THE PHARMA INNOVATION

A mathematical model of two phase, non-Newtonian renal blood flows in venules, remote from the heart and proximate to the kidney with special reference to diabetes

V. Upadhyay*, Harish Chandra, P.N. Pandey

In our research work there have been formulated the renal blood flow along the venules in case of renal disease Diabetes. keeping in the view the nature of renal circulatory system in human body. Blood have been considered as two phased one of which is that of red blood cells and other is plasma. According to Fahreaus-Lindqvist effect the blood flow in two separated layers while passing through capillaries and then venules. We have collected a clinical data in case of Diabetes for hematocrit v/s blood pressure. The graphical presentation for particular parametric value is much closed to the clinical observation. 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 drop in case of renal disease Diabetes.

1. INTRODUCTION

1.1 Structure of the tissue (Kidney)

The kidney has bean shaped structure that serve the several essential regulatory roles in vertebrates. Each kidney has a convex and concave surfaces. The concave surface, the renal hilum, is the point at which the renal artery enter the organs and, renal veins & ureter leave.

The kidney is surrounded by tough fibrous tissue, renal capsule, which is itself surrounded by perinephric fat, renal fascia (Gerota) and paranephric fat. The anterior part of this tissue is peritoneum,

while the posterior (rear) border is the transversalis fascia [1-3]. The normal adult kidney is about 10-12 cm long, 5-7 cm wide and 2-3 cm thick and its weighs 125-170g Each kidney composed of parenchyama and collecting system. The parenchyama cosists of an outer cortex and inner medulla. The medulla is divided into an outer (towards the cortex) and inner medulla (toward pelvis). The collecting system includes the calyces, renal pelvis and the ureter. The major calyces unite to form the renal pelvis. The renal pelvis drain into ureter which connect the kidney to the bladder [4-6].

Corresponding Author's Contact information:
V. Upadhyay*
Dept. of Physical Sciences, M.G.C. Gramodaya Vishwavidyala, Chitrakoot, Satna, M.P, India
E-mail: drvirendra.upadhyay@gmail.com

V. Upadhyay*, Harish Chandra, P.N. Pandey

[7]
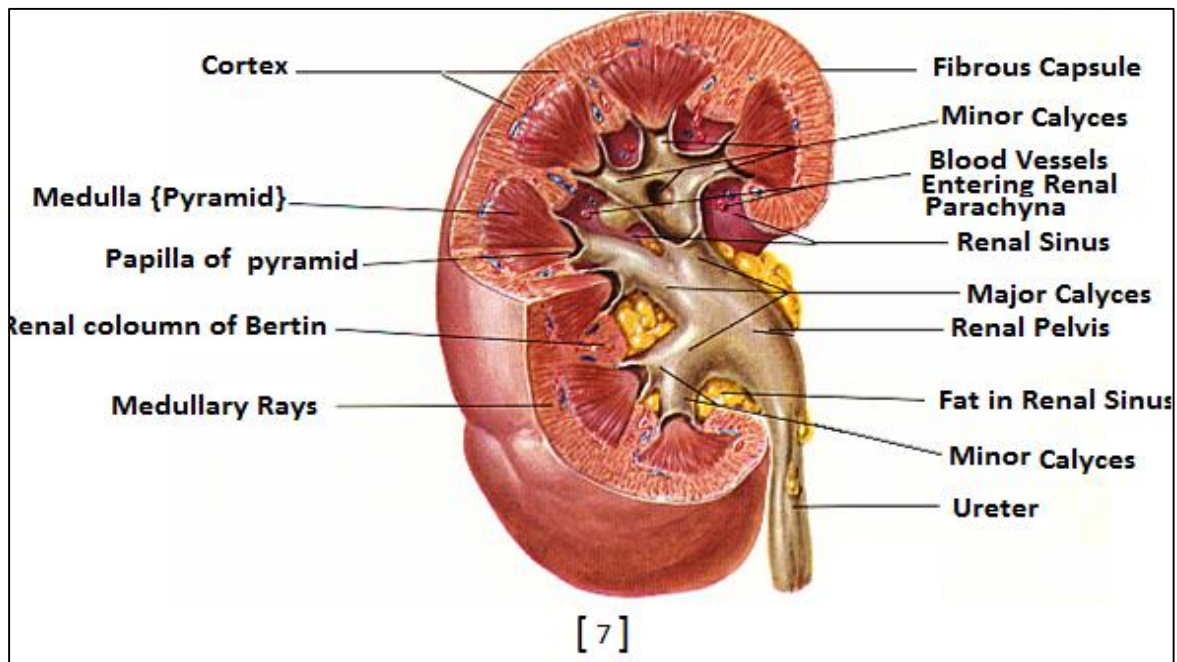
1.2 Nephron is the functional unit of the kidney: Each Human kidney contains about one million nephrons, each capable of forming urine. The kidney cannot regenerate new nephrons, therefore with renal injury, disease or normal aging, there is a gradual decrease in nephron numbers. After the the age of 40 years number of functioning nephrons usually decrease about 10 percent every 10 years thus the age of 80 many people have 40 percent fewer functioning nephrons than they did at age of 40.


Fig 1.: Kidney anatomy and enhanced view of nephron.

[8] [9]
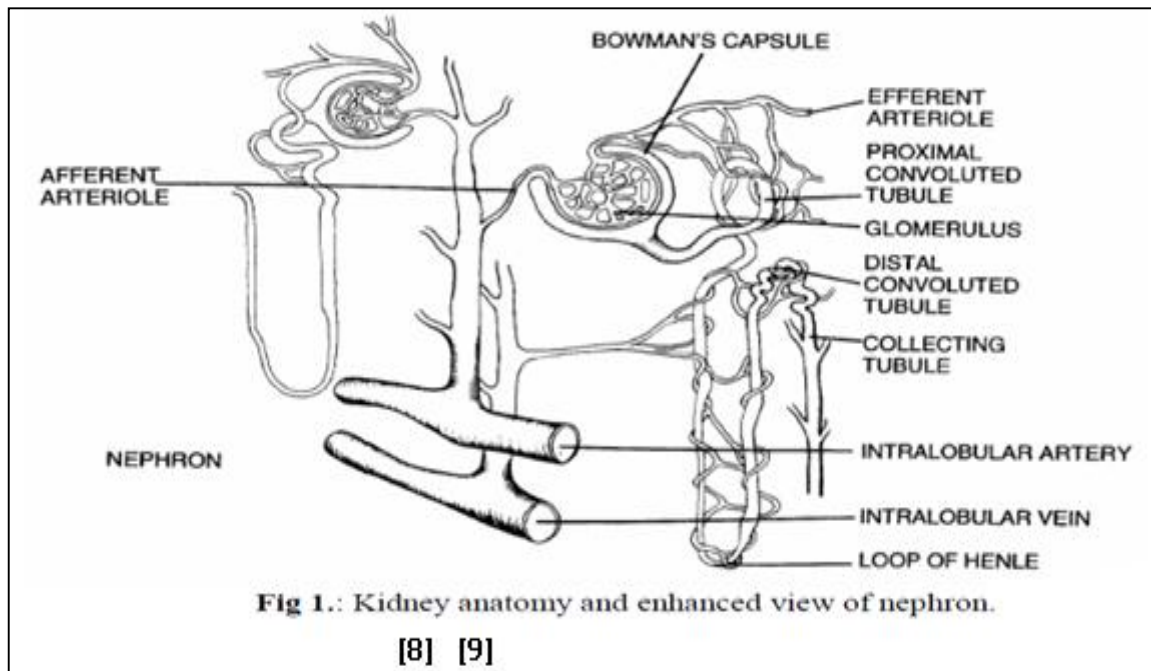
1.3 Structure & functions of Renal veinules

