

Mathematical Modeling on Two Phase Hepatic Mean Blood Flow through in Liver Lobule with Special Reference to Hepatitis A

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ABSTRACT: In this paper, we develop a mathematical model of blood circulation in the liver lobule, keeping in view the nature of Hepatic circulatory system in human body. The viscosity increases in the arterioles due to formation of rouleaux along the axis of red blood cells, as we know the arterioles are remote from heart and proximate to the Liver Lobule. The liver can be divided into functional units called lobules. Each lobule has a hexagonal cross-section, and we assume that its longitudinal extent is large compared with its width. We consider an infinite lattice of identical lobules and study the two-dimensional flow in the hexagonal cross-sections. P.N. Pandey and V. Upadhyay have considered the blood flow has two phased, one of which is that of red blood cells and other is plasma. They have also applied the Herschel Bulkley non-Newtonian model in bio fluid mechanical set-up. We have collected a clinical data in the case of Hepatitis A for Hematocrit versus Blood Pressure. The overall presentation is in tensorial form and solution technique adapted is analytical as well as numerical. The role of Hematocrit is explicit in the determination of blood pressure in case of Hepatitis A infection. The graphical presentation for particular parametric value is much closer to the clinical observation.

KEYWORDS: Hepatitis A, Hematocrit, Blood Flow, Herschel Bulkley non-Newtonian model, circulatory system.

I. INTRODUCTION

The liver is the largest gland in the body, weighing around 1.5 kg in normal adults. It receives blood from two main sources: Approximately 80% is partially deoxygenated blood from the hepatic portal vein, and the remainder is fully oxygenated blood supplied by the hepatic artery^[1]. The basic functional unit of the liver is the liver lobule, which is a cylindrical structure several millimeters in length and 0.8 to 2 millimeters in diameter. The human liver contains 50,000 to 100,000 individual lobules. The Liver functions as a blood reservoir because the liver is an expandable organ, large quantities of blood can be stored in its blood vessels. Its normal blood volume, including both that in the hepatic veins and that in the hepatic sinuses, is about 450 milliliters, or almost 10 per cent of the body's total blood volume^[2].

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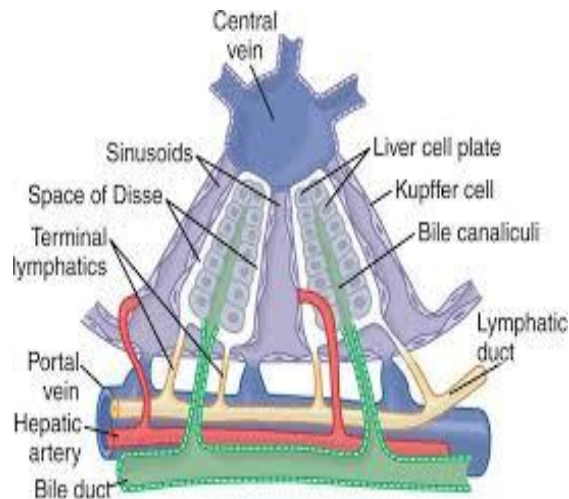


Figure 1. Basic Structure of Liver Lobule

Each lobule has an approximately hexagonal cross-section, with a centrilobular vein in the center surrounded by sinusoids. The surface bounding the lobule is formed by the vascular septa (that correspond to the edges of the hexagon) and portal tracts (at the vertices). Each portal tract contains three vessels: a branch of the hepatic portal vein, a branch of the hepatic artery, and a bile duct^[3-4]. Blood flows through the walls of the portal tract into the sinusoidal space, then traverses the sinusoids, and flows into the centrilobular vein. The overall resistance of the small liver to blood flow is greater than that of a full liver, and this may result in high blood pressures in the sinusoids, leading to damage to the liver^[5].

The disease described as “jaundice” in ancient Greek, Roman, and Chinese literature probably was viral hepatitis. A viral etiology was postulated as the cause of certain forms of jaundices early as 1912, and the term “infectious hepatitis” was used because the disease often occurred in epidemics^[6]. Hepatitis A, a term first introduced by Krugman et al. in 1967^[7], is now known to be caused by infection with hepatitis A virus (HAV), one of five viruses, each belonging to a different family, whose primary site of replication is the liver. In 1973, HAV was identified in the stools of infected persons^[8], which eventually led to development of diagnostic tests, propagation in cell culture, molecular characterization, and development of a vaccine^[8, 9].

