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Mathematical modeling on two phase hepatic systolic blood flow through arteries due to liver cirrhosis

Anil Kumar, V Upadhyay, AK Agrawal and PN Pandey

Abstract
In this investigation, we are considering the two phase blood flow in artery presented here. 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 non-Newtonian power law model in bio fluid mechanical set-up. We have collected a clinical data in the case of Liver Cirrhosis. 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 Liver Cirrhosis infection. The graphical presentation for particular parametric value is much closer to the clinical observation.

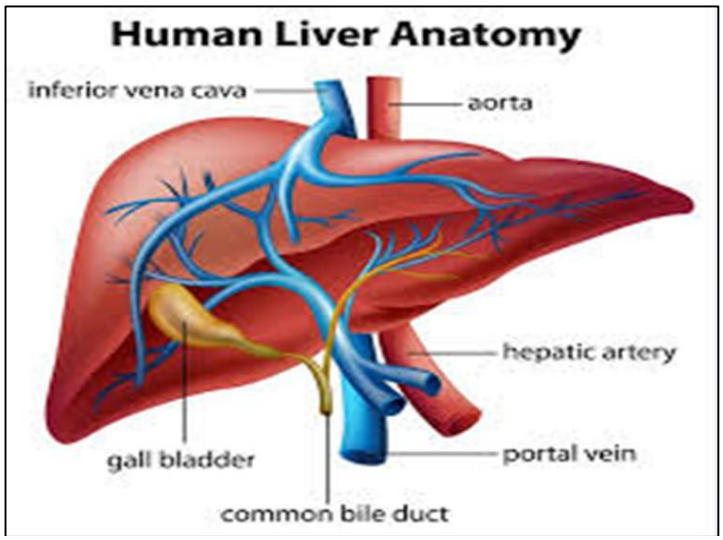
Keywords: Liver cirrhosis, hematocrit, hepatic blood flow, non-newtonian power law model, circulatory system, liver

Introduction

Structure and Function of Liver

The liver is the most vital organ in the human body and performs all important functions that impact all body systems. The liver has lobular structure and lies in the abdominal cavity below diaphragm. The liver is made up of two major lobes comprising many smaller lobules. The liver plays many physiological roles including metabolism, decomposition of red blood cells, synthesis of serum proteins and detoxification. The liver also produces bile, an enzyme that aids in digestion by emulsifying lipids. The circulatory system of the liver is different from that of other organs. Roughly 75% of the blood entering in liver through the portal vein is the venous blood returning back from the small intestine, stomach, pancreas, and spleen. From this portal venous blood all nutrients along with drugs and other potentially harmful substances are absorbed. The remaining 25% of the arterial blood received by liver is the oxygenated blood being carried from the pulmonary system to the liver by the hepatic artery. The blood contents of the hepatic artery as well as hepatic portal vein empty into sinusoids. Sinusoidal blood moves towards the central vein of each lobule and empties its content. Hepatic veins carry deoxygenated blood from liver to the inferior vena cava [1].

The liver is covered with a connective tissue capsule (Glasson's capsule) except at a region where blood vessels and hepatic/bile ducts enter and/or leave the organ. Branches of the connective tissue extend throughout the liver as spate. This connective tissue provides a network, support and the highway along which lymphatic vessels, bile ducts and afferent blood vessels can traverse across the liver. The parenchyma of the liver divides into small units called lobules with the help of connective tissue sheet [2]. These hepatic lobules are the structural unit of the liver. This lobule consists of a hexagonal arrangement of plates named as hepatocytes, radiating outward from a central vein. The portal triad is a triangular area, comprising of a bile duct and a terminal branch, each, of the hepatic artery and the portal vein. The lateral branches of these vessels are confluent with the thin-walled hepatic sinusoids that are present between the branching hepatic plates/cords [3]. Unlike other capillaries, liver sinusoids provide large surface area for the exchange of metabolites between blood and hepatocytes as sinusoids have their endothelium which 4 lacks the basal membrane. The sub-endothelial space called as the space of per sinusoidal space, separates endothelium from the hepatocytes plates [4].



