International Journal of Statistics and Applied Mathematics

**ISSN:** 2456-1452
Maths 2023; 8(3): 234-241

https://www.mathsjournal.com
Received: 21-03-2023
Accepted: 22-04-2023

**Ram Naresh Yadav**
1) Research Scholar, Assistant Professor, MGCG Vishwavidyalay Chitrakoot Satna, Madhya Pradesh, India
2) GT Govt., PG College Karwi Chitrakoot, Uttar Pradesh, India

**V Upadhyay**
Associate Professor, MGCG Vishwavidyalay Chitrakoot, Satna, Madhya Pradesh, India

**AK Agrawal**
Associate Professor MGCG Vishwavidyalay Chitrakoot, Satna, Madhya Pradesh, India

**Corresponding Author:**
**Ram Naresh Yadav**
1) Research Scholar, Assistant Professor, MGCG Vishwavidyalay Chitrakoot Satna, Madhya Pradesh, India
2) GT Govt., PG College Karwi Chitrakoot, Uttar Pradesh, India

# Mathematical model on two phase of hepatic blood flow in venules with special reference to malaria

**Ram Naresh Yadav, V Upadhyay and AK Agrawal**

**DOI:** https://doi.org/10.22271/maths.2023.v8.i3c.1039

**Abstract**
In this paper, we analyzed blood flow in hepatic venules. The viscosity of blood increases in venules due to more formation of rouleaux along the axis of red blood cells as at the same venules are remote from the heart and proximate to liver so, we applied the Herschel-Bulkley non-Newtonian model in Bio-fluid mechanical setup. Employing Navier-Stoke equation and equation of continuity in cylindrical co-ordinate system and all required mathematical formations are in tensorial form. Using by numerical method we calculated the value of parameter for a clinical data. Finally, we obtained a linear relationship between blood pressure drop and hematocrit for particular value of parameter and discussed in the graph.

**Keywords:** Hepatic circulation, malaria, hematocrit, viscosity, venule, Rouleaux

## 1. Introduction

### 1.1 Structure and Function of Liver

The liver has the most complicated circulation of any one of the organ. According to the anatomical peculiarity of the double afferent blood supply of the liver, 75%-80% of the blood entering the liver is partially deoxygenated venous blood provided by the portal vein, which collects all the blood that leaves the spleen, stomach, small and large intestine, gallbladder and pancreas (Vollmar, B. *et al*. 2009, Rappaport, AM *et al*. 1980) [24].

The liver has a wide range of functions including detoxification of various metabolites and toxic matter regulation of glycogen storage, decomposition of red blood cells, hormone production and the production of biochemicals necessary for digestion and other metabolic activities. The liver is the only human internal organ capable of natural regeneration of lost tissues as little as 25% of a liver can regenerate into a whole liver [10].

The portal venous system is responsible for directing blood from parts of the gastrointestinal tract of the liver. Blood flow to the liver is unique in that it receives both oxygenated and deoxygenated blood. Blood passes from branches of the portal vein through cavities between "plates" of hepatocytes called sinusoids blood also flows from branches of the hepatic artery and mixes in the sinusoids to supply the hepatocyte with oxygen. This mixture percolates through the sinusoids and collects in a central vein which drains into the hepatic vein. The hepatic vein subsequently drains into the inferior vena cava. The hepatic artery provides 30 to 40% of the oxygen to the liver, while only accounting for 25% of the total liver blood flow. The rest comes from the partially deoxygenated blood from the portal vein [3, 4].

### 1.2 Structure and Function of Venule

The hepatic microcirculation generally refers to the circulatory system beginning with portal venule, extending to the terminal portal venule, and then reaching the sinusoid network, followed by the postcapillary terminal hepatic venule and ending with the muscular venule. [27]

The basic structural unit of the hepatic microcirculation is the hepatic lobule, in which a terminal hepatic venule is located at the center and several portal venules at the periphery, with the hepatic sinusoids running from the terminal portal venule, forming a hexagonal vascular structure [28].