Infection by HAV confers life-long immunity and can produce effects that range from asymptomatic to fulminant hepatic failure, which in some cases can cause death^[10]. However, the fatality rate in HAV infections is lower than 0.1%, and the higher risk is usually exposed to young children and older adults with underlying chronic liver disease^[11]. The likelihood of clinically apparent disease associated with HAV infection increases with age (18). More than 70% of cases of HAV infection occur in children less than 6 years old are asymptomatic, or, if illness occurs, it is not accompanied by jaundice^[12]. However, in older children and adults, HAV infection causes more-severe clinical illness, including jaundice malaise, fever and dark urine, in 70% of cases^[13].

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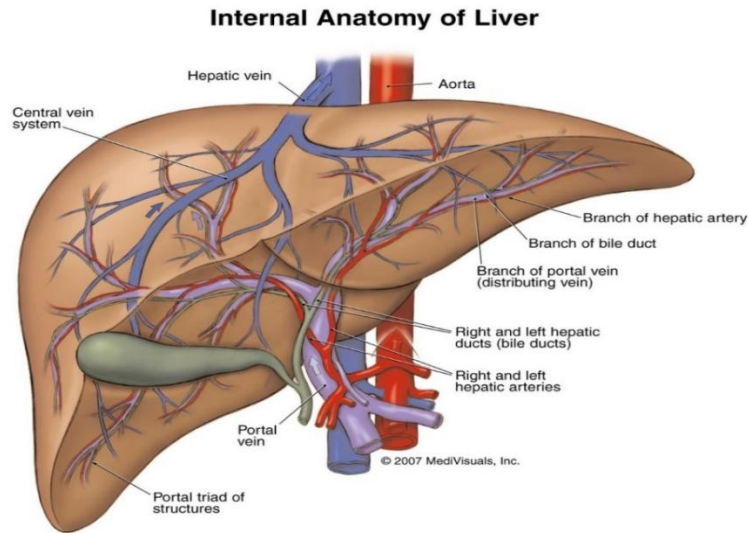


Figure 2. Internal Anatomy of Liver

Blood is a complex fluid consisting of particulate corpuscles suspended in a non-Newtonian fluid. The particulate solids are red blood cells (RBCs), white blood cells (WBCs) and platelets. The fluid is plasma, which constitutes 55% of blood fluid is mostly water (92% by the volume)^[14]. Blood accounts for 7% of the human body weight^[15,16]. By volume, the red blood cells constitute about 45% of whole blood, the plasma about 54.3% and white cells about 0.7%^[17]. The percentage of volume covered by blood cells in the whole blood is called hematocrit. Two phase hepatic blood flow is a study of measuring the blood pressure if hemoglobin known.

II. EQUATION OF CONTINUITY FOR TWO PHASE BLOOD FLOW

The blood flow is affected by the presence of blood cells. This effect is directly proportional to the volume occupied by blood cells^[18]. Let the volume portion covered by blood cells in unit volume be X , this X is replaced by $H/100$, where H is the Hematocrit the volume percentage of blood cells. Then the volume portion covered by the plasma will be $(1 - X)$. The mass ratio of blood cells to plasma is given below:

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

Where ρ_c and ρ_p are densities of blood cells and blood plasma respectively. Usually this mass ratio is not constant, even then it may be supposed to constant in present context^[19].

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The both phase of blood, i.e. blood cells and plasma move with the common velocity. Campbell and Pitcher has presented a model for two phase of blood separately^[20].The equation of continuity for two phases according to the principle of conservation of mass defined by Kapur J.N.and Gupta R.C.^[19]as follow:

$$\frac{\partial(X\rho_c)}{\partial t} + (X\rho_c v^i), i = 0 \quad \dots\dots\dots(2)$$

And

$$\frac{\partial(1-X)\rho_p}{\partial t} + ((1-X)\rho_p v^i), i = 0 \quad \dots\dots\dots (3)$$

Where, v is the common velocity of two phase blood cells and plasma. If we define the uniform density of the blood ρ_m as follow:

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

From equation (2) and (3) we can written as,

$$\frac{\partial\rho_m}{\partial t} + (\rho_m v^i), i = 0 \dots\dots\dots(5)$$

Where $\rho_m = X\rho_c + (1-X)\rho_p$

III. EQUATION OF MOTION FOR TWO PHASE 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 are always in equilibrium state in blood^[21]. Taking viscosity coefficient of blood cells to be η_c and applying the principle of conservation of momentum, we get the equation of motion for the phase of blood cells as follows:

