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Extention of two phase power law model in three phase blood flow in human renal artery in case of dengue

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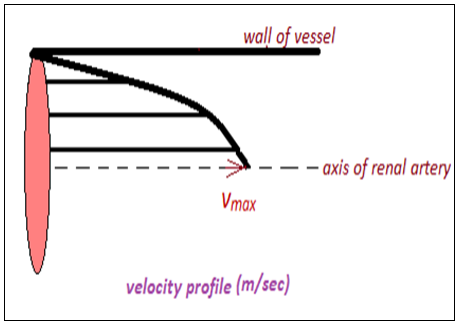
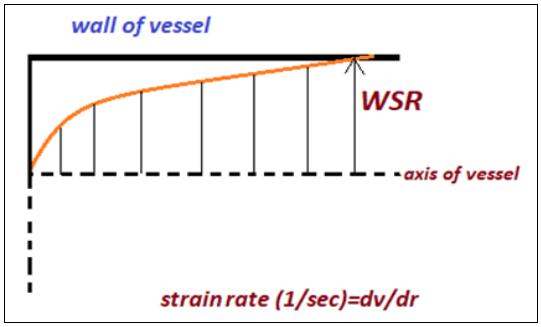
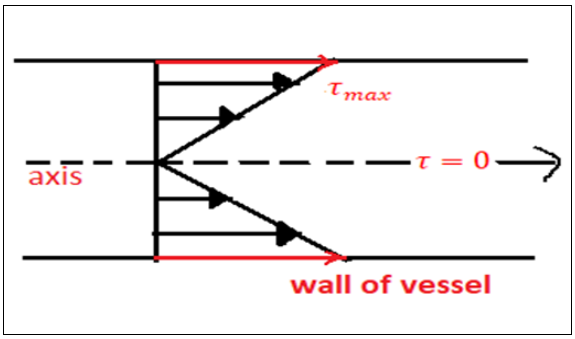
Abstract
In this paper, two-phase power law model blood flow model has been extended in three phase for human renal subsystem in case of dengue disease. Here, we have seen it from the perspective of a decline in hematocrit and blood pressure. Hematocrit and platelets are more affected by this disease than any other blood parameter. we have calculated the power index using a numerical methods for renal artery and. Finally, we have discovered a linear relationship between the decline in blood pressure and hematocrit datewise. max MBPD is 2609 pascal and min MBPD is 2333.11 pascal. v_{max} of blood is 101.91 cm/sec at the axis of renal artery and vanished in wall of artery.

Keywords: Phases, Hematocrit, Renal artery, Modulated blood pressure drop.

1. Introduction
Blood, one of the body's most complex fluids and a necessary fluid, has quite different properties from traditional, one-phase fluids. Because blood contains both a liquid (plasma) and a dispersed cellular component (primarily red blood cells, white blood cells, and platelets), it is a two- or multi-phase fluid. Because of the numerous non-Newtonian behaviors and spatial heterogeneities brought about by their multi-phase nature, blood flow dynamics are particularly challenging to predict. Blood as a multi-phase fluid must be understood in order to create accurate mathematical and numerical models of blood flow through the vascular system, which are necessary for microcirculation, pathological conditions, and medical device design. Approximately 55% of the volume of blood is made up of the straw-colored liquid known as plasma.
Blood possesses non-Newtonian rheological properties including viscoelasticity and shear-thinning viscosity (the viscosity decreases as the shear rate increases) because it includes both plasma and cells.
Two-phase fluid dynamics must be used instead of more conventional Newtonian fluid models due to the intricacy of these behaviours. Two-phase modelling, which sees blood flow as made up of two interpenetrating continua the fluid phase, which includes plasma, and the particulate phase, which includes cells usually focusses on red blood cells because of their volumetric dominance and dynamic activity. Two-phase flow models can mathematically describe a single fluid or a mixture of fluids. The homogeneous models assume that the two phases move with the same velocity field and include the interaction between them into averaged characteristics like effective viscosity and density.







But in A three-phase blood flow model is needed for close investigation behavior of blood, especially in microcirculation, where different components like plasma, red blood cells (RBCs), and white blood cells (WBCs) behave differently and interact with each other.

2. Structure and Function of Renal Artery

The mean peak systolic velocity (PSV) was highest on the left side in males measuring 65.75 ± 28.41cm/sec and 60.7 ± 24.20cm/sec on the right [6].

The main renal arteries are approximately 4 to 6 cm long with a 5 to 6 mm diameter. The right renal artery, which is longer than the left, arises from the anterolateral aorta and runs in an inferior course posterior to the inferior vena cava (IVC) to reach the right kidney [7]. In arteries, capillaries and veins, the endothelium is exposed to various levels of shear stress ranging from 1 to 70 dyn/cm 2. In arteries, shear ranges between 10 and 70 dynes/cm2 while in veins it is ~1–6 dynes/cm 2. Overall in most arteries, shear stress is maintained between 10 and 20 dynes/cm2 (Natarajan et al., 2016). Peak Reynolds number in renal artery is 1145±140 (870-1320) [8].