### 1.3 Composition of Blood

Blood is bio fluid or fluid connective tissue. Blood consists of a suspension of cells in an aqueous solution called plasma which composed of about 90% water and 7% protein. There are about 95% are red cells or erythrocytes whose main function is to transport oxygen from lungs to all the cells of the body and removal of carbon dioxide formed by metabolic process in the body to lungs. About 45% of the blood volume in an average human is occupied by red cells. This fraction is known as the hematocrit of the remaining white cells or leucocytes constitute about one sixth or 1% of total and these play an impartment role in the resistance of the body to infection and platelets form 5% of the total blood and they perform a function related to blood clotting [2, 18].

### 1.4 Description of Disease and RBC deformability in Malaria

Malaria is caused by single celled microorganisms of the *Plasmodium* group. It is spread exclusively through bites of infected *Anopheles* mosquitoes. The mosquito bite introduces the parasites from the mosquito's saliva into a person's blood. The parasites travel to the liver where they mature and reproduce [31]. The Plasmodium life cycle is very complex and takes place in two phases; sexual and asexual, the vector mosquitoes and the vertebrate hosts. In the vectors, mosquitoes, the sexual phase of parasite's life cycle occurs. The asexual phase of life cycle occurs in humans, the intermediate host for malaria [29, 30].

Red blood cells host plasmodium parasites that cause malaria, of which plasmodium falciparum is the most pathogenic. The deformability of RBC is markedly modified by invasion and development of *P. falciparum*. The deformability of RBC depends on three parameters: (i) the membrane elasticity (ii) the cytoplasmic viscosity that depends on intracellular ion and haemoglobin concentration and (iii) the surface to volume ratio. The balance among these three parameters can be altered during malaria (Lavazec, 2017).

## 2. Real Model

### 2.1 Frame of reference

In this model, we employ Navier-Stoke's equation and equation of continuity and have chosen orthogonal curvilinear generalized three-dimensional coordinate system denoted by $E^3$ called three dimensional Euclidean space of the moving blood. All quantities related to blood flow are written in tensorial form which is comparatively more realistic. Let P be any point in space with co-ordinate $x^i$ with respect to axes $OX^i$, O as origin where i = 1, 2, 3. At time t, $v^k = v^k$ ($x^i$, t) be velocity of blood, p = p ($x^i$, t) thermodynamical pressure and ρ = ρ ($x^i$, t) density. Since blood vessels are cylindrical the governing equations have to transform into cylindrical co-ordinates system

## 3. Formulation

According to Sherman I.W. and Sherman V.G. blood is mixed fluid. There are two phases in the blood, one is plasma and other is blood cells. The blood cells are enclosed with a semi–permeable membrane whose density is greater than that of plasma. These blood cells are uniformly distributed in plasma.

### 3.1 Equation of continuity for two phase blood flow

According to Upadhyay V. 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, where X=$\frac{H}{100}$ and H is hematocrit the volume percentage of blood cells. Then the volume portion covered by plasma will be 1-X. If the mass ratio of blood cells to plasma is r then

$$r=\frac{X\rho_c}{(1-X)\rho_p} \tag{3.1}$$

Where $\rho_c$ and $\rho_p$ are densities of blood cells and plasma respectively. The both phase the blood cells and plasma move with common velocity. Campbell and Pitcher have presented a model for this condition. Equation of continuity for two phase according to principle of conservation of mass defined by J.N. and Gupta R. C. as follows

$$\frac{\partial(X\rho_c)}{\partial t}+(X\rho_c v^i)_{,i}=0 \tag{3.2}$$

And

$$\frac{\partial(1-X)\rho_p}{\partial t}+\left((1-X)\rho_p v^i\right)_{,i}=0 \tag{3.3}$$

Where $v$ velocity of mixture of two is phase blood cells and plasma, $(X\rho_c v^i)_{,i}$ is covariant derivative of $(X\rho_c v^i)$ with respect to $x^i$ and $\left((1-X)\rho_p v^i\right)_{,i}$ is covariant derivative of $\left((1-X)\rho_p v^i\right)$ with respect to $x^i$.