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

The equation of motion for plasma will be as follows:

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$$(1-X)\rho_p \frac{\partial v^i}{\partial t} + \left\{ (1-X)\rho_p v^j \right\} v^i_{,j} = -(1-X)p_{,j} g^{ij} + (1-X)\eta_p (g^{jk} v^i_{,k})_{,j} \quad \dots\dots\dots (7)$$

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:

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

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

IV. MATHEMATICAL MODEL

As the velocity of blood flow decreases, the viscosity of blood increases. The velocity of blood decreases successively. The Herschel Bulkley law hold good on the two phase blood flow through veins arterioles, veinules and whose constitutive equation is as follows:

$$T' = \eta_m e^n + T_p (T' \geq T_p) \text{ and } e = 0 (T' < T_p) \text{ where, } T_p \text{ is the yield stress.}$$

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

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

$$\Rightarrow r_p = 2 \frac{T_p}{p} \quad \dots\dots\dots (9)$$

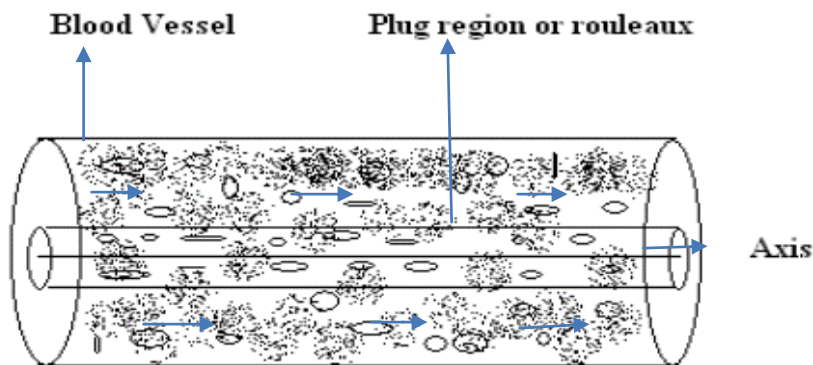


Figure 3. Herschel Bulkley blood flow model

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The Constitutive equation for test part of the blood vessel is given by

$$T^i = \eta_m e^n + T_p \text{ or } T^i - T_p = \eta_m e^n = T_e$$

Where, T_e =effective stress, whose generalized form are given below:

$$T^{ij} = -Pg^{ij} + T_e^{ij} \text{ Where, } T_e^{ij} = \eta_m (e^{ij})^n \text{ while } e^{ij} = g^{jk} v_k^i$$

Now we describe the basic equations for Herschel Bulkley blood flow as follows:

The equation of Continuity is given below:

$$\frac{1}{\sqrt{g} \sqrt{(gV^i)_{,i}}} = 0 \tag{10}$$

The equation of motion is:

$$\rho_m \frac{\partial v^i}{\partial t} + \rho_m V^j V_{,j}^i = -T_{e,j}^i \tag{11}$$

Where all the symbols have their usual meanings, since, the blood vessels are cylindrical; the above governing equations have to be transformed into cylindrical co-ordinates.

As we know $x^1 = r, x^2 = \theta, x^3 = z$, Matrix of metric tensor in cylindrical co-ordinates is $[g_{ij}]$ and matrix of conjugate metric tensor is $[g^{ij}]$ whereas the Chritoffel's symbols of 2nd kind are as follows:

$$\left\{ \begin{matrix} 1 \\ 2 \end{matrix} \right\} = -r, \left\{ \begin{matrix} 1 \\ 2 \end{matrix} \right\} = \left\{ \begin{matrix} 1 \\ 2 \end{matrix} \right\} = \frac{1}{r} \text{ Remaining others are zero.}$$

The governing tensorial equations can be transformed into cylindrical forms which are as follows:

The equation of continuity are given below

$$\frac{\partial v}{\partial z} = 0$$

The equation of motion: r-component: $-\frac{\partial p}{\partial z} = 0$, θ -component: $0 = 0$ and

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$$\text{Z-component: } 0 = -\frac{\partial p}{\partial z} + \frac{\eta_m}{r} \left[r \left(\frac{\partial v_z}{\partial r} \right)^n \right]$$

Here, this fact has been taken in view that the blood flow in axially symmetric in arteries concerned, i.e. $v_\theta = 0$ and v_r, v_z and p do not depend upon θ .