Representation of liver anatomy

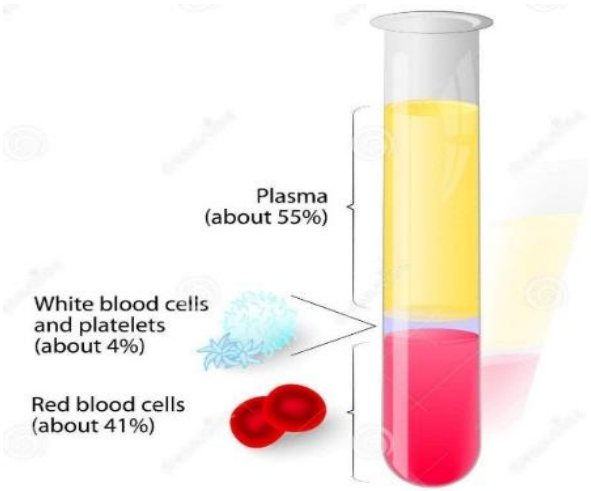
Although only limited data exist, it appears that hepatic blood volume ranges from 25to 30mL/100g liver weight, and accounts for 10%-15% of the total blood volume [5]. Furthermore, rough estimation suggests that more than 40% of the hepatic blood is held in large capacitance vessels (portal vein, hepatic artery and hepatic veins), while the sinusoids accommodate up to 60%. As small vessel content [6]. Of note is the high compliance of the hepatic vascular bed, calculated as the change in blood volume per unit change in venous pressure [7].

Structure and Function of Hepatic Artery

The common hepatic artery is one of the final branches of the celiac artery. It supplies oxygen-rich blood to the liver, pylorus, pancreas, and duodenum. It runs on the right inside the lesser sac, a cavity near the middle of the abdomen, and enters the lesser momentum, a folded membrane that attaches the stomach to the liver. The artery then passes upward toward the portahepatis, a deep groove in the back of the liver through which many neurovascular structures enter and leave the liver. The common hepatic artery splits into the proper hepatic artery and the gastro duodenal artery. The proper hepatic artery enters the portahepatis where it splits into the left and right hepatic arteries that supply the liver. As in any other artery of the body, oxygen saturation portal blood during the fasting state range up to 85%,which is greater than that of other systemic veins; however, it substantially drop after food ingestion. It is generally accepted that 50% of the oxygen requirements of the liver are provided by portal venous blood and the other half derives from the hepatic artery [8]. The liver normally receives more oxygen than it requires, and it can extract more oxygen to compensate for reduced delivery [9]. The hepatic arterial blood Flow was on the average 35% of the hepatic venous blood. The function of the hepatic artery has been extensively studied in animals [10]. Great differences have been found from one Species to another [11], and our knowledge of the significance of the arterial blood supply to the human liver is limited mainly to observations of the late effect of occlusion of the hepatic artery [12] or the portal veins [13]. The liver volume and portal blood flow decreases after the age of 50 [14].

Constitutions of Blood

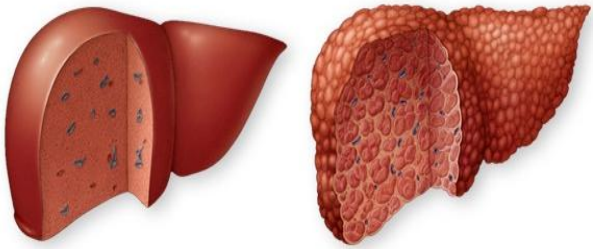
Blood circulates through the body bringing O2 and nutrients to the tissues and removing CO2 and other waste products. As it moves around the body it aids interchange between the fluid compartments, dissipates heat and distributes hormones, thus helping to maintain homeostasis and to coordinate the activities of the various organs. In addition blood contains hemostatic components that control bleeding. Finally, it performs a role in defending the body against foreign invaders as it carries cells and antibodies foreign proteins. That seek out and destroy microorganisms and Blood can be separated into two components - a yellowish fluid, plasma, and cells which are suspended in it. Plasma is that part of the extracellular fluid which is restricted to the blood vessels.