If $\rho_m$ be uniform density of blood then

$$\frac{1+r}{\rho_m}=\frac{r}{\rho_c}+\frac{1}{\rho_p}$$

Where

$\rho_m = X\rho_c + (1-X)\rho_p$ (3.4)

Combined equation (3.2) and (3.3) and using (3.4) we get

$\frac{\partial \rho_m}{\partial t} + (\rho_m v^i)_{,i} = 0$ (3.5)

**3.2 Equation of motion for two phase blood flow-**

According to Ruch T.C. and H.D. the hydro dynamical pressure p between two phases of can be supposed to be uniform because the both phases are always in equilibrium state in blood (1973). According to principle of conservation of momentum the equation of motion of two phase blood cells and plasma

$X\rho_c \frac{\partial v^i}{\partial t} + (X\rho_c v^j) v^i_{,j} = -Xp_{,j} g^{ij} + X\eta_c \left(g^{jk} v^i_{,k}\right)_{,j}$ (3.6)

And $(1-X)\rho_p \frac{\partial v^i}{\partial t} + \{(1-X)\rho_p v^j\} v^i_{,j} = -(1-X)\, p_{,j} g^{ij} + (1-X)\eta_p \left(g^{jk} v^i_{,k}\right)_{,j}$ (3.7)

Now adding (3.6) and (3.7) and using (3.4) then equation of motion for blood flow will be

$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j) v^i_{,j} = -p_{,j} g^{ij} + \eta_m \left(g^{jk} v^i_{,k}\right)_{,j}$ (3.8)

Where $\eta_m = X\eta_c + (1-X)\eta_p$ is the viscosity coefficient of blood as a mixture of two phases. As velocity of blood flow decreases, the viscosity of blood increases. Since the venules are remote from heart therefore velocity of blood decreases. The Herschel Bulkley law hold good on two phase blood flow through the venules and whose constitutive equation as follows.

$T' = \eta_m e^n + T_p$ $(T' > T_p)$ and $e = 0$ $(T' < T_p)$ where $T_p$ is yield stress.

When strain rate e=0 $(T' < T_p)$ a core region is formed which flow just like a plug. Let radius of plug is $r_p$ and the stress acting on the surface of plug will be $T_p$.

Equation of force acting on the plug,

P л$r_p^2 = T_p 2$л$r_p$ or, $r_p = 2\frac{r_p}{P}$

The constitutive equation for rest part of blood vessel is

$T' = \eta_m e^n + T_p$ or, $T' - T_p = \eta_m e^n = T_e$ where $T_e$ is effective stress whose generalized form will be $T^{ij} = -pg^{ij} + T_e^{ij}$ where $T_e^{ij} = \eta_m (e^{ij})^n$, $e^{ij} = g^{jk} v^i_k$

Equation of continuity - $\frac{1}{\sqrt{g}} (\sqrt{g} v^i)_{,i} = 0$ (3.9)

Equation of motion - $\rho_m \frac{\partial v^i}{\partial t} + \rho_m v^j v^i_{,j} = -T^{ij}_{e,j}$ (3.10)

Where all the symbols have their usual meaning

**Newton Raphson Method:** The General Newton Raphson Method Formula is

$x_{n+1} = x_n - \frac{f(x_n)}{f'(x_n)}$ (3.11)

The above formula is repeated until a sufficiently precise value is obtained.

**4. Solution**

Let $x^1$ = r, $x^2$ = Θ and $x^3$ = z be cylindrical co-ordinates and square length of small element $ds^2 = dr^2 + r^2 d\Theta^2 + dz^2$
Christoffel's symbols of first and second kind are given below.