$$\text{We get } v_z = v(r) \text{ and } dp = p(z) \text{ and } 0 = -\frac{dp}{dz} + \frac{\eta_m}{r} \left[r \left(\frac{dv}{dz} \right)^n \right] \dots\dots\dots(12)$$

And the pressure gradient $-\frac{dp}{dz} = p$

$$r \left(\frac{dv}{dz} \right)^n = -\frac{pr^2}{2\eta_m} + A, \text{ We apply boundary condition: at } r = 0. V = V_0 \text{ then } A = 0$$

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

Replace r from $r - r_p$

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

$$\text{Or } \frac{dv}{dr} = -\left(\frac{p}{2\eta_m} \right)^{\frac{1}{n}} (r - r_p)^{\frac{1}{n}} \dots\dots\dots(13)$$

Integrating above equation (13) under the no slip boundary condition: $v = 0$ at $r = R$ we get:

$$V = \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{n}{n+1} \left[(R - r_p)^{\frac{1}{n}+1} - (r - r_p)^{\frac{1}{n}+1} \right] \dots\dots\dots (14)$$

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This is the formula for velocity of blood flow in arterioles, veinules and veins. Putting $r = r_p$ to get the velocity V_p of plug flow are given by,

$$V_p = \frac{n}{n+1} \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} (R - r_p)^{\frac{1}{n}+1} \dots\dots\dots (15)$$

Where the value of r_p is taken from equation (7), we have

V. RESULT & DISCUSSION (BIO-PHYSICAL INTERPRETATION)

Observation: Hematocrit Vs. Blood pressure Sharda Hospital, School of Medical Science and Research Centre Greater Noida by Dr. Mamta kumari

Patient Name: Mrs. Sunita

Diagnosis: Hepatitis A

S. No.	Dated	HB (Hemoglobin)	B.P. (in mmhg)	Hematocrit
1	29/12/2014	12.0	110/80	36.0
2	30/12/2014	12.5	120/80	37.5
3	1/01/2015	13.4	124/90	40.2
4	2/01/2015	14.2	120/88	42.6
5	3/01/2015	14.9	112/84	44.7

Table 1

The hematocrit is normally about three times the hemoglobin concentration^[22]. The flow flux of two phased blood flow in arterioles, veinules and veins is given below:

$$Q = \int_0^{r_p} 2\pi r V_p dr + \int_{r_p}^R 2\pi r V dr$$

$$Q = \int_0^{r_p} 2\pi r \frac{n}{n+1} \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} (R - r_p)^{\frac{1}{n}+1} dr + \int_{r_p}^R 2\pi r \frac{n}{n+1} \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \left[(R - r_p)^{\frac{1}{n}+1} - (r - r_p)^{\frac{1}{n}+1} \right] dr$$

Using (13) and (15), we get

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$$Q = \frac{\pi n}{(n+1)} \left(\frac{p}{2\eta_m}\right)^{\frac{1}{n}} R^{\frac{1}{n+3}} \left[\frac{r_p^2}{R^2} \left(1 - \frac{r_p^2}{R}\right)^{\frac{1}{n+1}} + \left(1 + \frac{r_p}{R}\right) \left(1 - \frac{r_p}{R}\right)^{\frac{1}{n+2}} - \frac{2 \left(1 - \frac{r_p}{R}\right)^{\frac{1}{n+2}}}{\left(\frac{1}{n} + 2\right)} + \frac{2 \left(1 - \frac{r_p}{R}\right)^{\frac{1}{n+3}}}{\left(\frac{1}{n} + 2\right) \left(\frac{1}{n} + 3\right)} \right] \dots$$

(16)

$$Q = 0.0167l / \text{sec}, R = 1, r_p = \frac{1}{3} \quad [23]$$

$$\eta_p = 0.0015 \text{ (Pascal-sec.)} \quad [24]$$

$$\eta_m = 0.035 \text{ (Pascal - sec)}, H = 40.2$$

Substituting the value of η_m and η_p in the relation $\eta_m = X\eta_c + (1-X)\eta_p$ where $X = \frac{H}{100}$ we get, $\eta_c = 0.0848$ and again using same relation we get,

$$\eta_m = 0.000833H + 0.0015 \quad \dots \quad (17)$$

Substituting the value of r_p and R in equation (16), we get

$$Q = \pi \left(\frac{2P}{6\eta_m}\right)^{\frac{1}{n}} \left(\frac{2}{27}\right) \left[\frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1} \right]$$

$$\frac{27Q}{2\pi} = \left(\frac{P}{3\eta_m}\right)^{\frac{1}{n}} \left[\frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1} \right] \quad \dots \quad (18)$$

$$\frac{P}{3\eta_m} = \left(\frac{27Q}{2\pi A}\right)^n \quad \text{Where } A = \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$\Rightarrow P = \left(\frac{27Q}{2\pi A}\right)^n \times 3\eta_m$$