The renal venules (small veins) accompany the arterioles and arteries and are referred to by similar names. The venules that lie just beneath the renal capsule, called stellate venules because of their radial arrangement, drain into interlobular venules. In turn these combine to form the tributaries of the arcuate

interlobar and lobar veins. Blood from the renal pyramids passes into vessels, called venae rectae, which join the arcuate veins. In the renal sinus the lobar veins unite to form veins corresponding to the main divisions of the renal arteries and they normally fuse to constitute a single renal vein in or near the renal hilus [10]. At the venous site, capillaries converge to form the venules where smooth muscle cells gradually reappear. Sometimes the first joining of capillaries which are slightly larger in diameter than the parent capillaries but are still deyoid of any encircling smooth muscle cells are called post-capillary venules [11].

A venules is a thin walled vessel, 15-50 micrometer in diameter and 50-700 micrometer in length, formed by the confluence of several postcapillary length [12].

1.4 Function of the tissue (kidney): Kidney perform three major type of of functions (i) Maintenance of fluid and acid –base balance (ii) Removal of Nitrogenous waste products (iii) synthesis of hormones; such as renin, erythropoietin and active vitamin D_3 (calcitriol) [13] Each human kidney contains about one million nephrons, each of which consists of a renal corpuscle and a renal tubule. The renal corpuscle consists of a tuft of capillaries, the glomerulus, surrounded by Bowman's capsule. The renal tubule is divided into several segments. The part of the tubule nearest the glomerulus is the proximal tubule. This is subdivided into a proximal convoluted tubule and proximal straight tubule. The straight portion heads toward the medulla, away from the surface of the kidney. The loop of Henle includes the proximal straight tubule, thin limb, and thick ascending limb. Connecting tubules connect the next segment, the short distal convoluted tubule, to the collecting duct system. Several nephrons drain into a cortical collecting duct, which passes into an outer medullary collecting duct. In the inner medulla, inner medullary collecting ducts unite to form large papillary ducts.

[14]

V. Upadhyay*, Harish Chandra, P.N. Pandey
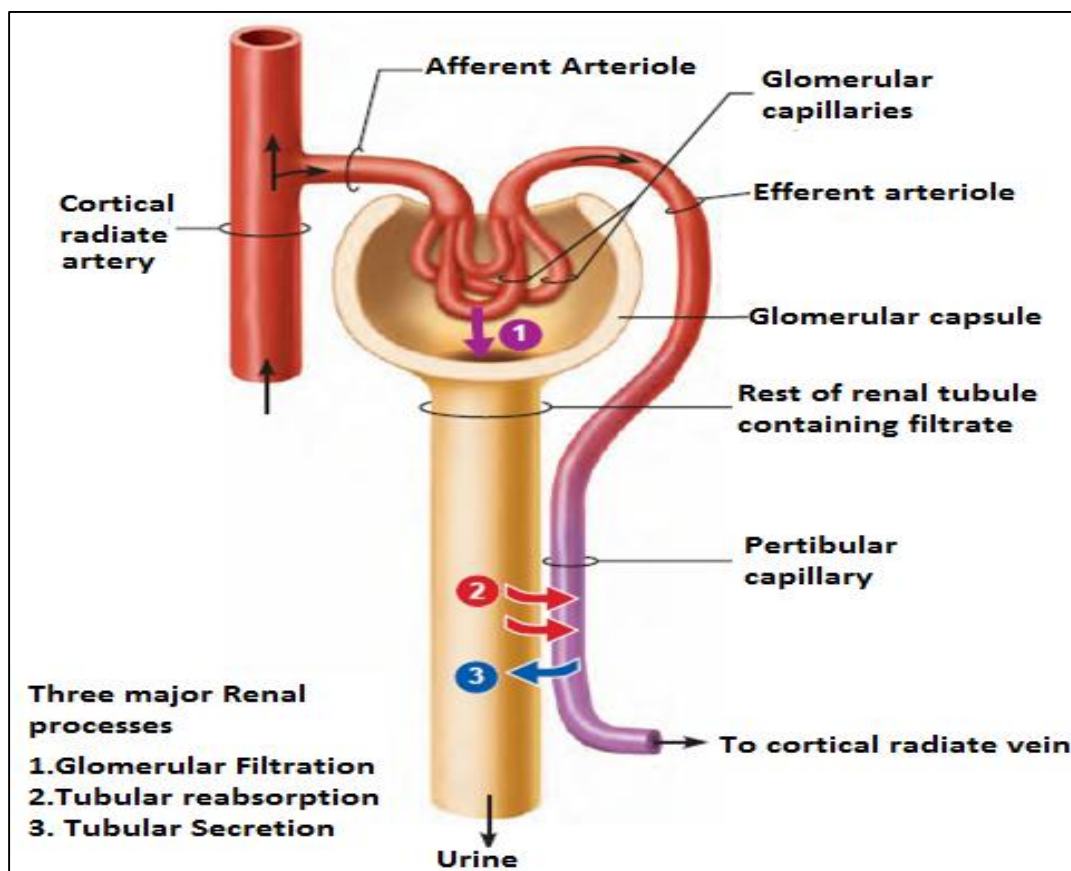
1.5 Blood Supply: Each kidney is typically supplied by a single renal artery, which branches into anterior and posterior divisions, which give rise to a total of five segmental arteries. The segmental arteries branch into interlobar arteries, which pass toward the cortex between the kidney lobes. At the junction of the cortex and medulla, the interlobar arteries branch to form arcuate arteries. These, in turn, give rise to smaller cortical radial arteries, which pass through the cortex toward the surface of the kidney. Several short, wide, muscular afferent arterioles arise from the cortical radial arteries. Each afferent arteriole gives rise to a glomerulus. The glomerular capillaries are followed by an efferent arteriole. The efferent arteriole then divides into a second capillary network, the peritubular capillaries, which surround the kidney tubules. Venous vessels, in general, lie parallel to the arterial vessels and have similar names [15]. In resting adult kidney receive 1.2 to 1.3 l blood per minut or 25% of cardiac output. Renal Blood flow canbe measured with electromagnetic or other type of flow meter or it canbe determined by applying the Fick principle [16].