[i j, k] = $\frac{1}{2}\left[\frac{\partial g_{jk}}{\partial x^i} + \frac{\partial g_{ik}}{\partial x^j} - \frac{\partial g_{ij}}{\partial x^k}\right]$ and $\begin{Bmatrix} k \\ ij \end{Bmatrix} = g^{k\alpha}[ij, \alpha]$

$[g_{ij}]$ be matrix of metric tensor and $[g^{ij}]$ be matrix of conjugate matrix tensor where

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

Metric elements $g_{rr}$ = 1, $g_{\theta\theta}$ =$r^2$, $g_{zz}$ = 1

Or $g_{11}$ = 1, $g_{22}$ =$r^2$, $g_{33}$ = 1

Christoffel's symbols of second kind for cylindrical co-ordinates
Except of these all are zero.

$$\begin{Bmatrix} 1 \\ 22 \end{Bmatrix} = -r \, , \begin{Bmatrix} 2 \\ 21 \end{Bmatrix} = \begin{Bmatrix} 2 \\ 12 \end{Bmatrix} = \begin{Bmatrix} 1 \\ r \end{Bmatrix}$$

**Physical components**
Since $\sqrt{g_{11}}\, v^1 = v_r$ or, $v_r = v^1$

$\sqrt{g_{22}}\, v^2 = v_\theta$ or, $v_\theta = rv^2$

and $\sqrt{g_{33}} v^3 = v_z \, or, v_z = v^3$

Matrix of physical components of shearing stress tensor

$T'^{ij} = \eta_m \, (e^{ij})^n = \eta_m \, (g^{jk} v^i_{,k} + g^{ik} v^j_{,k})^n$ (4.1)

$$T'^{ij} = \begin{bmatrix} 0 & 0 & \eta_m \left(\frac{dv}{dr}\right)^n \\ 0 & 0 & 0 \\ \eta_m \left(\frac{dv}{dr}\right)^n & 0 & 0 \end{bmatrix}$$

The covariant derivative of $T'^{ij}$

$$T'^{ij}_{,j} = \frac{1}{\sqrt{g}} \frac{\partial}{\partial} (\sqrt{g} T^{ij} + \begin{Bmatrix} i \\ j\ k \end{Bmatrix} \quad (4.2)$$

According the above facts, the governing tensorial equation can be transformed into cylindrical form which is as follow

The equation of continuity $\frac{\partial v}{\partial z} = 0$ (4.3)

The equation of motion

r – Component - $\frac{\partial p}{\partial r} = 0$ (4.4)

Θ - Component 0 = 0 (4.5)

z- Component

$$-\frac{\partial p}{\partial z} + \frac{\eta_m}{r} \frac{\partial}{\partial r} \left[ r \left( \frac{\partial v_z}{\partial z} \right)^n \right] \quad (4.6)$$

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

$\frac{\partial p}{\partial t} = \frac{\partial v_r}{\partial t} = \frac{\partial v_\Theta}{\partial t} = \frac{\partial v_z}{\partial t} = 0$

From (4.3) $v_z = v(r)$ (4.7)

since $v_z$ does not depend upon θ

From equation (4.4) p = p (z) (4.8)

Because p does not depend upon, Θ using equation (4.7) & (4.8) in (4.6) then

$- \frac{dp}{dz} + \frac{\eta_m}{r} \frac{d}{dr} [r (\frac{dv}{dr})^n] = 0$ (4.9)

The pressure gradient $- \frac{dp}{dz}$ = P of blood flow in venules remote from heart can be supposed to be constant, therefore equation (4.9) takes the following form

$$\frac{d}{dr} [r (\frac{dv}{dr})^n = - \frac{Pr}{\eta_m} \quad (4.10)$$

On integrating (4.10), we get

$$r (\frac{dv}{dr})^n = - \frac{pr^2}{2\eta_m} + C \quad (4.11)$$

Since the velocity of blood flow on the axis of the cylindrical venules is maximum and constant so we apply the boundary condition at r=0, $v = v_0$ (constant) on equation (4.11) we get C=0 then equation (4.11) takes the following form

$$r (\frac{dv}{dr})^n = - \frac{p\, r^2}{2\eta_m} \text{ or, } - \frac{dv}{dr} = (\frac{pr}{2\eta_m})^{1/n} \quad (4.12)$$

Replace r from r - $r_p$ for non plug region

$$\frac{dv}{dr} = - (\frac{P}{2\eta_m})^{\frac{1}{n}} ( r - r_p)^{\frac{1}{n}} \quad (4.13)$$

