}{n+1}(P/2\eta_m)^{1/n}(R-r_p)^{1/n+1}\left[r^2/2\right]_0^{r_p} +$$

$$\frac{2\pi n}{n+1}(P/2\eta_m)^{1/n}\left[r^2/2(R-r_p)^{1/n+1} - \frac{r(r-r_P)}{\frac{1}{n}+2} + \frac{(r-r_P)^{\frac{1}{n}+3}}{(\frac{1}{n}+2)(\frac{1}{n}+3)}\right]_{r_P}^{R}$$

$$= \frac{2\pi n}{(n+1)}(P/2\eta_m)^{1/n} r_p^2 (R-r_p)^{\frac{1}{n}+1} + R^2(R-r_p)^{\frac{1}{n}+1}$$

$$- \frac{2R(R-r_p)^{\frac{1}{n}+2}}{\frac{1}{n}+2} + \frac{2(R-r_p)^{\frac{1}{n}+3}}{(\frac{1}{n}+2)(\frac{1}{n}+3)} - r_P^2(R-r_p)^{\frac{1}{n}+1}$$

$$= \frac{2\pi n}{(n+1)}(P/2\eta_m)^{1/n} R^{\frac{1}{n}+3}\left[\frac{r_p^2}{R^2}(1-\frac{r_p}{R})^{\frac{1}{n}+1} + (1+\frac{r_p}{R})(1-\frac{r_p}{R})^{\frac{1}{n}+2}\right.$$

$$\left. - \frac{2(1-\frac{r_p}{R})^{\frac{1}{n}+2}}{(\frac{1}{n}+2)} + \frac{2(1-\frac{r_p}{R})^{\frac{1}{n}+3}}{(\frac{1}{n}+2)(\frac{1}{n}+3)}\right]$$

P = Pressure gradient
v = Viscosity of mixture (Blood)
n = Parameter

Now we have,

Q = **425** *ml/min*
R = 1, r_p= 1/3
η_m = 0.027 Pascal second
η_P = 0.0013 (Pascal-sec) and
H = 20.1 P = 100

We also know that

$$\eta_m = \eta_c X + \eta_P (1 - X),$$

Where $X = \frac{H}{100} = \frac{20.1}{100} = 0.201$

$\Rightarrow$ 0.027 = η_c (0.201) + (0.0013) (1-0.201)
$\Rightarrow$ η_c = 0.129160

Again,

$$\eta_m = (0.1291)\frac{H}{100} + (0.0013)\left(1 - \frac{H}{100}\right)$$

$$\eta_m = (0.0453)(0.201) + (0.0013)(0.799)$$

$$\eta_m = 0.02698$$

$$1827.23 = (1728.40)^{1/n}\left[\frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}\right]$$

Solving above equation by numerical method, we get n= 0.9955

$$P = ((0.000453)\,H + 0.0039)\left[1827.23 \times \frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}\right]^n$$

P= ((0.001291)H+ 0.0039) (5637.18) (0.2868) at n = 0.9955

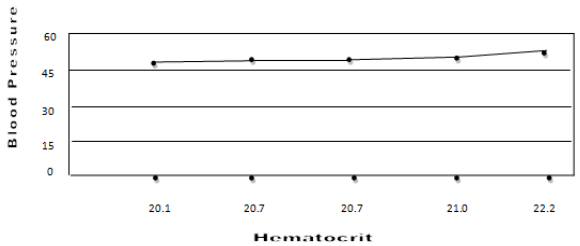
at H = 20.1 P = 48.258 ≈ 48

at H = 20.7 P = 49.510 ≈ 49

at H= 20.7 P = 49.510 ≈ 49

at H= 21.0 P = 50.136 ≈ 50

at H= 22.2 P = 52.641 ≈ 52



Conclusion

A simple survey of the graph between blood pressure and hematocrit in COPD (chronic obstructive pulmonary diaease) patient shows that when Hematocrit is increased then blood pressure also increased. Hence Hematocrit is directly propotional to blood pressure. i.e.

$$\text{Hematocrit} \propto \text{Blood pressure}$$

Remark

If this would have been possible to get blood pressure on the particular tissue then the relation between blood pressure and hamatocrit has been measured more accurately.

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