From renal plasma flow, the renal blood flow can be calculated by dividing by one minus the hematocrit: Hematocrit (HCT) - 45% The renal Blood flow = RPF $\times 1/(1\text{-HCT})$ → $700 \times 1/(1\text{-}0.45)$ = 1273 ml/ Minut [17].
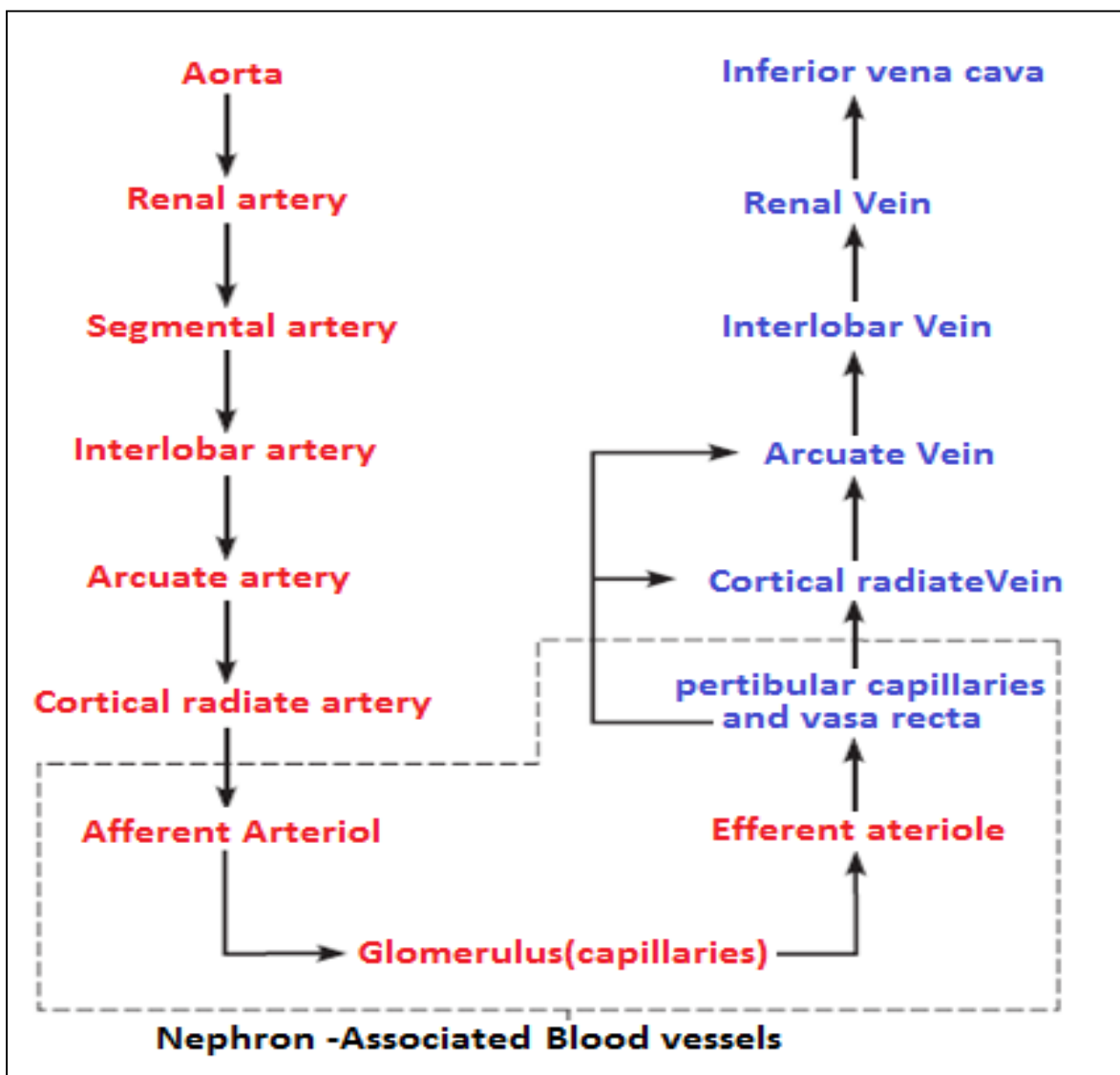
Path of the blood flow through renal blood vessels [14]

2. Real Model

2.1 Blood: 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. 55% of the plasma and 45% of the blood cells in a whole blood and approximately 98% of RBCs in 45% of blood cells and there are a few parts (approximately 2%) of the other cells. Which are ignorable, so one phase of the bloods plasma and 2nd phase of blood is RBCs [18].

The first and foremost reason is that the blood is not an ideal fluid but it is a mixture of the two phases one is of plasma and other one is of blood cells. These blood cells, semi permeable packages of liquid of a density greater than that of plasma, are capable of changing their shape and size while flowing through different blood vessels [19]. Plasma is a liquid containing semi permeable packages of RBCs.

The behavior of blood is almost Newtonian at high shear rate, while at low shear rate the blood exhibits yield stress and non-Newtonian behavior [20]. When blood flows through larger diameter arteries at high shear rates, it behaves like a Newtonian fluid. The apparent viscosity of blood decreases with decreasing blood vessel diameter less than 300 micrometer [21].

Historical Development of Two phase renal system

Two-phase models can be used to describe the dynamics of mixed materials and can be applied to many physical and biological phenomena. For example, these types of models have been used to describe the dynamics of cancer, biofilms, cytoplasm, and hydro gels. Frequently the physical domain separates into a region of mixed material immersed in a region of pure fluid solvent [22].

Two-phase models are useful for capturing the interactions between fluids and/or viscoelastic material. Each phase is averaged over a control volume, where the volume- averaged phases are incompressible. There is no inertial component to the system, and the phases are immiscible. Each phase is governed by conservation equations. These models have been successful at describing how emergent structures develop through the interactions of the two phases. There are several known applications [22].