Integrating equation (4.13), we get

$$v = - \left(\frac{p}{2\eta_m}\right)^{1/n} \frac{(r-r_p)^{\frac{1}{n}+1}}{\frac{1}{n}+1} + B \quad (4.14)$$

Apply no slip boundary condition $v = 0$ at r = R in equation (4.14), we get

$$B = \left(\frac{p}{2\eta_m}\right)^{1/n} \frac{(R-r_p)^{\frac{1}{n}+1}}{\frac{1}{n}+1} \quad (4.15)$$

Using (4.15) in equation 4.14) then

$$v = (\frac{p}{2\eta_m})^{\frac{1}{n}} \frac{n}{n+1} [(R - r_p)^{\frac{1}{n}+1} - (r - r_p)^{\frac{1}{n}+1}] \quad (4.16)$$

This is velocity of blood flow in venules

Putting r = $r_p$ we get the velocity of plug flow as follows

$$v_p = \frac{n}{n+1} (\frac{P}{2\eta_m})^{1/n} (R - r_p)^{\frac{1}{n}+1} \quad (4.17)$$

Where value of $r_p$ taken from equation of motion

**5. Result**

The flow flux of two phased blood flow in venule

$$Q = \int_0^{r_p} 2\pi r v_p \, dr + \int_{r_p}^{R} 2\pi r v dr$$

$$= \int_0^{r_p} 2\pi r \frac{n}{n+1} \left(\frac{P}{3\eta_m}\right)^{\frac{1}{n}} (R - r_p)^{\frac{1}{n}+1} dr + \int_{r_p}^{R} 2\pi r \frac{n}{n+1} (\frac{P}{2\eta_m})^{\frac{1}{n}} [(R - r_p)^{\frac{1}{n}+1} - (r - r_p)^{\frac{1}{n}+1}] dr$$

(Using (4.16) & (4.17)

$$Q = \frac{2\pi n}{n+1} \left(\frac{P}{3\eta_m}\right)^{\frac{1}{n}} (R - r_p)^{\frac{1}{n}+1} \left[\frac{r^2}{2}\right]_0^{r_p} + \frac{2\pi n}{n+1} (\frac{P}{2\eta_m})^{1/n} \left[\frac{r^2}{2} (R - r_p)^{\frac{1}{n}+1} - \frac{r(r-r_p)^{1/n}}{\frac{1}{n}+2} + \frac{(r-r_p)^{\frac{1}{n}+1}}{(\frac{1}{n}+2))(\frac{1}{n}+3)}\right]_{r_p}^{R}$$

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

**Observations:** Hemoglobin and blood pressure is taken from Anurag Nursing Home Banda by Dr. Anurag Srivstava
Patient Name – Gyanchandra, Age – 43 years / male Annual No.- 0645/2022
Diagnosis – Falsiparum Malaria

**Table 1:** Hematocrit and Cli. Blood Pressure Drop

| Sl. No. | Date | HB (Hemoglobin) gm/dl | BP (Blood Pressure) mmhg | Hematocrit (3×HB) | BP (In Pascal) | Blood Pressure Drop |
|---|---|---|---|---|---|---|
| 1 | 22.10.2022 | 11.7 | 135/92 | 35.1 | 17998.2/12265.44 | -3044.14 |
| 2 | 24.10.2022 | 11.4 | 142/94 | 34.2 | 18931.44/12532.08 | -3140.43 |
| 3 | 26.10.2022 | 10.7 | 140/90 | 32.1 | 18664.8/11998.8 | -3036.73 |
| 4 | 28.10.2022 | 10.1 | 148/96 | 30.3 | 19731.36/12798.72 | -3229.31 |
| 5 | 30.10.2022 | 10.4 | 138/90 | 31.2 | 18398.16/11998.8 | -3021.92 |

Blood Pressure Drop in $= \frac{2}{3}\left[\frac{\frac{S+D}{2}+D}{3}\right] - \left[\frac{\frac{S+D}{2}+D}{3}\right]$

Q = 1000 ml/min = 0.01666 litre/sec = 0.0000167 $m^3$/sec

R = 1, $r_p$ = 1/3

According to Gustafason Daniel R, (1980) [11]

$\eta_p$ = 0.0015 (Pascal-Sec)

According to Glenn Elert (2010)

$\eta_m$ = 0.035 (Pascal –Sec)

Average length of terminal hepatic venule = 0.15cm = $15\times10^{-4}$ meter

Average systolic pressure S= 18744.792 and average diastolic pressure D= 12318.768

H= 32.58, Blood pressure drop = 3094.50533 (Pascal Sec.)