Table 1: Shear stress in human blood vessels

Vessels	Shear stress dyne/cm^2
Arteries	10–60
Veins	1–10
Stenosis in arteries	>100
High Stenosis in arteries	>1,000
Ascending Aorta 12	12
Descending Aorta 5–8	5-8
Pulmonary Artery 5	5
Small vein 11	11
Large vein	5

Approximate normal values from studies reported by Samet and Lelkes, 1999; Waite and Fine, 2007)

3. Dengue and Kidney

From clinically undetectable forms to severe hemorrhage and shock that ultimately leads to death, dengue presents in a variety of ways with unexpected clinical development and consequences [1]. Serious clinical symptoms are linked to reinfection with a different serotype, most likely as a result of cross-reactive antibodies [2]. Fever, often between 39 and 40°C, is the initial sign of classic dengue. Other symptoms include headache, prostration, myalgia, arthralgia, retro-orbital discomfort, and itchy or non-maculopapular exanthema. There may also be diarrhea, vomiting, nausea, and anorexia. Similar to the classic type, severe dengue begins with symptoms including bleeding and/or cavity effusion, hemodynamic instability, and/or shock.

Thrombocytopenia (less than 100,000 platelets/mm3), hemoconcentration, and one or more of the following clinical signs of plasma extravasation pleural effusion, ascites, and a rise in the haematocrit exceeding 20% of the baseline value are linked to hemorrhagic manifestations. Hypovolemic shock may result from the selective loss of plasma into the serous cavities, including the pleural and peritoneal

cavities. The hemorrhagic symptoms, which include a positive tourniquet test, petechiae (limbs, face, and axillae), ecchymosis, epistaxis, gingival bleeding, uterine haemorrhage, and upper gastrointestinal bleeding, start three to seven days after the disease starts. Patients with severe dengue may experience severe stomach discomfort, pallor, clammy and chilly skin, anxiety, lethargy, breathing difficulties, and a rapid and weak pulse after a raised fever that lasts for two to seven days [1].

Acute kidney injury, acute tubular necrosis, haemolytic uremic syndrome, proteinuria, glomerulopathy, nephrotic syndrome, and elevated blood creatinine levels are among the several types of renal involvement that have been seen in dengue patients [3, 4]. An in situ immune-mediated mechanism triggered by viral antigens bound to glomerular structures, tissue damage caused by immune complexes composed of viral antigens and antiviral antibodies, damage caused by inflammatory mediators released in response to the glomerular or tubular cytopathic effects of the viral antigens, or a direct cytopathic effect of the viral protein on the glomerular and tubular cells are all possible causes of viral infection-induced renal injury [5].

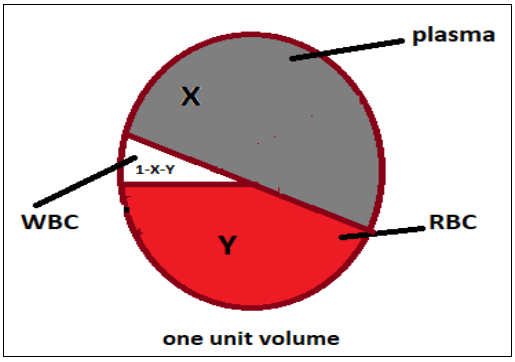
4. Real model

It is appropriate to assume Newtonian blood behaviour when the blood is passing via a bigger artery. When the blood vessel is tiny (radius less than 1 mm), it is invalid. Blood would not be anticipated to follow Newton's extremely basic, one parameter, linearised law of viscosity from the perspective of biofluid mechanics. Only higher order constitutive equations, such the power-law paradigm, can adequately represent the non-Newtonian properties of blood (Enderle et al).

4.1 Parametrization

The blood's velocity $v^k = v^k(X^i, t) k = 1,2,3$ and any two thermodynamic quantities related to it, such as pressure, $P = P(X^i, t)$ and density, $\rho = \rho(X^i, t)$,were distributed according to functions that affected the mathematical description of the state of a moving blood. All thermodynamic quantities, together with the equation of state, are determined by the values of any two of them, as is often known. Thus, we may fully ascertain the condition of flowing blood if we have five variables: the density ρ, the pressure P, and the three components of velocity v^k.

The coordinates $X^i, i = 1,2,3$, and the time t are functions of all these values. It stressed that the blood's velocity at a given position X^i in space and at a given time t was represented by the expression $v^k(X^i, t)$.



In our three phases of blood assumption
Let one unit volume of whole blood and
X= volume fraction of plasma
Y= volume fraction of RBC
Z=1-*X*-*Y* =volume fraction of WBC the mass ratio of RBC to plasma is m_1 and WBC to plasma is m_2.

$$m_1 = \frac{Y\rho_C}{X\rho_P} \quad , m_2 = \frac{Z\rho_w}{X\rho_P}$$

Where ρ_C ,ρ_P ,ρ_W are the densities of RBC, plasma, WBC.
We define density of blood mixture ρ_m as follows

$$\frac{1+m_1+m_2}{\rho_m} = \frac{m_1}{\rho_C} + \frac{1}{\rho_P} + \frac{m_2}{\rho_w}$$

And viscosity of blood mixture η_m as follows

$$\eta_m = Y\eta_c + X\eta_p + Z\eta_w$$

4.2 Boundary conditions

1. The velocity of blood flow on the axis of blood vessels at r = 0 will be maximum and finite, say v_0 = maximum velocity.
2. The velocity of blood flow on the wall of blood vessels at *r* = *R*, where, R is the radius of blood vessels, will be zero. This condition is well known as no slip condition.

4.3 Equation of Continuity

continuity equation for three phases

$$\frac{\partial(Y\rho_c)}{\partial t} + \left(Y\rho_c v^i\right)_{,i} = 0 \qquad [1]$$

$$\frac{\partial(X\rho_P)}{\partial t} + \left(X\rho_P v^i\right)_{,i} = 0 \qquad [2]$$

$$\frac{\partial((1-X-Y)\rho_w)}{\partial t} + \left((1-X-Y)\rho_w v^i\right)_{,i} = 0 \qquad [3]$$

Where, v^i is the common velocity of two phase blood cells and plasma. Again $\left(X\rho_c v^i\right)_{,i}$ is co-variant derivative of $(X\rho_c v^i)$ with respect to X^i.