Plasma which constitutes 55% of blood fluid is mostly water (92%) by the volume) [15]. Blood account for 7% of the human body weight [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] Whole blood (plasma and cells) exhibits non-Newtonian fluid dynamics. Red blood cells contain the blood's hemoglobin and distribute oxygen [18]. White blood cells are the part of the body's immune system. They destroy and remove old or aberrant cells and cellular debris as well as attack infection agents [19]. Thrombocytes also called platelets; thrombocytes are responsible for blood clotting about 55% of blood is blood plasma. 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. Hematocrit is three times of hemoglobin concentration Blood shows anomalous viscous properties. The anomalous behavior of blood is principally due to the suspension of particles in plasma. The two type of anomaly are due to „low shear" and „high shear" effect [20]. When blood flows through larger diameter fluid. The apparent viscosity of blood decreases with decreasing blood vessel diameter, when measurements are made in capillaries of diameter less than 300μm [21]. This apparent dependence of viscosity on capillary radius is known as the Fahraeus-Lindqvist effect. But, when blood flow in smaller blood vessels of diameter 20μm-100μm the apparent viscosity increases as the blood vessel diameter decreases and it shows a non-Newtonian character. This non-Newtonian character of blood is typical in small arteries and veins where the presence of cells induces that specific behavior the analysis of two-fluid models for blood flow is better applied to small vessels such as femoral arteries carotid, coronaries, very small arteries of diameter 130μm-200μm where the non-Newtonian effect are excepted to significant [22]. The study of two-fluid models of Newtonian fluid, power law fluid and Bingham fluid can be possible by using this model as these fluid models are the particular cases of Herschel-Bulkley fluid model. Thus, in this paper, we study two phase model for systolic blood flow in hepatic artery due to Liver Cirrhosis. P.N. Pandey and V. Upadhyay (2001) discussed some phenomenon in two phase blood flow gave an idea on the two phase hepatic blood flow in artery with a liver disease Liver Cirrhosis. The work of P.N. Pandey and Upadhyay in whole circulatory system but this work will be focus on Hepatic circulatory system, and Hepatic circulatory system is a sub system of whole circulatory system. In this work, applied the Herschel Bulkley non-Newtonian power law model.

Description of Disease

Liver is also prone to many diseases [23] one of these is Cirrhosis of Liver. Cirrhosis is a slowly progressing disease in which healthy liver tissue is replaced with scar tissue, eventually preventing the liver from functioning properly. The scar tissue blocks the flow of blood through the liver and slows the processing of nutrients, hormones, drugs, and naturally produced toxins. It also slows the production of proteins and other substances made by the liver. According to the National Institutes of Health, cirrhosis is the 12th leading cause of death by disease. Cirrhosis Caused by the Hepatitis C, fatty liver, and alcohol abuse are the most common causes of cirrhosis of the liver in the U.S., but anything that damages the liver can cause cirrhosis, including: Fatty liver associated with obesity and diabetes Chronic viral infections of the liver (hepatitis types B, C, and D; Hepatitis D is extremely rare) Blockage of the bile duct, which carries bile formed in the liver to the intestines. Bouts of heart failure with fluid backing up into the liver certain inherited diseases. Although less likely, other causes of cirrhosis include reactions to prescription drugs, prolonged exposure to environmental toxins, or parasitic infections. The term cirrhosis denotes chronic tissue degeneration in which cells are destroyed leading to the formation of fibrous scar tissue. As the cellular destruction continues, blood, lymph and bile channels within the liver become distorted and compressed, leading to intrahepatic congestion, portal hypertension and impaired liver function. The fibrous changes within the organ cause it to become firmer and smaller. The surface, however, becomes rough and bumpy because of the development of nodules on the surf ace of the organ. The nodules are regenerated hepatic cells.



Normal liver v/s cirrhotic Liver

Cirrhosis was the commonest liver disease (25%) followed by chronic hepatitis (22%). Hepatic statuses accounted for 17% of the cases, portal triadic is for 15%, and congestive liver and miscellaneous cases accounted for 5% each. Majority (74%) of the livers were of normal weight between 1000-1500 grams, followed by 19 cases of hepatomegaly i.e.14 cases weighing between 1501-2000 grams and 5 cases weighing between 2001-2500 grams. Only 7 cases weighed less than 999 grams [24]. Globally, 57% of cirrhosis was attributable to either HBV (30%) or HCV (27%) and 78% of HCC was attributable to HBV (53%) or HCV (25%). Regionally, these infections usually accounted for >50% of HCC and cirrhosis. Applied to 2002
worldwide mortality estimates, these fractions represent 929,000 deaths due to chronic HBV and HCV infections, including 446,000 cirrhosis deaths (HBV: n = 235,000; HCV: n = 211,000) and 483,000 liver cancer deaths (HBV: n = 328,000; HCV: n = 155,000) [25].