Since $\eta_m = \eta_c$ X + $\eta_p$ (1-X) where X = $\frac{H}{100}$

Substituting the values of $\eta_m$, $\eta_p$, and H in above relation we get $\eta_c$ = 0.104323818 again from above relation

$\eta_m$ = 0.001028238187H + 0.0015

Substituting the value of $r_p$ and R in equation (28) we get

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

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

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

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

Since P = - $\frac{dp}{dz}$ $or$, dp = -Pdz

$$p_f - p_i = \left(\frac{27Q}{2\pi A}\right)^n 3\eta_m\,(z_f - z_i) \qquad (5.2)$$

Where $p_f - p_i$ pressure drop and $z_f - z_i$ = length of hepatic venule
Substituting the values of Q, $p_f - p_i$, $and$ $\eta_m$ in above equation and solve by numerical method we get n = -1.4445, and again

$p_f - p_i = 3\eta_m\ (z - z_i)\ (\frac{27Q}{2\pi A})^n$

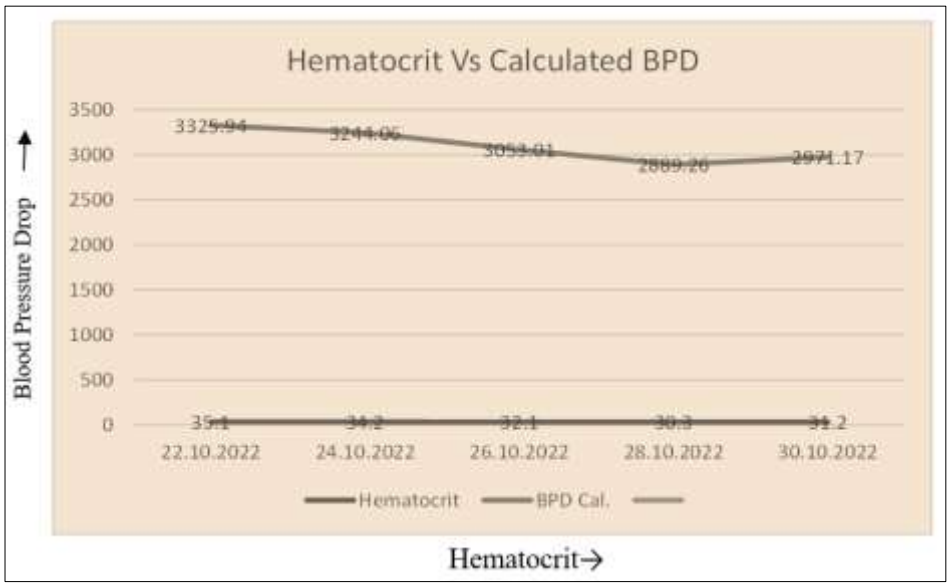
Substituting the value of $3\eta_m$ , Q and n we get

$p_f - p_i$ = 90.975056 H + 132.714954 (5.3)