Equation of motion for blood flow with the three phases
using the principle of force conservation (or momentum conservation) in hepatic arteries and assuming that the consistency coefficient (or viscosity coefficient) of RBC cells is η_c.

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

Similarly, taking the viscosity coefficient of plasma to be the equation of motion for plasma will be as follows-

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

For WBC

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

Then equation of motion for blood flow with the all three phases will be as follows-

$$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j)v^i{}_{,j} = -P_{,j}g^{ij} + \eta_m\left(g^{jk}v^i{}_{,k}\right)_{,j} \quad [4]$$

Whenever percentage of blood is reduces the blood has been supposed Newtonian but in case of increasing the hematocrit, the effective viscosity of blood flowing through arteries remote from the heart depends on the strain rate.
For this reason, the blood will flow as non-Newtonian fluid. When strain rate is in between 5 to 200 per second, the power law

$$\tau' = \eta_m e^n$$

where $0.68 \leq n \leq 0.80$ Describes the flow of blood very well. The constitutive equation of blood is as follow
Blood's constitutive equation is as follows:

$$\tau^{ij} = -pg^{ij} + \eta_m(e^{ij})^n = -pg^{ij} + \tau'^{ij} \quad [5]$$

Where τ^{ij} is stress tensor and τ'^{ij} is shearing stress tensor.

4.4 Mathematical formulation
The equation of continuity for power law flow will be as follows:

$$\frac{1}{\sqrt{g}}(\sqrt{g}v^i)_{,i} = 0 \quad [6]$$

Again the equation in tensorial form is as follows:

$$\rho_m \frac{\partial v^i}{\partial t} + \rho_m v^j v^i{}_{,j} = \tau^{ij}{}_{,j} \quad [7]$$

Since the blood vessels are cylindrical, the above governing equation have to transformed into cylindrical co-ordinates.

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

Matrix of corresponding metric tensor in cylindrical form is as follow:

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

So Matrix of conjugate metric tensor is

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

Where as Christoffel's symbols of 2nd kind are as follows:

$\begin{Bmatrix} 1 \\ 2 \quad 2 \end{Bmatrix} = -r \ , \ \begin{Bmatrix} 2 \\ 2 \quad 1 \end{Bmatrix} = \begin{Bmatrix} 2 \\ 1 \quad 2 \end{Bmatrix} = \frac{1}{r}$ except of these all are zero.

Contravarient and physical components of velocity of blood flow will be related as

$$\sqrt{g_{11}}v^1 = v_r => \ v_r = v^1$$

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

$$\sqrt{g_{22}}v^3 = v_z => \ v_z = \ v^3$$

Further the physical component of $-p_{,j}\, g^{ij} \ are - \sqrt{g_{ii}} p_{,j}\, g^{ij}$

The matrix of physical component of shearing stress – tensor

$\tau^{,ij} = \eta_m (e^{ij})^n = \eta_m (g^{ik} v^i_{,k} + g^{jk} v^j_{,k})^n$ [8]

Will be as follows

$$\begin{bmatrix} 0 & 0 & \eta_m (dv/dz)^n \\ 0 & 0 & 0 \\ \eta_m (dv/dr)^n & 0 & 0 \end{bmatrix}$$

The covariant derivative of $\tau^{,ij}$ is

$$\tau^{,ij}_{,j} = \frac{1}{\sqrt{g}} \frac{\partial}{\partial x^j} \left(\sqrt{g} \tau^{,ij}\right) + \begin{Bmatrix} i \\ j \quad k \end{Bmatrix} \tau^{,kj} \qquad [9]$$

Keeping in view the above facts the governing tensorial equation can be transformed into cylindrical form which are as follows :

The Equation of continuity

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

The Equation of motion

r-Component

$$-\frac{\partial p}{\partial r} = 0$$

θ-Component

$0 = 0$

Z-Component

$$0 = -\frac{\partial p}{\partial z} + \frac{\eta_m}{r} \frac{\partial}{\partial r} \left(r \left(\frac{dv}{dr} \right)^n \right)$$

These are the r, θ , z components respectively

Further the fact has been considered that axial flow in artery is symmetric, so that $v_\theta = 0 \ and \ v_r, \ v_z \ and \ p$ do not depend upon θ. Also the blood flows steadily, i.e.

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

On integrating equation, we get $v_z = v(r)$ because v does not depend upon θ
The integration of equation of motion, we get $p = p(z)$ since p does not depend upon θ
Now, with the help of equation, the equation of motion converts in the following form:

$$0 = -\frac{dp}{dz} + \frac{\eta_m}{r}\frac{d}{dr}\left(r\left(\frac{dv}{dr}\right)^n\right) \quad [10]$$

The pressure gradient $-\left({}^{dp}/_{dz}\right) = P$ of blood flow in the arteries remote from liver can be supposed to be constant and hence the equation takes the following form:

$$\frac{d}{dr}\left(r\left(\frac{dv}{dr}\right)^n\right) = -\frac{Pr}{\eta_m}$$

On integrating equation (10), we get

$$r\left(\frac{dv}{dr}\right)^n = -\frac{Pr^2}{2\eta_m} + A \quad [11]$$

We know that the velocity of blood flow on the axis of the cylindrical arteries is maximum and constant. So that the apply the boundary condition at r=0, v = V_0 (contant), on equation (11) to get the arbitrary constant A = 0 Hence the equation (11) takes the following form