Different constitutive equations for blood

Generally blood is non-homogeneous mixture of plasma and blood cells. Though for practical purposes it may be considered to be homogeneous two-phase mixture of plasma and blood cells. The constitutive equations proposed for whole blood mixture are as follows:

(1) Newtonian equation

$$\tau = \eta\, e$$

Where η is the viscosity coefficient.
This is found to hold good in the broad blood vessels where there is low hematocrit [23].

(2) The non-Newtonian power law equation

$$\tau = \eta\, e^{n}$$

This is found to be conformable for strain rate between 5 and 200 sec-1,

$$0.68 \leq n \leq 0.80 \ [24]$$

The non-Newtonian Herschel – Bulkley equation [25]

$$\tau = \eta\, e^{n} + \tau_0 \; (\tau \geq \tau_0)$$

$$e = 0 \; (\tau < \tau_0)$$

It holds good when blood shows yield stress τ_0. We notice that the yield stress arise because blood cells form aggregates in the form of rouleaux at low strain rate.

If $\tau < \tau_0$, no blood flow-takes place. It is found that yield stress is given by the following formula:

$$\tau_0^{\frac{1}{3}} = \frac{A\,(H - H_m)}{100} \quad \text{Where,} \quad A = \left(0.008 \pm 0.002\, \text{dyne/cm}^2\right)^{\frac{1}{3}}$$

H is normal hematocrit and H_m is the hematocrit below which there is no yield stress.

Hematocrit

Hematocrit is the volume percentage (%) of red blood cells in blood. It is normally 45% for men and 40% for women [26]. Hematocrit is the most important determinant of whole blood viscosity [27]. Blood viscosity and vascular resistance affect total peripheral resistance to blood flow [28], According to Berkow, Robert The hematocrit (expressed as percentage points) is normally about three times the hemoglobin concentration (reported as grams per deciliter) [29].

2.2 Choice of frame of references: Since we are going to analyze about mathematical modeling of the state of circulation of the blood in our body, so regarding with the problem and generality of the blood we have selected generalized three dimensional orthogonal curvilinear co-ordinate system, briefly prescribed as E3 called as 3-dim Euclidean space. Here we have some quantities related to moving blood in cylindrical vessels: blood velocity $V^k = V^k(x^i, t)$, k=1,2,3 blood pressure $P = p(x^i, t)$ and density $\rho = \rho(x^i, t)$ Let OX^i be the co-ordinate axes where O be the origin and x^i be the co-ordinates of any point in space and i-1,2,3 If let us consider that the both phases- plasma and blood cells are equally distributed in whole blood. Then blood treated as homogeneous mixture. We have interpreted all the quantity to the blood flow in tensorial form which is comparatively more realistic for the problem. The biophysical laws thus expressed fully hold good in any co-ordinate system, which is compulsion for the truthfulness of the laws.

2.3 Equation of Continuity

When there is absence of source and sink in any region of flowing fluid, the fluid mass is conserved in that region. As we observed that there is no source or sink in the whole circuit of the human blood circulatory system, the heart behaves merely like a pumping station, so the law of conservation of mass can well be applied to hemodynamic [30]. Since, whole blood flow circuit of the kidney is called a Renal Circulatory System. Hence renal circulatory system is a sub system of human circulatory system. Blood enter in kidney by arteries and out by veins and in a kidney no source or sink.

Hence, Mass of enter the blood = mass of outer the blood

Therefore law of conservation of mass can also be applied for renal circulatory system. The flow of blood is affected by the presence of blood cells. This effect is directly proportional to the volume occupied by blood cells.

2.4 Equation of Motion: According to this principle, the total momentum of any fluid system is conserved in absence of external force. So the law of conservation of momentum can well apply to

renal circulatory system. In other words, the rate of change of momentum of a fluid particle with respect to time equals to external force exerted on it. This is also called Newton's 2nd law of motion. So, the rate of change of momentum is equal to sum of about two mentioned forces, which may be symbolically presented as follows.

$$\frac{dp}{dt} = -P + F$$

Where,

$$\frac{dp}{dt} = \text{Rate of change of momentum}$$

P= internal pressures
F= viscous force

2.5 Boundary Condition: Boundary Conditions are as follows:
1. The velocity of blood flow on the axis of capillaries 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 capillary, will be zero. This condition is well known as no-slip condition.

2.6 Blood Pressure
Blood pressure is a measure of the force that the circulating blood exerts on the walls of the main arteries. The pressure wave transmitted along the arteries with each heartbeat is easily felt as the pulse—the highest (systolic) pressure is created by the heart contracting and the lowest (diastolic) pressure is measured as the heart fills [31]. Blood pressure fluctuates from minute to minute and normally shows a circadian rhythm over a 24-hour period, with highest readings in the afternoons and lowest readings at night [32-33]. Loss of the normal fall in blood pressure at night is associated with a greater future risk of cardiovascular disease and there is evidence that night-time blood pressure is a stronger predictor of cardiovascular events than day-time blood pressure [34].
Blood pressure almost always is measured in millimeters of mercury (mm Hg) because the mercury manometer has been used since antiquity as the standard reference for measuring pressure. Actually, blood pressure means the force exerted by the blood against any unit area of the vessel wall. When one says that the pressure in a vessel is 50 mm Hg, one means that the force exerted is sufficient to push a column of mercury against gravity up to a level 50 mm high. Occasionally, pressure is measured in centimeters of water (cm H_2O) [35].
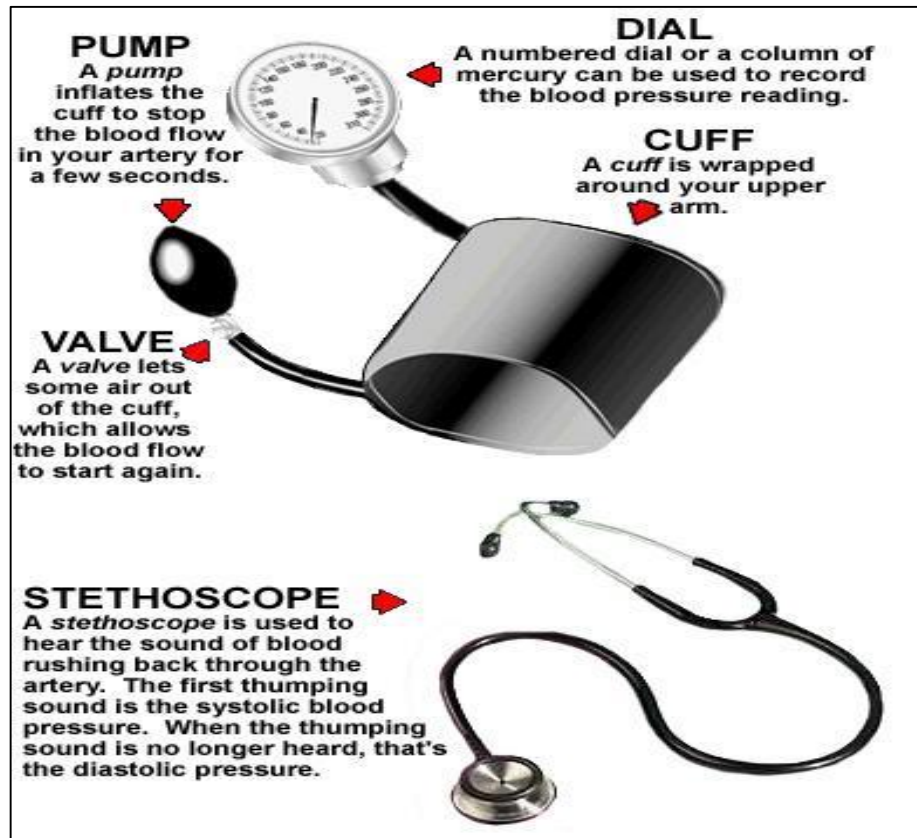
2.7 Pressure drop