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Pressure gradient $-\frac{dp}{dz} = P$

$$-dp = Pdz$$

$$\int_{p_f}^{p_i} dp = -\int_{z_f}^{z_i} \left(\frac{27Q}{2\pi A}\right)^n \times 3n_m dz$$

$$p_i - p_f = \left(\frac{27Q}{2\pi A}\right)^n \times 3n_m (z_f - z_i) \dots\dots\dots(19)$$

Where,

Pressure drop = $p_i - p_f$

Length of hepatic arterioles = $z_f - z_i = 0.00005$ meter^[25]

Substituting the value of Q , p_i , p_f , n_m in equation (18) and solve by Numerical method we get,

$n = -4.8591$, Again from equation (19)

$$p_i - p_f = 0.00015 \times \left(\frac{27Q}{2\pi A}\right)^n \times n_m$$

Using equation (17) and putting the value of Q and n we get,

$$p_i - p_f = 103.8315H + 186.9716$$

From above equation we get blood pressure drop, if hematocrit is known.

H(Hematocrit) (g/dl)	36	37.5	40.2	42.6	44.7
P (Blood Pressure drop)(Pascal)	3924.9056	4080.6529	4360.9979	4610.1935	4828.2396

Table 2

Table 2 shows change in blood pressure drop with increase in hemoglobin. The graph of above table is given in figure-4.

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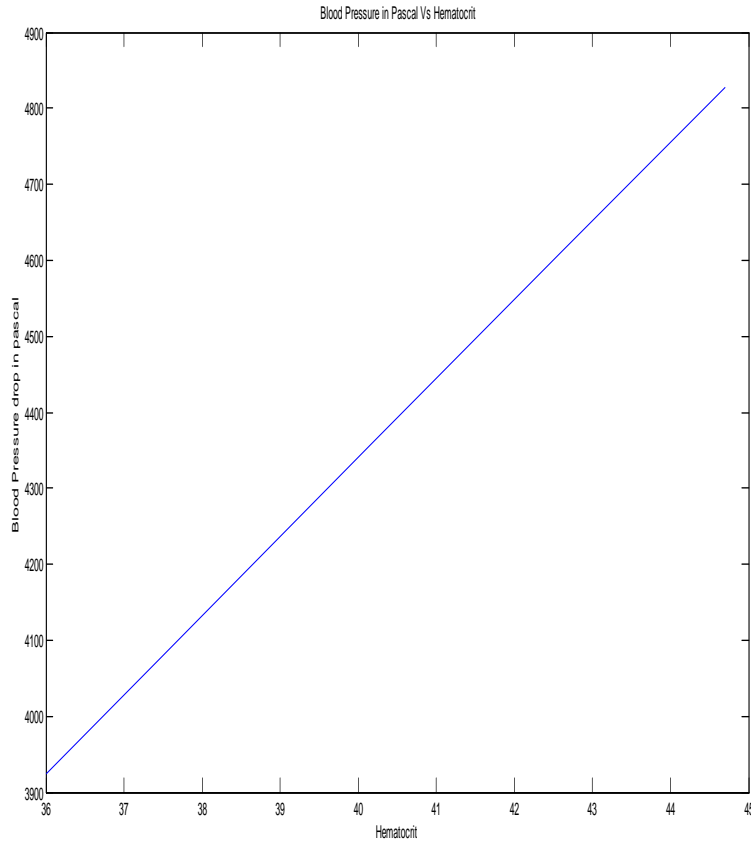


Figure 4

VI. CONCLUSION

1. The two phase model is $\eta_m = X\eta_c + (1-X)\eta_p$ where $X = \frac{H}{100}$ and H is Hematocrit which is 3 times of Hemoglobin. Explained the role of Hematocrit in determination of blood pressure drop.
2. Figure 4 shows that Blood pressure drop is linearly ratio to Hematocrit i.e. directly proportional to Hematocrit where slope is 103.8315 Pascal dl/g.

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