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

$$-\frac{dv}{dr} = \left(\frac{Pr}{2\eta_m}\right)^{1/n} \quad [12]$$

Integrating equation (4.4.2) once again, we get

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

To determine the arbitrary constant B, we apply the no –slip condition n the inner wall of the arteries : at $r = R$, $V = 0$, where R = radius of vessel, on equation (13) so as to get

$$B = \left(\frac{P}{2\eta_m}\right)^{1/n} \frac{nR^{\frac{1}{n}+1}}{n+1}$$

Hence the equation takes the following form:

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

Which determines the velocity of blood flow in the arteries remote from the liver where P is gradient of blood pressure and η_m is the viscosity of blood mixture.
Shear stress

$$\tau = \left(\frac{Q(1+3n)}{\pi n}\right)^n \frac{r\eta_m}{R^{3n+1}}$$

Strain rate $\frac{dv}{dr} = \left(\frac{\Delta Pr}{2\Delta z\,\eta_m}\right)^{1/n}$

The total flow- flux of blood through the transverse section of the arteries is

$$Q = \int_0^R v.2\pi r\, dr = \int_0^R \left(\frac{P}{2\eta_m}\right)^{1/n} . \frac{1}{n+1}\left(R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right) 2\pi r\, dr$$

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

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

$$Q = \left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} . \frac{\pi n R^{\frac{1}{n}+3}}{(3n+1)} \quad , \quad where\ P = -\frac{dp}{dz}$$

$$Q = \left[\frac{P_i - P_f}{2\eta_m(z_i - z_f)}\right]^{\frac{1}{n}} . \frac{\pi n R^{\frac{1}{n}+3}}{(3n+1)}$$ [15]

5. Observation

Table 2: Pathological data of dengue patient for blood

S. N	Parameter	28/09/2024	29/09/2024	30/09/2024	02/10/2024	04/10/2024
1	WBC[$\times\ \mathbf{10^3/\mu L}$]	10.2	25.1	33.4	27.2	11.2
2	Hb[g/dL]	13.9	12.3	11.4	10.9	11.4
3	Platelets[$\times\ \mathbf{10^3/\mu L}$]	18	29	58	110	220
4	RBC[$\times\ \mathbf{10^6/\mu L}$]	4.2	4.0	3.6	3.2	3.9

Table 3: Clinical data blood pressure and hemoglobin

S.N	Date	B.P(mmHg)	Hb	Hct
1	28/09/2024	111.02/71.08	13.9	41.7
2	29/09/2024	108.10/72.60	12.3	36.9
3	30/09/2024	109.90/72.60	11.4	34.2
4	02/10/2024	112.50/75.90	10.9	32.7
5	04/10/2024	113.90/77.00	11.4	34.2

$$\boldsymbol{Y} = \frac{3.78 \times 10^6}{\mu L}$$

$$Z = \frac{21.48 \times 10^3}{\mu L}, palatelet = \frac{87 \times 10^3}{\mu L}$$

HERE RBC % = 35.94%, WBC % =0.5524%
equation of viscosity

$$\eta_m = Y\eta_c + (1 - Y - Z)\eta_p + Z\eta_w$$

$$0.0045 = 0.0070\left(\frac{35.94}{100}\right) + 0.0014(1 - 0.3594 - 0.005524) + \eta_w \times 0.005524$$

$$\eta_w = 0.19824\ Pascal\ Sec$$

Average Systolic Pressure =111.04 mm Hg
Average Diastolic Pressure = 74.132 mm Hg
H= Average hematocrit = 35.94

$$\Delta P = P_i - P_f = 2463.25\ pascal$$

According to Glenn Elert (2010)

η_m = viscosity of mixture = 0.0045 pascal sec
According to Gustafson, Daniel R. (1980)
η_p = Viscosity of plasma =0.0015 pascal sec
η_c= 0.0075 pascal sec[9]

Length of renal artery$(z_i - z_f)$ = 0.05 meter

We know that

$$\eta_m = Y \times \eta_c + (1 - Y - Z)\eta_p + Z\eta_w$$

$$where\ Y = \frac{H}{100}$$

$$\eta_m = \frac{H}{100} \times \eta_c + (1 - \frac{H}{100} - Z)\eta_p + Z\eta_w$$

$$\eta_m = 0.0070\frac{H}{100} + 0.0014\left(1 - \frac{H}{100} - 0.005524\right) + 0.19824 \times 0.005524$$

$$\eta_m = 5.6 \times 10^{-5}\, H + 0.00243104$$

$$Q = 660\frac{ml}{min} = 1.1 \times 10^{-5}\, m^3/sec$$

$$Q = \left[\frac{P_i - P_f}{2\eta_m(z_i - z_f)}\right]^{\frac{1}{n}} \cdot \frac{\pi n\, R^{\frac{1}{n}+3}}{(3n+1)}$$

$$1.1 \times 10^{-5} = (15053.194)^{\frac{1}{n}}\left(\frac{n}{3n+1}\right)(6.530218 \times 10^{-8})$$

After solving above equation by newton Raphson method
1.3434911262360782
1.486434780188817
1.4972113394441415
1.4968621093104466
1.4968759808626908
1.4968754331424243
1.496875454774358
1.4968754539200237
1.496875453953765
1.4968754539524323