Pressure difference of the blood between the two ends of the vessel(also frequently called " Pressure gradient"), which is the force that pushes the blood through the vessel. Let us consider in any blood vessels of renal circulatory system. P_1 Represents the pressure at the origin of the vessels, at the other end point pressure is P_2 . Then the pressure difference is represented by $P_1 - P_2$ blood pressure of first end point is greater than the blood pressure of other end point [35], that is

$$P_1 > P_2$$

$$\Delta p = -(P_2 - P_1)$$
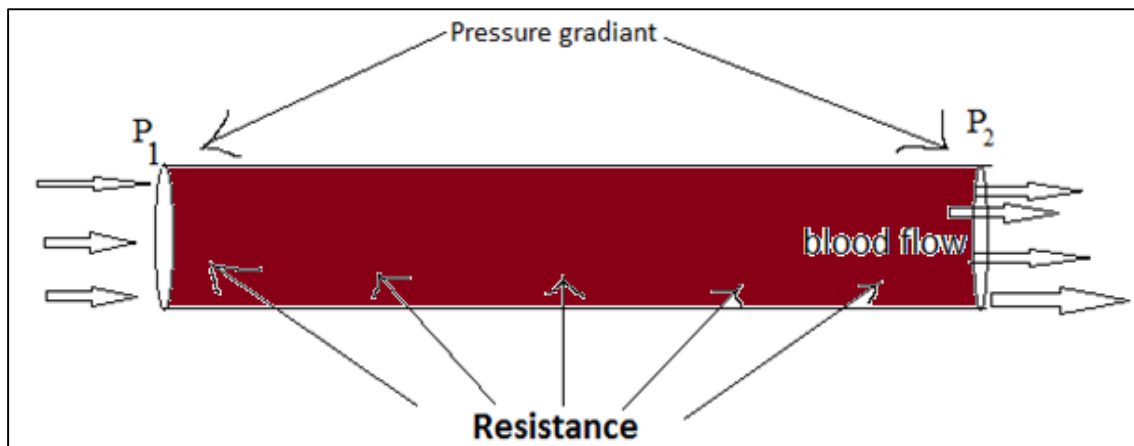
2.8 Disease (diabetes): Dibetes is the chronic disease that occurs when the pancreas does not producing enough insuline or when the body cannot effectively use the insuline it produces. Diabetes is the life threating condition affecting the millions of people [36, 37].
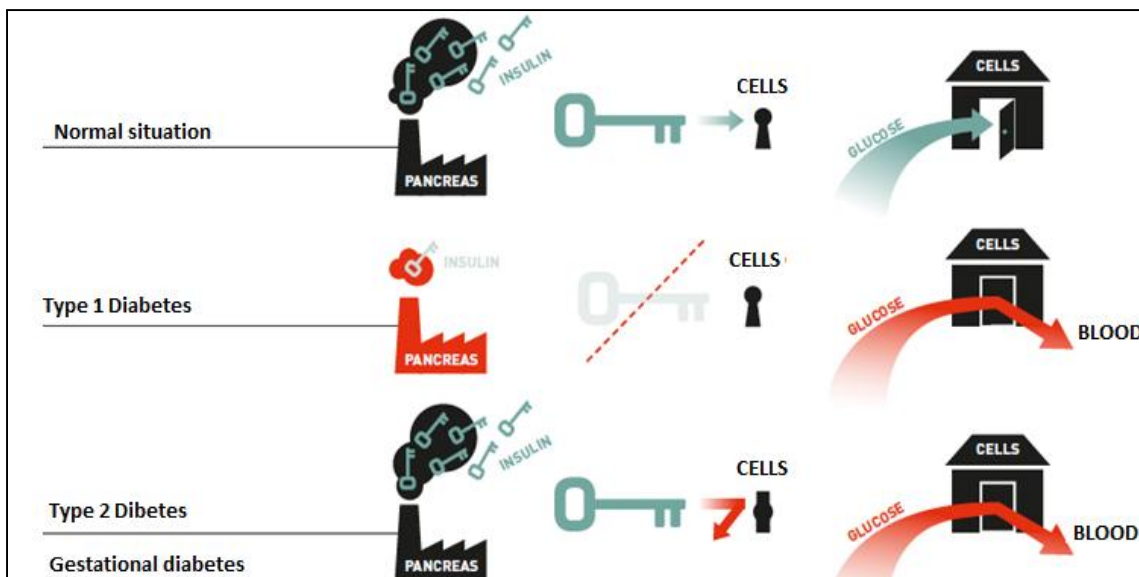

Fig 5

3. Discription of the Problem

As the velocity of Blood flow decreases, the viscosity of blood increases. The velocity of blood decreases successively because of the fact that veinules are relatively a far enough from the heart. Hence the pumping of the heart on these vessels is relatively low [38]. Secondly these vessels 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. [A c Guyton].

