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A mathematical model on the two –Phase hepatic blood flow in arterioles with special reference 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 arterioles 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 sub-system, Liver etc

Introduction

Structure and Function of Liver

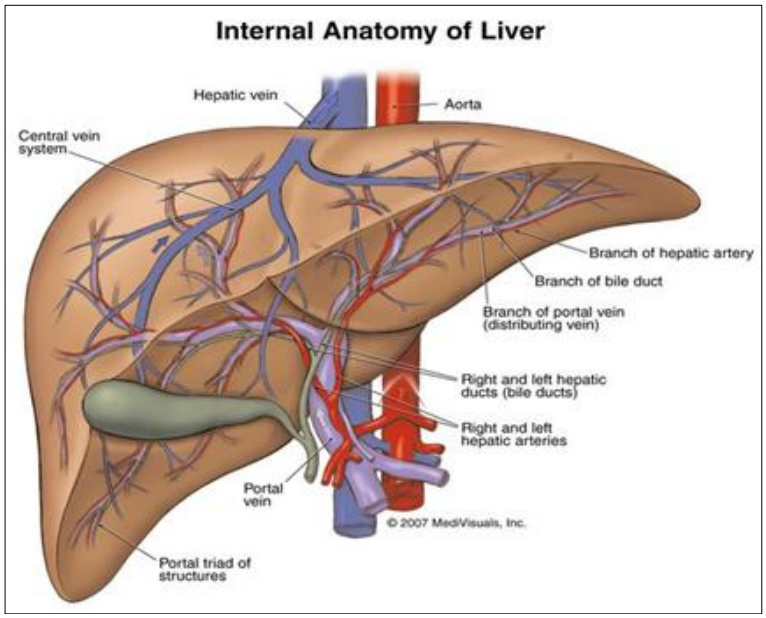
Liver is a vital organ of human's body. The human liver in adults weighs between 1.4-1.6 kilograms (3.1-3.6 pounds). It is a soft, pinkish-brown, triangular organ. It is both the largest internal organ and the largest gland in the human body. It is located in upper right quadrant of Abdomen. The liver can be divided into functional units called lobules. The liver has 2 main lobes: the larger right lobe and smaller left lob. Each lobe is divided into segments. The lobes are separated by a band of tissue called the falciform ligament, which help attach the liver to the diaphragm. Liver lobes are surrounded by a thick capsule, mostly overlaid with reflected periforinum [2].The liver has wide range of functions generally cite it being around 500. Liver plays an important role in human body like decomposition of red blood cells, protein production, blood clotting to cholesterol, produce bile which helps indigestion. Many bio chemical reactions occur in liver like synthesis and breakdown of small and complex molecules

The liver has most complicated circulation of any organ. It receives blood from two main sources: approximately 80% is partially deoxygenated blood from the hepatic portal vein, which collects all the blood from that leaves different tissues and the remainder is fully oxygenated blood supplied by the hepatic artery [1]. The blood in the hepatic portal vein is rich in nutrients that are used by the liver, and regulation of the blood flow is achieved by control of hepatic artery originating from the celiac artery. Total hepatic blood flow range 800-1200 ml/min, which is equivalent to approximately 100 ml/min per 100g of liver weight [3]. Liver volume and Portal blood flow decreases after the age of 50 [4].

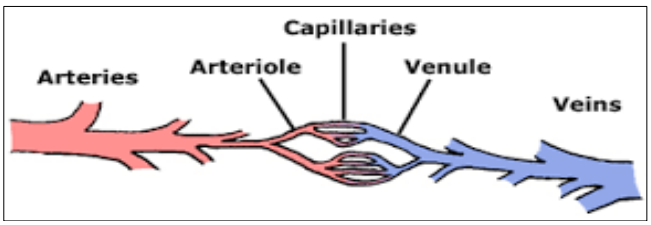
Structure and Function of Hepatic arterioles

An arteriole is a small diameter blood vessel in the microcirculation that extends and branches out from an artery and leads to capillaries [5]. Arterioles are very small arteries that deliver blood to capillaries. As arterioles branch off an artery, they have smooth muscle and a few elastic fibers in the tunica media. These gradually taper away as the arteriole becomes smaller, leaving mostly the endothelium and a few smooth muscle fibers by the time the arteriole connects to the capillaries Arterioles play a key role in regulating blood flow into capillaries. Vasoconstriction of arterioles decreases blood flow into capillaries; vasodilatation

increases flow. A change in the diameter of a large number of arterioles at once will also affect blood pressure. This work is important for human health. There are several researches, who examined the blood flow in the artery and veins. This work will focus on two phase hepatic blood flow in arterioles with special reference to Liver cirrhosis.