Initial Guess taken: 1
Final result
n: 1.4968754539524323, abs_per_error: 8.903290619410403e-11
by above take absolute value of power index $n = 1.49687545395$

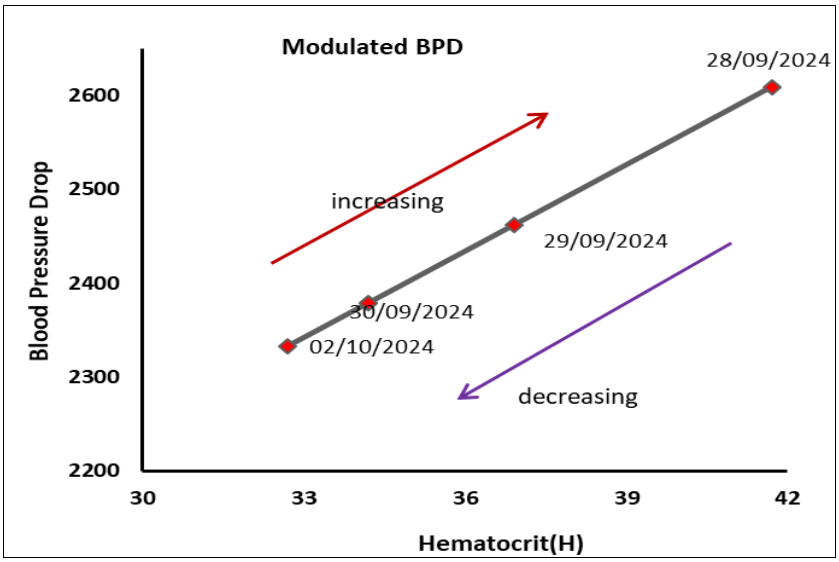
$$\triangle P = \left[\frac{(3n+1)Q}{\pi n R^3}\right]^n \left[\frac{2\eta_m \Delta z}{R}\right]$$

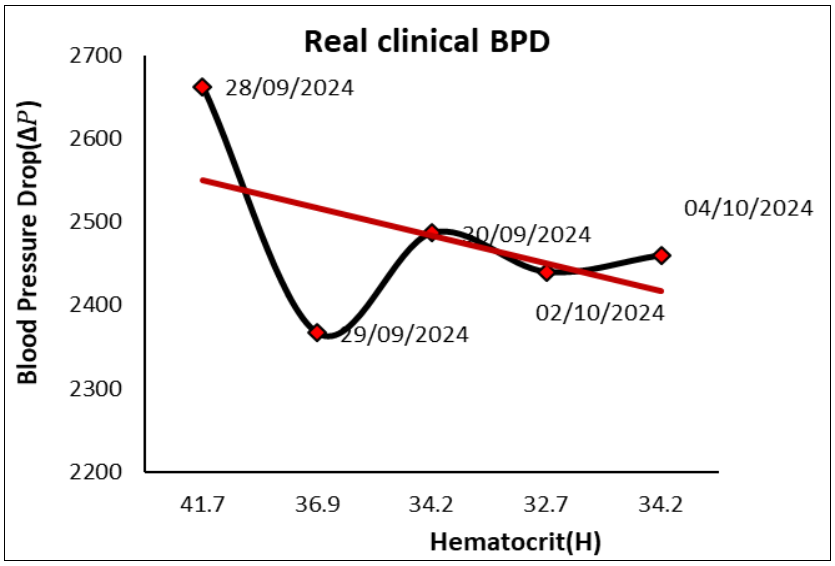
Substituting values in above equation, we have

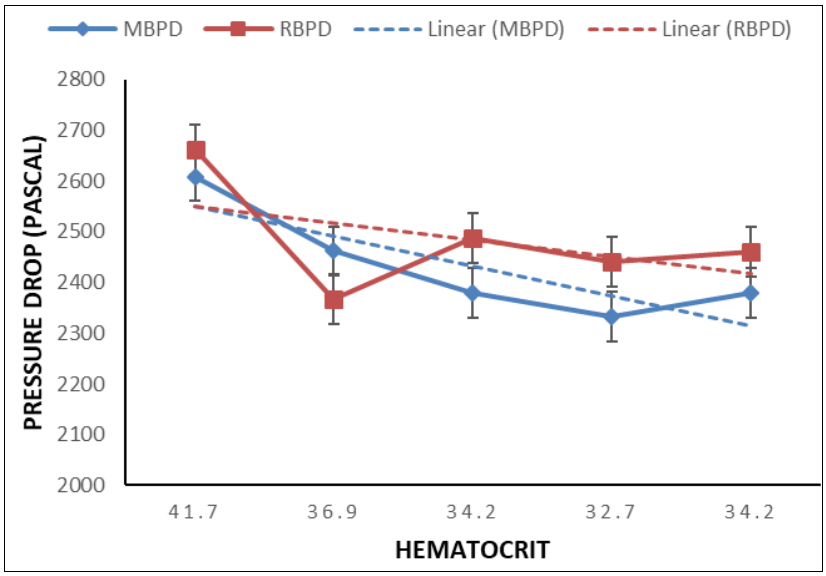
$$\Delta P = 30.6539\, H + 1330.7328$$

Table 4: Modulated Blood Pressure Drop Vs Hematocrit

S.N	DATE	HCT (H)	MBPD (ΔP_{modu}) in Pascal	Real Clinical pressure drop $\Delta P_{real\ cli}$ In Pascal
1	28/09/2024	41.7	2609	2662.60
2	29/09/2024	36.9	2461.86	2366.60
3	30/09/2024	34.2	2379.09	2486.60
4	02/10/2024	32.7	2333.11	2439.93
5	04/10/2024	34.2	2379.09	2459.93