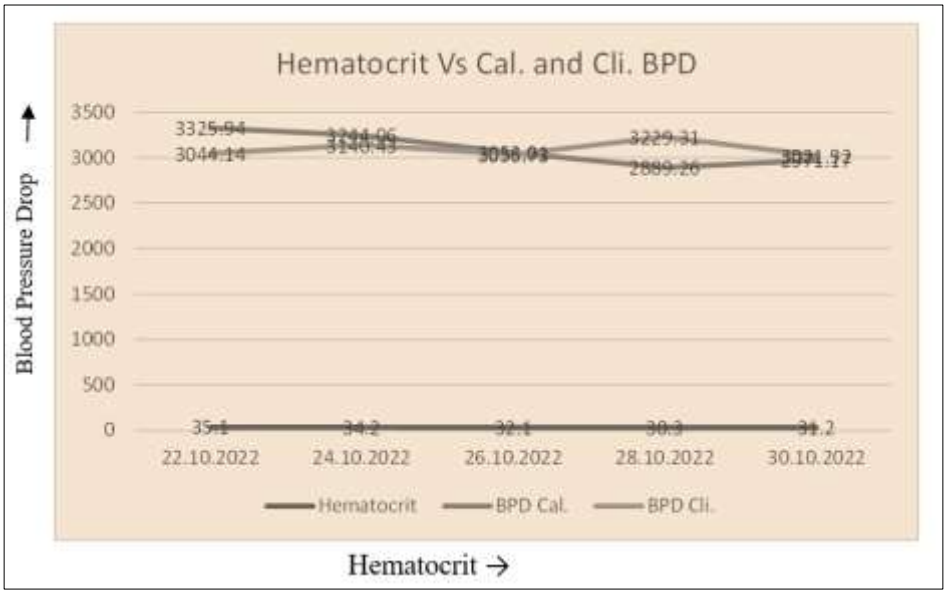
This is relation between hematocrit and blood pressure drop

**Table 2:** Hematocrit and Modulated Blood Pressure Drop

| Date | 22.10.2022 | 24.10.2022 | 26.10.2022 | 28.10.2022 | 30.10.2022 |
|---|---|---|---|---|---|
| Hematocrit | 35.1 | 34.2 | 32.1 | 30.3 | 31.2 |
| Blood pressure drop | 3325.94 | 3244.06 | 3053.01 | 2889.26 | 2971.17 |




**Graph 1:** Hematocrit Vs Modulated Blood Pressure Drop




**Graph 2:** Hematocrit Vs (Modulated and Cli.) Blood Pressure drop

## 6. Observation of graph

The graph-1 shows that fluctuations of blood pressure drop with respect to hematocrit of five different dates. We observed minimum blood pressure drop 2889.26 on dated 28/10/2022 and maximum value obtains 3325.94 on dated 22/10/2022. At the hematocrit value from 35.1 to 30.3 via 34.2 and 32.1 the blood pressure drop straightly decreases on dated from 22/10/2022 to 28/10/2022 via 24/10/2022 and 26/10/2022 and hematocrit value from 30.3 to 31.2 the blood pressure drop straightly increases on dated from 28/10/2022 to 30/10/2022. The graph-2 shows that the comparative study of two graph (i) Graph between hematocrit Vs clinical blood pressure drop (ii) Graph between hematocrit Vs mathematically modulated blood pressure drop.

## 7. Conclusion

In graph-1 the slope of straight line is the absolute value and when hematocrit increases the blood pressure drop also increases and when hematocrit decreases the blood pressure drop also decreases. When the trend of straight line decreasing sense then medicine dose slowly increases, when steepness of curve low then we can give high dose of medicine and when trend of straight line increases sense then we suggest normal dose of medicine. A comparative study of both graphs shows that nearly have the same character.

## 8. Acknowledgement

I give my sincere thanks to Dr. P N Yadav physician of CHC Baberu Banda (U.P.).