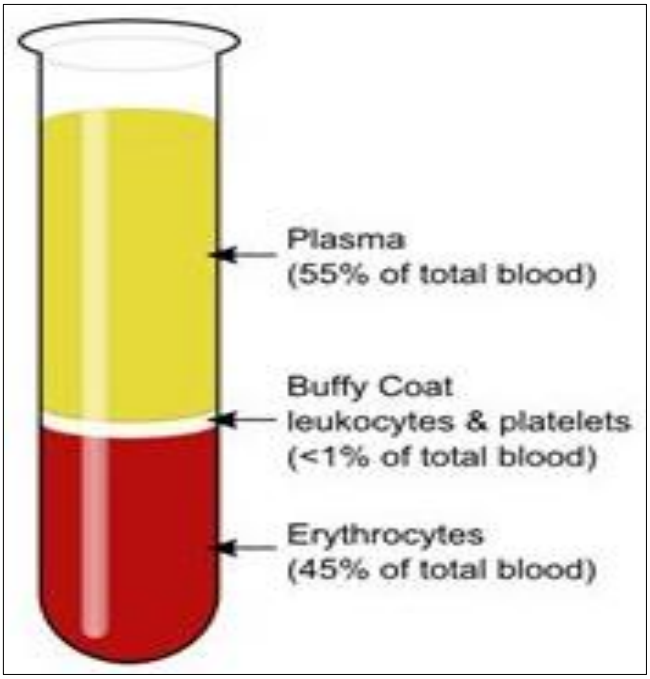
A lot of work is available, but P. N. Pandey and V. Upadhyay (2001) discussed a some phenomenon in two phase blood flow gave an idea on the two phase hepatic blood flow in arterioles with a Liver disease of Liver cirrhosis. The work of P.N. Pandey and V. Upadhyay in whole circulatory system but this work will 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 model. We present an improvement on the previous work in the field and this is discussed separately below. The ultimate use of this model is to predict normal reference levels of two phase blood flow in arterioles for individual patients undergoing disease of Liver cirrhosis.



Typical sub-circulatory system

Constitution of blood

Blood is a concentrated suspension of formed elements in an aqueous solution called plasma. Plasma is a saline solution consisting of 90% water by weight, 7% proteins which include fibrinogen, globulin and albumin. The function of albumins and globulin is to maintain the osmotic balance, and there by, control the movement of water between blood and various tissues. Fibrinogen plays an important role in the clotting mechanism of blood. The formed elements, called as hematocrit, are essentially red blood cells (erythrocytes), white blood cells (leucocytes) and platelets (thrombocytes). The red cells dominate the particulate matter in blood, occupying on the average about 40 - 45% of the whole blood.

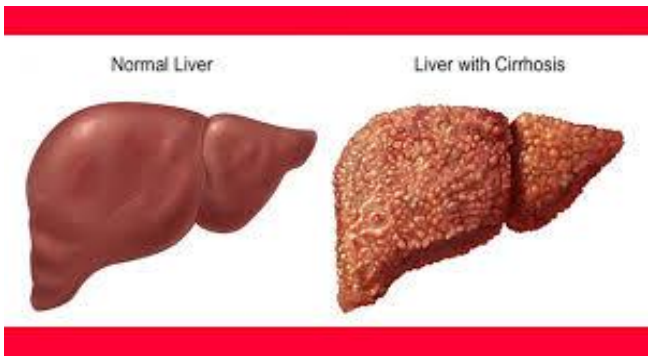


The primary function of red cells is to transport oxygen from lungs to all the tissues of body and the removal of carbon dioxide formed during metabolic process [6]. Whole blood (plasma and cells) exhibits non- Newtonian fluid dynamics. The percentage of volume covered by blood cells in the whole blood 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 [7]. 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 [8]. 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.

Description of Disease

Liver is also prone to many diseases [9] 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 [10]. 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) [11].

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-$\boldsymbol{\tau = \eta e^n}$ Where,$\boldsymbol{\eta}$ is the viscosity of coefficient [12]. 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 $\boldsymbol{P_i}$ represents the pressure at the origin of the vessels, at the other end point pressure is $\boldsymbol{P_f}$. Then the pressure difference is represented by $\boldsymbol{P_i - P_f}$ blood pressure of the first end point is greater than the blood pressure of other end point

, that is $\boldsymbol{P_i > P_f}$

$$\Delta \boldsymbol{P} = -\left(\boldsymbol{P_f - P_i}\right)$$

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 [13]. According to the Sherman I.W. and Sherman V.G. blood is mixed fluid [14]. 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 [14].