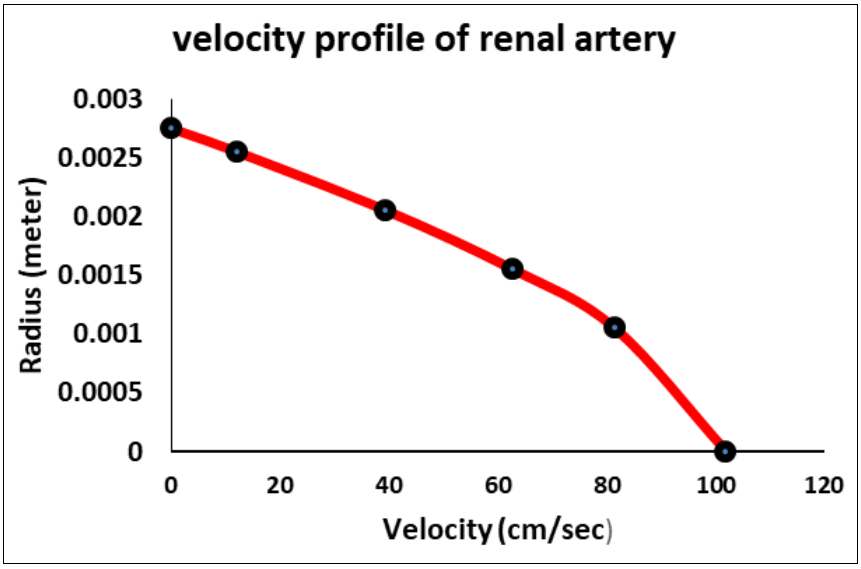
Velocity of blood in renal artery

$$v = \left(\frac{\Delta P}{2\eta_m \times \Delta z}\right)^{\frac{1}{n}} \frac{n}{n+1}\left[R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right]$$

Putting different values of radius of renal artery and calculate respective values of velocity

Table 5: Velocity Profile, Reynold No.

Radius(meter)	Velocity(cm/sec)	Reynold no R_e
0	101.91	713.73
0.00105	81.43	572.4
0.00155	62.71	443.15
0.00205	39.45	278.78
0.00255	12.04	85.08
0.00275	0	0



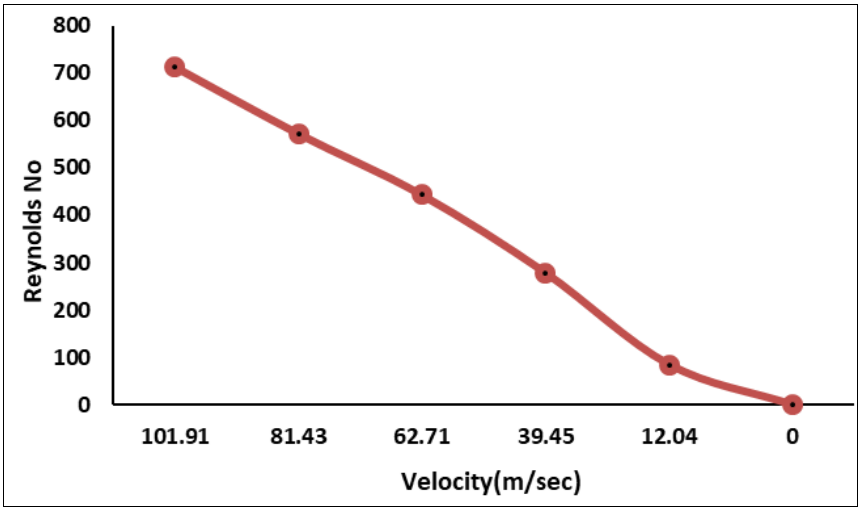
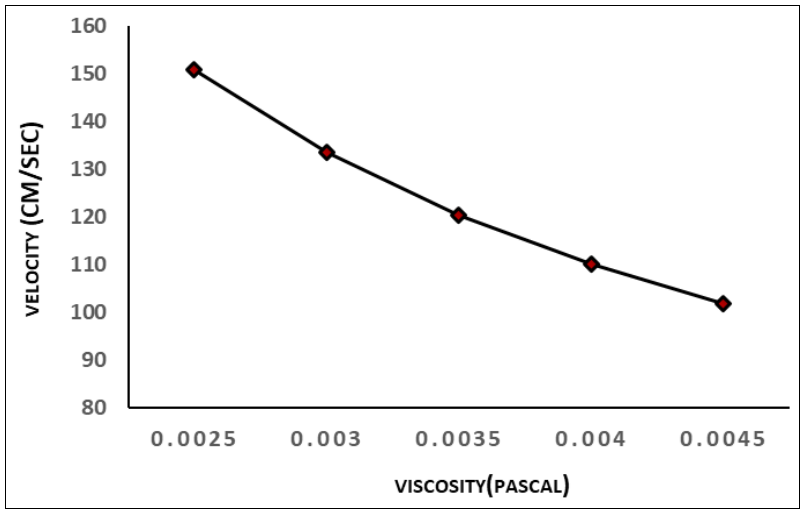