Real Modal

Blood is a complex fluid consisting of particulate corpuscles suspended in a non-Newtonian fluid. The particulate solid are red blood cells (RBCs), white blood cells (WBCs) and platelets. 50% of the plasma and45% of the blood cells in a whole blood and

approximately 98% of RBCs in 45% of blood cells and there are few parts (approximately 2%) of the other cells. Which are ignorable, so one phase of the blood plasma and second phase of blood is RBCs. Boundary conditions are as follows.

1. The velocity of blood flow on the axis of artery at r =0 will be maximum and finite, say v_0.
2. The velocity of blood flow on the wall of the blood vessels at r=R, Where, R is the radius of traverse. Section of artery, will be zero. This condition is well known as no-slip condition.
   The Newtonian power law equation-$\tau = \eta e^n$ Where,η is the viscosity of coefficient [26]. This is found to hold good in broad blood vessels where there is low hematocrit. Pressure difference is a difference of pressure of two end points of the vessels. Let us consider in any blood vessels of hepatic circulatory system.Let P_i represents the pressure at the origin of the vessels, at the other end point pressure is P_f. Then the pressure difference is represented by $P_i - P_f$ blood pressure of the first end point is greater than the blood pressure of other end point
   , that is $P_i > P_f \Delta P = -(P_f - P_i)$

Basic Bio-Fluid Equation for Two Phase Blood Flow

Let us problem of blood flow in hepatic circulatory system is different from the problems in cylindrical tube and select generalized three dimensional orthogonal curvilinear coordinate system. Briefly described as E3 called as Euclidean space. According to Mishra the biophysical laws thus expressed fully hold good in any coordinate system which is a compulsion for the truthfulness of the laws [27]. According to the Sherman I.W. and Sherman V.G. blood is mixed fluid [28]. Mainly there are two phases in blood. The first phase is plasma, while the other phase is that of the blood cells are enclosed with semi permeable membranes whose density is greater than that of plasma. These blood cells are uniformly distributed in plasma. Thus, blood can be considered as a homogeneous mixture of two phases [28].

Equation of Continuity for two phase blood flow

According to Singh P. and Upadhyay K.S. The flow of blood is affected by the presence of blood cells. This effect is directly proportional to the volume occupied by blood cells [29]. 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. If the mass ratio of blood cells to plasma is r then clearly.

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

Where ρ_c and ρ_p are densities of blood cells and blood plasma respectively. Usually this mass ratio is not a constant, even then this may be supposed to constant in present context (1986) The both phase of blood, i.e. blood cells and plasma move with the common velocity. Campbell and Pitcher has presented a model for this situation. According to this model, we consider the two phase of blood separately (1958) Hence equation of continuity for two phase according to the principle of conservation of mass defined by J.N and Gupta R.C. as follow $\frac{\partial(X\rho_c)}{\partial t} + (X\rho_c v^i)_{,i} = 0$ (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^i= Common velocity of two phase blood cells and plasma. And again $(X\rho_c v^i)_{,i}$ co-variant derivative of $(X\rho_c v^i)$ with respect to X^i.In the same way $\left[(1-X)\rho_p v^i\right]_{,i}$is co-variant derivative of $(1-X)\rho_p v^i$ with respect to X^i.If we define the uniform density of the blood $\boldsymbol{\rho_m}$ as follows:

$\frac{1+r}{\rho_m} = \frac{r}{\rho_c} + \frac{1}{\rho_p}$[30] (3.4)

Then equation (3.2) and (3.3) can be combined together as follow

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

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

Equation of Motion for two phase blood flow

According to Ruch, T.C. and H.D. 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 (1973) [31]. 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 two phase of blood cells as follows:

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

The equation of motion for plasma will be as follows:

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

Now adding equation (3.6) and (3.7) and using relation (3.4), the equation of motion for blood flow
With the both phases will be as follows:

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

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

Mathematical Modeling

We consider the two layer blood flow to be Newtonian. The first layer is that of plasma while second one is core layer. Let the viscosity of plasma layer be η_P and that of the core layer η_m where $\eta_m = X_{\eta_c} + (1 - X)\eta_P$ where $\eta_c$is the viscosity of the blood cells and X is portion of blood cells in unit volume. Now the basic equation can be written in a similar way as before. Now we describe the basic equation for Power law blood flow as follows: $\tau = \eta e^n$
Equation of continuity in tensorial form as follows:

$$\frac{1}{\sqrt{g}} \left(\sqrt{g}\, v^i \right)_{,i} = 0 \tag{4.1}$$

Equation of motion

$$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j) v^i_{,j} = -P_{,j} g^{ij} + \eta_m \left(g^{jk} v^i_{,k} \right)_{,j} \tag{4.2}$$

Where$\rho_m = X\rho_c + (1 - X)\rho_p$is the density of blood as mixture of blood cells and plasma.
While $\eta_m = X_{\eta_c} + (1 - X)\eta_P$ is the viscosity of mixture of the blood. Other symbols have their usual meanings. We have transformed in cylindrical form equation (4.1) and (4.2) the blood flow in artery is symmetric w.r.t. axis. Hence $v_\theta = 0, v_z, v_r and\ P$ do not depend upon θ since only one component of velocity which is along the axis is effective. We have $v_\theta = 0, v_r = 0, v_z = v$ since, flow is steady, and we have [32]

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

Keeping in view these facts, we obtain the following result. Equation of continuity reduces to

$$\frac{\partial v_z}{\partial t} = 0$$
$$\Rightarrow v_z = v(r) \tag{4.3}$$

The r-component of equation of motion reduces to

$$\rho_m(0) = -\frac{\partial p}{\partial r} + \eta_m(0)$$
$$\Rightarrow \frac{\partial p}{\partial r} = 0$$
$$\Rightarrow p = p(z) \tag{4.4}$$

θ- Component of equation of motion reduces to

$$\rho_m(0) = 0 + \eta_m(0)$$
$$\Rightarrow 0 = 0 \tag{4.5}$$

Similarly, the Z-component of equation of motion reduces to

$$\rho_m v_z = \frac{\partial v_z}{\partial t} = -\frac{\partial p}{\partial z} + \eta_m \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial v_z}{\partial r} \right) + \frac{\partial^2 v_z}{\partial z^2} \right] \tag{4.6}$$

Similarly, the Z-component of equation of motion reduces to

$$0 = -\frac{\partial p}{\partial z} + \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial v(r)}{\partial r} \right) \right] \tag{4.7}$$

Whereas, the equation (3.4) expresses the fact that the pressure p depends only on z. We also retain the fact that pressure gradient $-\frac{\partial p}{\partial z}$ in the arteries remote from the heart is constant, say p then the equation (3.7) takes the following form$0 = p + \eta_m \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial v(r)}{\partial r} \right) \right]$ (4.8)

Integrating the equation (4.8), we get,

$$r\frac{dv}{dr} = \frac{pr^2}{2\eta_m} + A \quad (4.9)$$

Where A be the constant of integration. Apply the first boundary condition on the equation (4.9), we get A=0.Hence equation (4.9) reduces to $r\frac{dv}{dr} = \frac{pr^2}{2\eta_m}$ (4.10)

Again integrating the equation (4.10), we get,

$$v = -\frac{pr^2}{4\eta_m} + B \quad (4.11)$$

Again using second boundary condition on the equation (4.11), we can evaluate the integration constant as follows: $B = \frac{PR^2}{4\eta_m}$

Inserting the value of B in the equation (4.11), we obtain the velocity of blood flow in the arteries as follows:

$$v = \frac{p}{4\eta_m}(R^2 - r^2) \quad (4.12)$$

Result (Bio-Physical Interpretation)

Observations: Hematocrit Vs. blood pressure is taken from Gastro liver research Institute Kanpur (UP)
Diagnosis- Dr. Bijendra Singh

Table 1

Date	HB in gm/dl	Systolic Blood pressure in mmhg	Hematocrit	Blood Pressure in Pascal
24-12-2016	9.2	100	27.6	13332.2
26-12-2016	9.8	110	29.4	14665.42
27-12-2016	10.5	115	31.5	15332.32
30-12-2016	10	105	30	13998.81
31-12-16	10.2	112	30.6	14932.06