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

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

When strain rate $e = 0 \left(T' < T_p \right)$ 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 plug will be T_p. Equating the forces acting on the plug, we get,

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

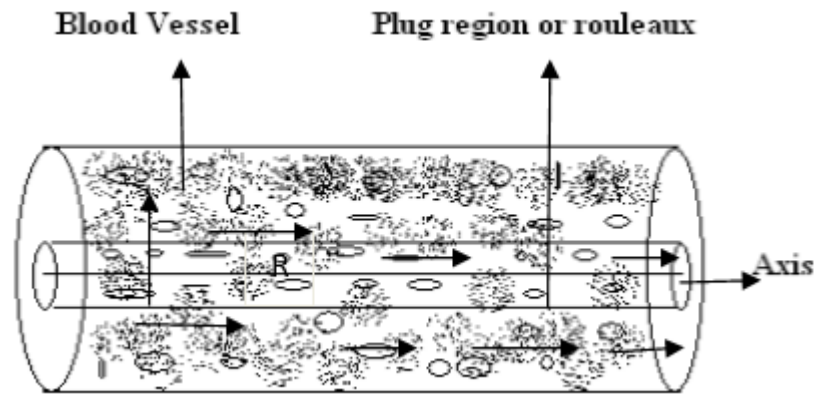$$\Rightarrow \ r_p = 2 \frac{T_p}{P} \tag{3.1}$$

R

Figure 26
Herschel Bulkley blood flow
The Constitutive equation for test part of the blood vessel is
The Constitutive equation for test part of the blood vessel is

$$T^{'} = \eta_m e^n + T_p \ \text{ or } T^{'} - T_p = \eta_m e^n = T_e$$
Where, T_e = effective stress, Whose generalized form will be as follows

$$T^{ij} = -P g^{ij} + T_e^{ij} \ \text{ where, } \ T_e^{ij} = \eta_m \left(e^{ij} \right)^n \ \text{ While } \ e^{ij} = g^{jk} V_k^i$$

Where, the symbols have their usual meanings.
Now we describe the basic equations for Herschel Bulkley blood flow as follows:

3.1 Equation of Continuity
When there is absence of source and sink in any region of flowing fluid, the fluid mass is conserved in that region. As we observed that there is no source or sink in the whole circuit of the human blood circulatory system, the heart behaves merely like a pumping station, so the law of conservation of mass can well be applied to hemodynamic [38]. Since, whole blood flow circuit of the kidney is called a Renal Circulatory System. Hence renal circulatory system is a sub system of human circulatory system. Blood enter in kidney by arteries and out by veins and in a kidney no source or sink.
Mass of enter the blood = mass of outer the blood
Therefore law of conservation of mass can also be applied for renal circulatory system.
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 X is the volume portion covered by the blood cells in unit volume. And X can be replaced by H/100, where H is the 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 clearly.

Unit volume of Blood
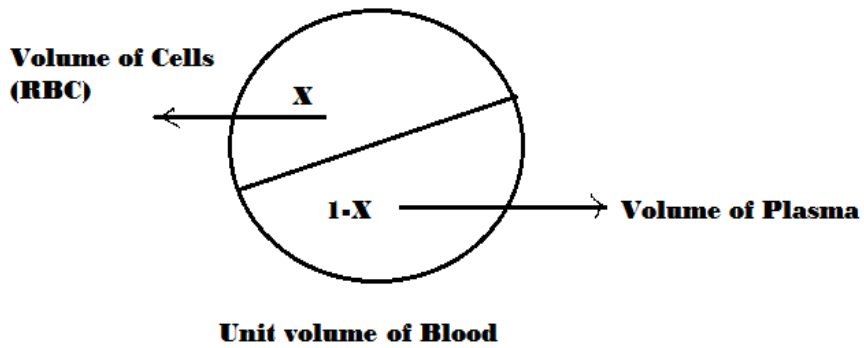
$$r = \frac{x\rho_c}{(1-x)\rho_p}$$

Where ρ_c and ρ_p are densities of blood cells and blood plasma respectively. Usually this mass ratio is not constant; even then this may be supposed to be constant in present context [39].

The both phase of blood, i.e., blood cells and plasma move with a common velocity. Campbell and Pitcher have presented a model for this situation. According to this model we consider the two phases of blood separately [40]. Hence according to principle of conservation of mass, the equations of continuity for the two phases are as follows [41].

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

........3.

1.1 [for cells (RBC)]

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

........3.

1.2 [for plasma]

Where v is the common velocity of the two phases blood cells and plasma and $\left(X\rho_c v^i\right)_{,i}$ is co-variant derivative of $\left(X\rho_c v^i\right)$ with respect to X^i, in the same way $\left((1-X)\rho_p v^i\right)$ with respect to X^i

If we define the uniform density ρ_m as follows:

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

(3.1.3)

Then the equations can be combined together as follows;

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

As we know that blood is incompressible fluid, hence ρ_m will be a constant quantity. Thus the equation of continuity for blood flow takes the following form:

$$v^i_{,i} = 0$$

i.e.

$$\frac{\partial v^i}{\partial x^i} + \frac{v^i \partial \sqrt{g}}{\sqrt{g} \partial x^i} = \frac{1}{\sqrt{g}} \left(\sqrt{g} v^i \right)_{,i} = 0$$

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

(3.1.4)

3.2 Equation of Motion

$$\rho_m \frac{\partial v^i}{\partial t} + \rho_m V^j V^i_{,j} = - T^{ij}_{e,j}$$

(3.2.1)

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 earlier: $X^1 = r, X^2 = \theta, X^3 = Z$ Matrix of metric tensor in cylindrical co-ordinates is $\left[g_{ij} \right]$ and matrix of conjugate metric tensor is $\left[g^{ij} \right]$ whereas the chritoffel's symbols of 2nd kind are as follows:

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

The governing tensorial equations can be transformed into cylindrical forms which are follows: the equation of Continuity-: $\frac{\partial v}{\partial z} = 0$

3.3 The equation of Motion

r-component: $-\frac{\partial p}{\partial z} = 0$, $\theta - component : 0 = 0$

Z-component:
$$0 = -\frac{\partial p}{\partial z} + \frac{\eta_m}{r} \left[r \left(\frac{\partial v_z}{\partial r} \right)^n \right] \dots\dots\dots\dots\dots\dots(3.2.2)$$

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

We get $v_z = v(r)$ and $dp = p(z)$

and

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

(3.2.3)

Since, pressure gradient

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

$$r\left(\frac{dv}{dz}\right)^n = -\frac{pr^2}{2\eta_m} + A ,$$

We apply boundary condition: at r=0. $V = V_0$ then

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

Replace r from $r - r_p$

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

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

(3.2.4)

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

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

(3.2.5)

This is the formula for velocity of blood flow in arterioles is.