Table 6: Viscosity V/S Velocity and Flow Flux in Artery

Viscosity of blood mixture η_m pascal sec	Velocity at r=0, $v_{r=0}$ (cm/sec)	Flow flux Q m^3/sec
0.0025	150.85	1.62×10^{-5}
0.0030	133.55	1.44×10^{-5}
0.0035	120.48	1.30×10^{-5}
0.0040	110.20	1.19×10^{-5}
0.0045	101.86	1.09×10^{-5}



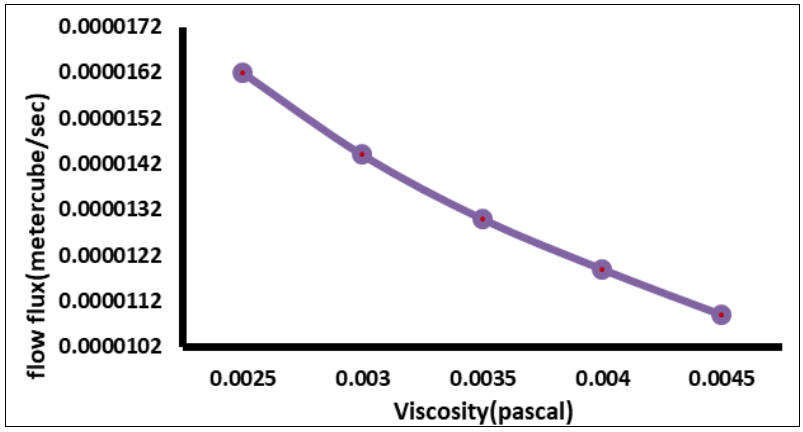
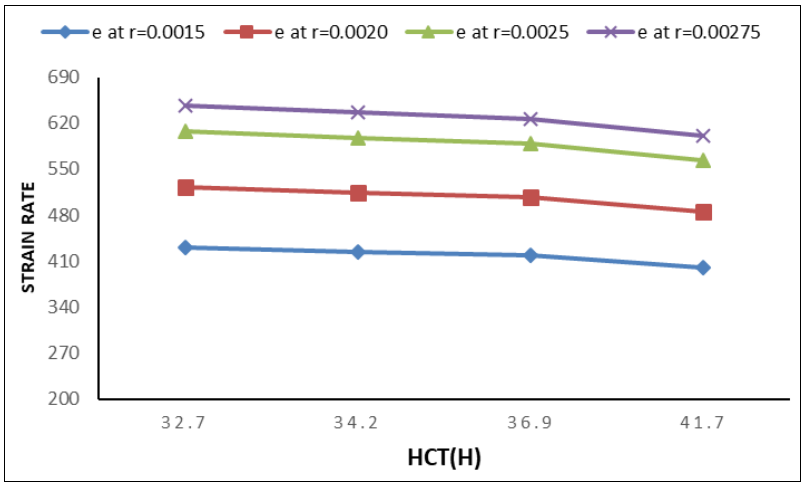


Table 7: Strain Rate $e(sec^{-1})$ and Hct(H)

Hct(H)	$e_{r=0.0015}$	$e_{r=0.0020}$	$e_{r=0.0025}$	$e_{r=0.00275}$
32.7	431.57	523.02	607.10	647.02
34.2	424.84	514.86	597.63	636.92
36.9	418.36	507.02	588.52	627.20
41.7	400.33	485.16	563.15	600.18



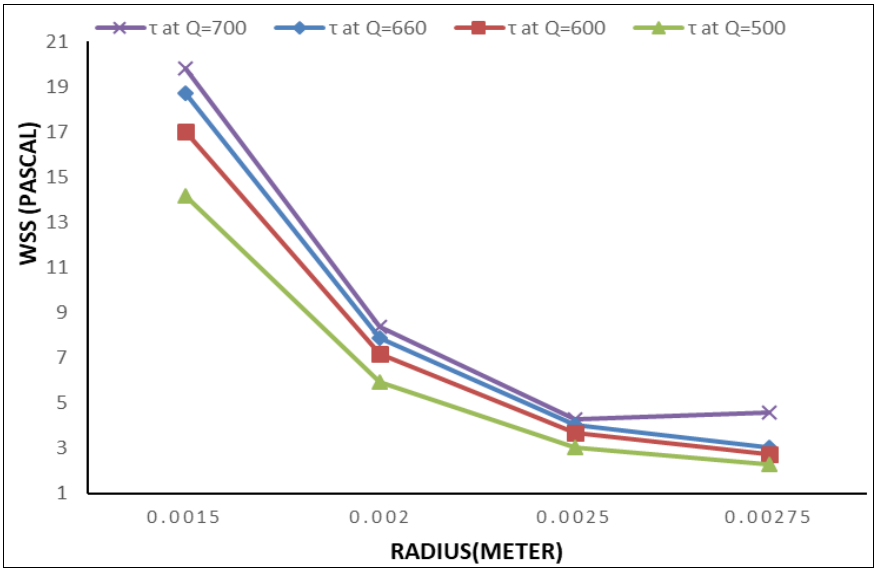
Wall Shear Stress in Newtonian

$$\tau = \frac{4\eta_m Q}{\pi R^3}$$

$\eta_m = 0.0045\ pascal\ sec$ and constant flow flux

Table 8: Radius v/s WSS

R	$\tau_{Q=500}$	$\tau_{Q=600}$	$\tau_{Q=660}$	$\tau_{Q=700}$
0.0015	14.14	16.98	18.68	19.81
0.002	5.9	7.16	7.88	8.36
0.0025	3.05	3.66	4.03	4.28
0.00275	2.29	2.75	3.03	4.59