According to Berkow, Robert, The hematocrit (expressed as percentage points) is normally about three Times the hemoglobin concentration (reported as grams per deciliter) [33]. The Flow flux of blood in Arteries is given bellow:

$$Q = \int_0^R 2\pi r v dr = \int_0^R \frac{P}{4\eta_m}(R^2 - r^2)\, 2\pi r dr$$
$$= \frac{P}{4\eta_m}\left[\pi R^2 r^2 - \frac{\pi r^4}{2}\right]_0^R$$
$$= \frac{\pi r^4 P}{8\eta_m} (5.1)$$
$$= \frac{\pi R^4}{8\eta_m}\left[-\frac{dP}{dz}\right]$$
$$\int_{z_i}^{z_f} Q\, dz = -\int_{p_i}^{p_f} \frac{\pi R^4}{8\eta_m} dp$$
$$Q\left[z_f - z_i\right] = \frac{\pi R^4}{8\eta_m}\left[p_i - p_f\right]$$
$$\left[p_i - p_f\right] = \frac{8\eta_m}{\pi R^4}\, Q\left[z_f - z_i\right] (5.2)$$ Where $\left[z_f - z_i\right]$ Length of arteries

Average (H) =29.82 gm. /dl
Average (BP) = 12052.162 Pascal
Average length of arteries =0.05 [34],
$\eta_m = 0.035\ Pa.s. and\ \eta_p = 0.0015\ Pa.s.$ [35]
Since, we know that
$\eta_m = X_{\eta_c} + (1 - X)\eta_P$ (5.3) $\eta_c = 0.11384$

From equation (5.3)

$$\eta_m = (0.11384 - 0.0015)\frac{H}{100} + 0.0015$$
$$\eta_m = 0.00112H + 0.0015$$

From equation (5.2)

$$p_i - p_f = \frac{8 \times 0.01833 \times 0.05}{3.14 \times (2.5 \times 10^{-3})^4}\eta_m$$
$$p_i - p_f = \frac{7.332 \times 10^{-3}}{1.227 \times 10^{-10}}\eta_m$$
$$p_i - p_f = 59755501.22(0.00112H + 0.0015)$$

$p_i - p_f = 66926.16H + 89633.25$

We get, values of blood pressure drop if hematocrit known by using above equation.
(Relation between Blood pressure drop and hematocrit)

Table 2

H (Hematocrit) (g/dl)	27.6	29.4	31.5	30	30.6
P (Blood Pressure drop) (Pascal)	1936795.266	2057262.354	2197802.25	2097418.05	2137573.746

Table 2 shows change in the blood pressure drop with increase in hemoglobin. The graph of above table is given in figure-6.

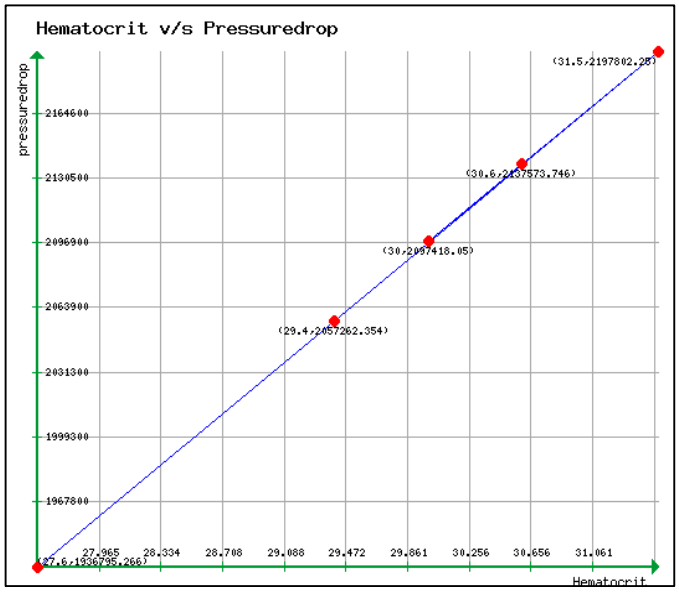


Fig 6

Conclusion

A simple survey of the graph between blood pressure drop and hematocrit in figure 6 shows that when hematocrit increased then blood pressure also increased. That is Hematocrit proportional to Systolic blood pressure.

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