Putting $r = r_p$ to get the velocity V_p of plug flow as follows:

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

(3.2.6)

4. Bio-physical interpretation
4.1 Clinical data-1
Patient Name – Jasbir kaur kohali

Table 4.1

Sno.	HB	Hematocrit	BP mmgh	Pascal S
1	8.8	26.4	120/80	15998.6/10665.8
2	8.2	24.6	130/90	17331.9/11998.9
3	8.9	26.7	120/70	15998.6/9332
4	9.3	27.9	130/90	17331.9/11998.9
5	9.1	27.3	110/80	14665.4/10665.8

According to Berkow, Robert The hematocrit (expressed as percentage points) is normally about three times the hemoglobin concentration (reported as grams per deciliter) [29].

The flow flux of two phased blood flow in arterioles, veinules and veins is

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

Average systolic blood pressure= 16265.28
Average diastolic blood pressure = 10932.4
Pressure at venules

$$\frac{2}{3}\left[\frac{\frac{D+S}{2}+D}{3}\right] = \frac{2}{3}\left[\frac{\frac{16265.3+10932.4}{2}+10932.4}{3}\right] = 5451.3 \text{ mmhg}$$

Pressure at veins

$$p_f = \frac{1}{3} \times p_i = \frac{1}{3} \times \frac{2}{3} \left[\frac{\frac{D+S}{2} + D}{3} \right]$$

$$= \frac{1}{3} \times \frac{2}{3} \left[\frac{\frac{16265.3 + 10932.4}{2} + 10932.4}{3} \right] = \frac{1}{3} \times 5451.08 = 1817.1 \ mmhg$$

Length of left venules $= 3.75 \times 10^{-4}$

Viscosity of plasma $= \eta_p = 1.2 \times 10^{-3}$

Viscosity of mixture (blood) $= \eta_m = 1.5 \times 10^{-3}$

Flow flux $Q = 1100 \ ml = 0.01833 \ meter/second$

Average hematocrit $= 26.58$

Calculation for η_c

Now, $\eta_m = \eta_c \left(\frac{H}{100} \right) + \eta_p \left(1 - \frac{H}{100} \right)$

$\Rightarrow 3.5 \times 10^{-3} = \eta_c \left(\frac{26.58}{100} \right) + 1.2 \times 10^{-3} \left(1 - \frac{26.58}{100} \right) \Rightarrow \eta_c = 0.01002$

From Herschelbelkeley model –

$$\left(\frac{27Q}{2\pi A} \right)^n = \left(\frac{p_f - p_i}{z_i - z_f} \right) \times \left(\frac{1}{3\eta_m} \right) \quad where \ A = \frac{29n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$\frac{Q \times 27}{2\pi} \left(\frac{z_i - z_f}{p_f - p_i} \right)^{\frac{1}{n}} (3\eta_m)^{\frac{1}{n}} = \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$\frac{27Q}{2\pi} = \left(\frac{p_f - p_i}{\text{length of vein}} \times \frac{1}{3\eta_m}\right)^{\frac{1}{n}} \times \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$\frac{27 \times 0.01833}{2 \times 3.14} = \left(\frac{5451.3 - 1817.1}{3.75 \times 10^{-4}} \times \frac{1}{3 \times 3.5 \times 10^{-3}}\right)^{\frac{1}{n}} \times \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$0.0788073 = (922971428.6)^{\frac{1}{n}} \times \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

On solving above equation by hid and trial method n= - 5.280944

$$\left(\frac{27Q}{2\pi A}\right)^n = \frac{\Delta p}{z_i - z_f} \times \frac{1}{3\eta_m}, \qquad \text{where} \quad A = \frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1}$$

$$\Delta p = 3\eta_m \left(z_i - z_f\right)\left(\frac{27Q}{2\pi A}\right)^n$$

$$\Rightarrow \quad \Delta p = 3\eta_m \left(3.75 \times 10^{-4}\right)\left\{\left(\frac{27 \times 0.01833}{6.28}\right)\left(\frac{6n^3 + 11n^2 + 6n + 1}{26n^3 + 33n^2 + 9n}\right)\right\}^n$$

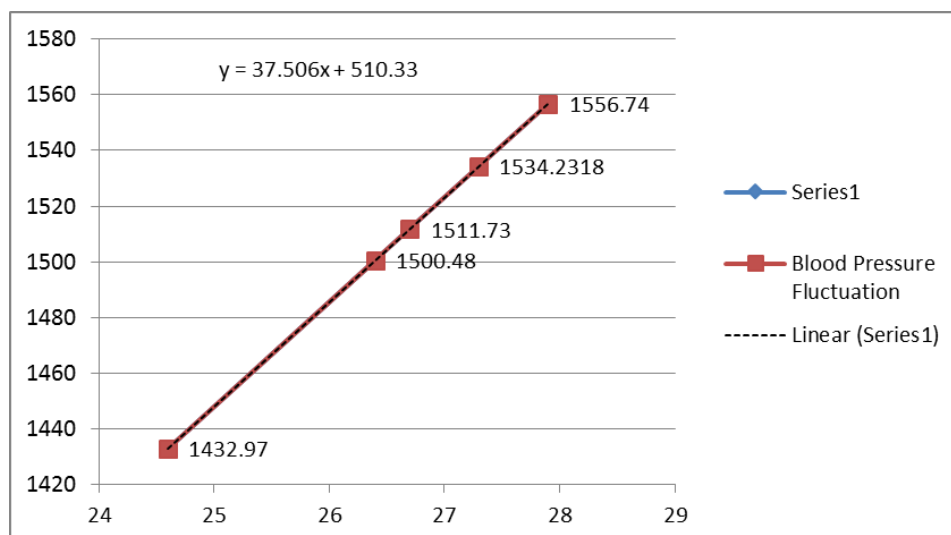$$\Delta p = 3\eta_m \left(3.75 \times 10^{-4}\right)(671691.83) \qquad \text{using } n = -5.280944$$

$$\Delta p = \eta_m \times 425265.32 \quad \Rightarrow \quad \Delta p = \left\{\eta_c \times \frac{H}{100} + \left(1 - \frac{H}{100}\right)\eta_c\right\} \times 425265.32$$

$$\Delta p = 37.506H + 510.318 \quad \dots\dots\dots\dots\dots\dots\dots\dots(4.1)$$

Using above relation (4.1)

4.1.1 Result and discussion

S. no.	1	2	3	4	5
H	26.4	24.6	26.7	27.9	27.3
Δp	1500.48	1432.97	1511.73	1556.74	1534.2318

Graph 4.1

4.1.2 Conclusion: In Bio physical Interpretation, We have taken clinical data regarding with Blood Pressure and Hematocrit of Diabetic Patient. And we get the relation $\Delta p = 37.506H + 510.318$, by using the Two phase non-Newtonian Model (Herschel Bulkley blood flow) and draw the graph between Blood pressure drop in renal A Venules in Non-Newtonian flow and Hematocrit. and trend of graph shows the relation between Blood Pressure drop and Hematocrit as linear as y= 37.5x+510.3. This linear ralation approves the two phase relation $\eta_m = \eta_c X + \eta_p (1 - X)$ where X= H/100 and slop of trend line is … 37.5