Shear Stress Non Newtonian power law model

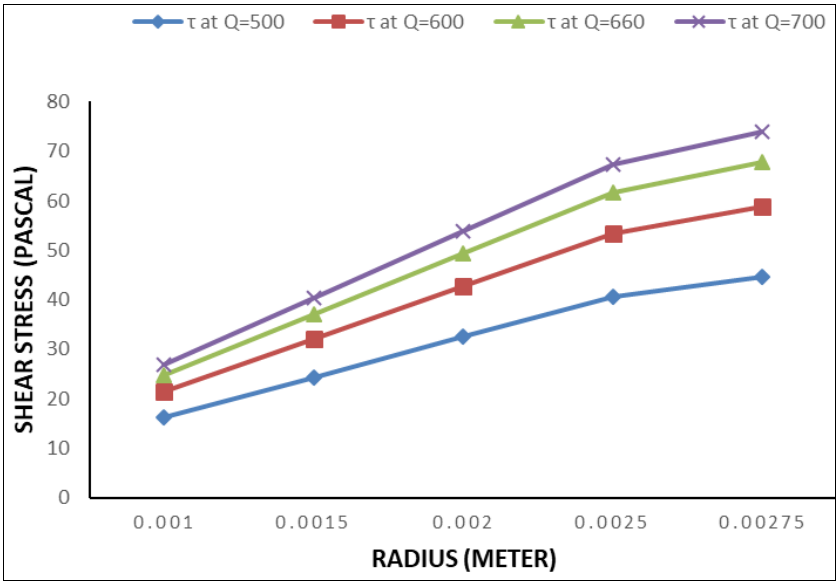
$$\tau = \left(\frac{Q(1+3n)}{\pi n}\right)^n \frac{r\eta_m}{R^{3n+1}}$$

Here

$$\frac{r}{R} \ll 1$$

Table 9: Shear Stress vs Radius

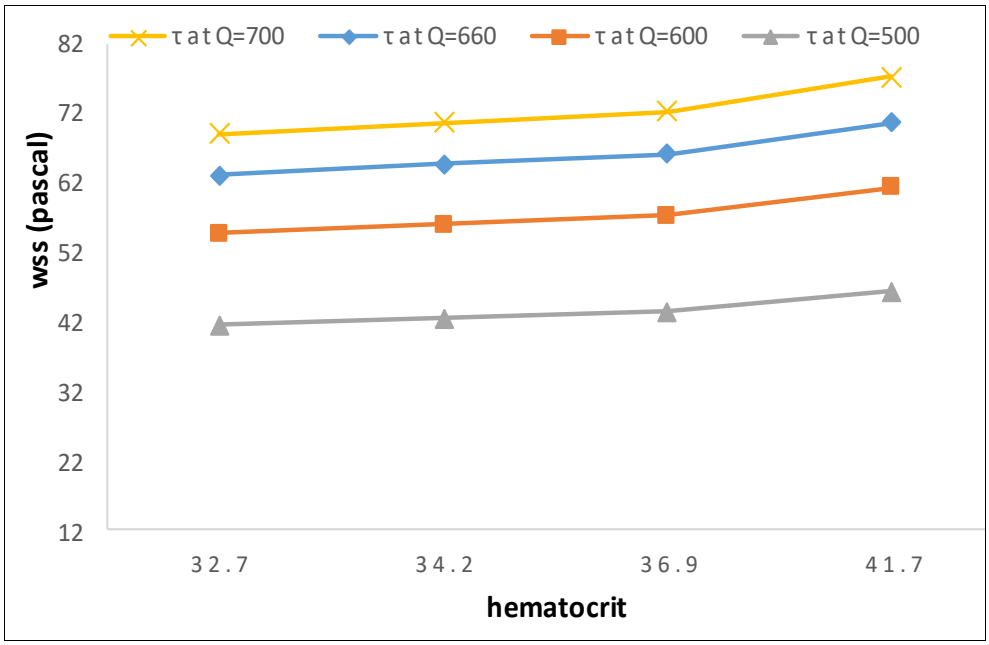
r	$\tau_{Q=500}$	$\tau_{Q=600}$	$\tau_{Q=660}$	$\tau_{Q=700}$
0.0010	16.24	21.35	24.63	26.90
0.0015	24.37	32.03	36.94	40.35
0.0020	32.49	42.71	49.26	53.80
0.0025	40.61	53.39	61.58	67.25
0.00275	44.67	58.73	67.73	73.97



WSS for fix radius $r = R = 0.00275 \ meter$

Table 10: WSS v/s HCT

Hct(H)	$\tau_{Q=500}$	$\tau_{Q=600}$	$\tau_{Q=660}$	$\tau_{Q=700}$
41.7	46.41	61.34	70.75	77.26
36.9	43.45	57.42	66.23	72.33
34.2	42.46	56.12	64.72	70.69
32.7	41.47	54.81	63.22	69.04



6. Conclusion

In this case $v_{max} = 101.91 \ cm/sec$ along the renal artery axis. At the vessel wall,. $v_{min} = 0$ and $R_e$is below 2000. Here, the WSS for the renal vessel is 3.03 pascal with a radius of 2.75 mm in the Newtonian case, and 67.73 pascal under the non-Newtonian power law model. WSS rises with an increase in flow flux from the center to the vessel wall. Both the case for mbpd and the actual clinical pressure decline are declining daily. Here, we see the same hematocrit behaviour in both drops. The trend lines are lowly sharp and pointing downward. Therefore, from a medical perspective, we will quickly raise the dosage of the medication, and the patient will recover quickly.

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