## 9. References

1. Guyton C. Medical Physiology. WBS; c1981. p. 207.
2. Alberts, Bruce. Table 22-1 Blood cells, Molecular Biology of the cell. NCBI Bookshelf; c2012.
3. Bergel DH, Schultz DL. Arterial elasticity and fluid dynamics; Progress in Biophysics and Molecular Biology. 1971;22:1-36.
4. Bhunchet E, Wake K. The portal lobule in rat liver fibrosis: A re-evaluation of the liver unit. Hepatology. 1998;27(2):481-487.
5. Dondorp AM, Kager PA, Vreeken J, White NJ. Abnormal blood flow and red blood cell deformability in severe malaria. Parasitol Today. 2000;16(6):228-232.
6. Fawcett Malarkey. New insights into functional aspects of liver morphology. Toxicologic pathology. 2005;33(1):27-34.
7. Fedosov DA, Peltomaki M, Gompper G. Deformation and dynamics of red blood cells in flow through cylindrical microchannels. Soft matter. 2014;10(24):4258-4267.
8. Fung Y. Biomechanics: Circulation. Springer Science & Business Media; c1997.
9. Elert G. Viscosity-The physics hypertextbook. The Physics Hypertextbook; c2010.
10. Goldstein S. Editor. Modern development in fluid dynamics, Oxford; c1938.
11. Gustafson Deniel R. Physics: Health and Human body, Wadsworth; c1980.
12. Haustring D. editor. Liver regeneration. Walter de Gruyter; c2011.
13. Kapur JN, Gupta RC. Power law fluid flow in the inlet length of a circular pipe; The Mathematics, Seminar. 1963;3:55-67.
14. Mackinnon MJW, Mwangi RW, Snow K, Marsh, Williams TN. Heritability of Malaria in Africa Plos Medicine. 2005;2(12):e340.
15. Mishra RS. Tensors and Riemannian geometry, Pothishala Pvt. Ltd. Allahabad; c1990.
16. Prakash O, Upadhyay V, Agrawal AK, Pandey PN. A mathematical model on two phase hepatic systolic blood flow in hepatic arterioles with special reference to Hepatitis B. International Journal of Applied Research. 2015;1(8):318-323.
17. Ruch TC, Patton HD. (Eds); Physiology and Biophysics Vols (II) and (III); W.B.S; c1973.
18. Sherman IW, Sherman VG. Biology: A Human Approach Oxford University press, New York, Oxford; c1989.
19. Michael S. Density of Blood The physics fact book; c2004.
20. Bhupendra S, Upadhyay V, Agrawal AK, Shrivastav MK, Pandey PN. A non-Newtonion model of two phase hepatic blood flow with special reference to liver abscess. The International Journal of Engineering and Science. 2013;2(10):87-93.
21. Singh JP, Agrawal AK, Upadhyaya V, Kumar A. Mathematical model and analysis of two phase Hepatic blood flow through arterioles with special reference to Hepatitis A. American Journal of Modeling and Optimization. 2015;3(1):22-25.
22. Singh P, Upadhyay KS. A new approach for the shock propagation in the two phase system; NAT. National Academy Science. 1986;8(2):112-118.
23. Upadhyay V. Some phenomena in two phase blood flow. Ph.D. Thesis, Central University, Allahabad; c2000. p. 123.
24. Vollmar B, Menger MD. The hepatic microcirculation, mechanistic contributions and therapeutic target in liver injury and repair. Physiological Reviews. 2009;89(4):1269-1339.
25. Lavazec C. Molecular mechanisms of deformability of Plasmodium-infected erythrocytes. Current Opinion in Microbiology. 2017;40:138-144.
26. Fedosov DA, Lei H, Caswell B, Suresh S, Karniadakis GE. Multiscale modeling of red blood cell mechanics and blood flow in malaria. PloS Computational Biology. 2011;7(12):1-13. DOI: 10.1371/journal.pcbi.1002270
27. Oda M, Yokomori H, Han JY. Regulatory mechanisms of hepatic microcirculation. Clinical Hemorheology and Microcirculation. 2003;29(3-4):167-182.
28. Young B, Lowe JS, Stevens A, Heath j W. Wheater's Functional Histology: A Text and Color Atlas. 4th ed. Oxford, UK: Churchill Livingstone; c2006. p. 288-301.
29. Vuk I, Rajic Z, Zorc B. Malaria and antimalarial drugs. Farm Glas. 2008;64:51-60.
30. Soulard V, Bosson-Vanga H, Lorthiois A, Roucher C, Franetich JF, Zanghi G, *et al*. Plasmodium falciparum full life cycle and plasmodium ovale liver stages in humanized mice. Nature Communications. 2015;6(1):7690.
31. Josling GA, Llinas M. Sexual development in Plasmodium parasites; knowing when it's time to commit. Nat. Rev. Genet. 2015;13(9):573-587.