5. Reference

1. International journal of innovative Research in electrical, Electronic, Instrumentation and Control Engg. 2011; 1st:1
2. Cotran RSS, Kumar Cotran RSS, Kumar Vinay, Fausto Nelson, Robbins Stanley L, Abbas Abul K. Robbins and Cotran pathologic basis of disease. St. Louis, MO: Elsevier Saunders, 2005. ISBN 0-7216-0187-1.
3. Cotran RSS, kumar VInay, Fausto Nelson, Robins Stanley L, Abbas Abdul K. Robbins and Catron Pathologic basis of disease. St. Louis, MO; Elsevier Saunders, 2005. ISBN 0-7216-0187-1
4. Reddi AS. Structure and function of kidney. In ReddiAS. Essential of renal physiology. New Jeresy. College book, Publisher, 1992, 21-43.
5. Madsen KM, Tisher CC. Anotomy of kidney. In Brenner BM, ed. Brenner and Rector's. The kidney, 7th ed. Pheladelphia: Saunders, 2004; 1:3-72.
6. Kriz W, Elgar M. Renal anatomy. In Johnson RJ, Feehally J, eds. Comprehenshive clinical nephrology, 2nd ed. Edinburgh Mosby, 2003, 1-11.
7. Essential of Clinical Nephrology; 1st edition; Published by; Dar EI Shorouk, 8 Sebawieh Al masry, Nasr City, Cairo, Egypt; post box: 33 Pnorama: dar@shrouk.com
8. Human Kidney. Diagram, with nephron, Pearson publishing. Lab Art Library. 96L, 12001. wwwlabartlibrary.com/symbiosis/96/96L12001.pdf.juli7, 2003.
9. International journal of Engineering Research and development e-ISSN: 227 067X, p-ISSN: 2278-800X, www.ijerd.com, 2012; 5(5):23-30.
10. Rogers kara. The Kidneys and the renal system; Book; Britannica Educational Publishing; Juvenile Nonfiction. 2011.

11. Esmail Koushanpour, Wilhelm Kriz. Renal Physiology; Principles, Structure and function; Springer Science & Business media. 2011; 24:14.

12. Radiovoj Krstic V. Human Microscopic anatomy: An Atlas for students of medicine and Biology; Springer Science & Business media. 1991.

13. Nutrition and Health. Nutrition Kidney disease; edited by LD Byham-Grey, JD. Burrowes and GM Chertow © Humana Press; Totowa; NJ.

14. Human Anatomy & Physiology; Elaine N. Marieb Katja Hoehn; Eighth Edition; chapter 25, Urinary System, 971.

15. Renal physiology and Body Fluids; chapter-22 Kidney Function; George A Tanner; Ph.D. 393.

16. Review of Medical physiology; 23 edition, Kim Barrett, Headwen, Brooks Scott Boitano Susan Baman, 644.

17. Kapur JN. Mathematical Models in Biology & Medicine, EWP New Delhi, 1992, 354.

18. Shmukler Michael ; " Density of blood " ; The physics factbook ; 2004

19. Sherman IW, Sherman VG. Biology- a human approach oxford univ. press New York, oxford, 1989, 276-277.

20. Sapna Ratan Shah. Mathematical analysis of blood flow throw atherosclerotic arterial segment having non-symmetric and mild stenosis; International journal of research in pure & applied physics. 2011.

21. Fahraeus R, Lindqvist R. The viscosity of the blood in narrow capillary tubes. American journal physiology. 1931; 96:562-568.

22. Breward CJ, Byrne HM, Lewis CE. The role of cell-cell interactions in a two-phase model for avascular tumor groth; J.Math. Biol, 2002; 45:125-152.

23. Taylor MG. Hemodynamics. Ann. Rev. Physiol. 1973; 35:87.

24. Kapur JN. Mathematical Models in Biology & Medicine, EWP New Delhi, 1992, 353.

25. Kapur JN. Mathematical Models in Biology & Medicine, EWP New Delhi, 1992, 354.

26. Purves William K, Sadava David, Orians Gordon H, Heller H. Craig. Life: The Science of Biology (7th ed.). Sunderland, Mass: Sinauer Associates. 2004, 954. ISBN 0-7167-9856-5.

27. Stuart J, Kenny MW. Blood rheology. J din Pathol. 1980; 3:417-429.

28. Chien S. Blood rheology in hypertension and cardiovascular disease. Cardiovasc Med. 1977; 2:356-360.

29. Berkow Robert. ed. Merk Manual of Medical information. White house station, NJ: Merck Raseach Laboratory, 1997.

30. Fogelson AL. A mathematical model and numerical method for studying platelet adhesion and aggregation during blood clotting; J. comput. physics. 1984, 56.

31. Carlene Lawes MM, Stephen Vander Hoorn, Malcolm Law R, Paul Elliott, Stephen Mac Mahon, Anthony Rodgers. Chapter 6; High blood pressure; Comparative Quantification of Health Risks, 284.

32. Table. Comparison of ambulatory blood pressures and urinary norepinephrine and epinephrine excretion measured at work, home, and during sleep between European–American (n = 110) and African–American (n = 51) women

33. Van Berge-Landry HM, Bovbjerg DH, James GD, Bovbjerg James. Relationship between waking-sleep blood pressure and catecholamine changes in African-American and European-American women. Blood Press Monit. 2008; 13(5):257-62. doi:10.1097/MBP.0b013e3283078f45

34. Hansen TW, Li Y, Boggia J, Thijs L, Richart T, Staessen JA. Predictive Role of the Nighttime Blood Pressure. Hypertension. 57(1):3-10. doi:10.1161/Hypertensionaha.109.133900. ISSN 0194-911X.

35. Guyton AC. Medical Physiology; Overview of the circulation; Medical physics of pressure, flow, and Resistannce; Chapter 14; Tenth Edition, 146.

36. Shoback. edited by David G Garner, Dolores chapter 17. Greenspan's basic and clinical endocrinology (9th Edit.). Newyork: McGraw-Hill Medical, 2011. ISBN 0-07 -162243-8

37. Walter Boron F. Medical physiology: A cellular and Molecular Aproach, 2004. Elsevier / Saunders. ISBN1-4160-2328-3

38. As Biology – Module -1; chapter -6; circulatory System; Topic- Heart