3.1 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 [15]. 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

$$\boldsymbol{r} = \frac{X_{\rho_c}}{(1-X)\boldsymbol{\rho_p}} \quad (1)$$

Where $\boldsymbol{\rho_c}$ and $\boldsymbol{\rho_p}$ 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

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

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

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

$$\frac{1+r}{\rho_m} = \frac{r}{\rho_c} + \frac{1}{\rho_p}$$ [16] (4)

Then equation (2) and (3) can be combined together as follow

$$\frac{\partial \rho_m}{\partial t} + (\rho_m v^i)_{,i} = 0 \quad (5)$$ Where $\rho_m = X\rho_c + (1-X)\rho_p$

3.2 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) [17]. 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:

$$X\rho_c \frac{\partial v^i}{\partial t} + (X\rho_c v^i)v^i_{,j} = -X_{P,j}\mathrm{g}^{ij} + X_{\eta_c}\left(\mathrm{g}^{jk}v^l_{,k}\right)_{,j} \quad (6)$$

The equation of motion for plasma will be as follows:

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

Now adding equation (6) and (7) and using relation (4), the equation of motion for blood flow with the both phases will be as follows:

$$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j)v^i_{,j} = -P_{,j}\mathrm{g}^{ij} + \eta_m\left(\mathrm{g}^{jk}v^i_{,k}\right)_{,j} \quad (8)$$

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

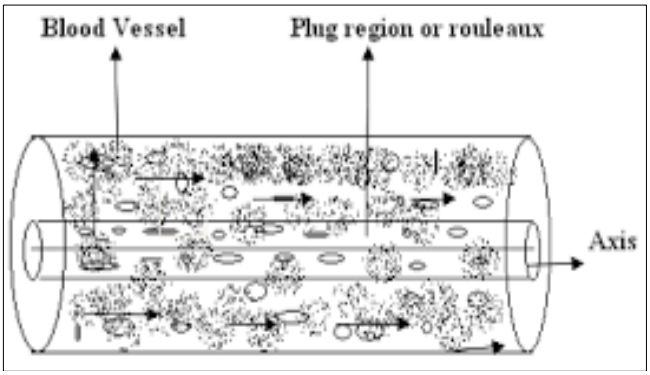
As the velocity of Blood flow decreases, the viscosity of blood increases. The velocity of blood deceases successively because of the fact that arterioles, veinules and veins these vessels are relatively a far enough from the heart. Hence the pumping of the heart on these vessels is relatively low [18]. 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 [19].

The Herschel Bulkley law holds good on the two phase blood flow through veins arterioles, veinules and whose constitutive equation is as follows: $T^* = \eta_m e^n + T_P\ (T^* \geq T_p)\ and\ e = 0\ (T^* < T_p)\ where\ T_p$ is yield stress.

When strain rate. $e = 0\ (T^* < T_p)$ a core region is formed which flow just like a plug. Let the radius of the plug be r_p. the stress acting on the surface of plug will be T_p. equating the forces on the plug, we get

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

$$\Longrightarrow r_p = 2\frac{T_p}{P} \quad (9)$$



Herschel Bulkley blood flow modal

The Constitutive equation for test part of 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:

$$T^{ij} = -P\mathrm{g}^{ij} + T_e^{ij}$$

Where $T_e^{ij} = \eta_m(e^{ij})^n$ While $e^{ij} = \mathrm{g}^{jk}V_k^i$

Where the symbols have their usual meanings.

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

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

Equation of motion - $\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j)v^i_{,j} = -T^{ij}_{e,j}$ (10)

Where all the symbols have their usual meaning.

Mathematical Modeling

Since the blood vessels are cylindrical; he above governing equations have to transform 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 as follows: $[g_{ij}] = \begin{bmatrix} 1 & 0 & 0 \\ 0 & r^2 & 0 \\ 0 & 0 & 1 \end{bmatrix}$ while matrix of

Conjugate metric tensor is as follows:

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

Whereas the chritoffel's symbols of 2nd kind are as follows: $\begin{Bmatrix} 1 \\ 2 \ 2 \end{Bmatrix} = -r, \begin{Bmatrix} 1 \\ 2 \ 2 \end{Bmatrix} = \begin{Bmatrix} 1 \\ 2 \ 2 \end{Bmatrix} = \frac{1}{r}$ Remaining others are zero.

Relation between contravarient and physical components of velocity of blood flow will be as follows:

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

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

$\sqrt{g_{33}v^3} = v_z \Longrightarrow v_z = v^3$

Again the physical components of $-P_{,j}g^{ij}$ are $-\sqrt{g_{ij}}P_{,j}g^{ij}$

Equation (9) and (10) are transformed into cylindrical form so as to solve them as power law modal to get

$$\frac{dv}{dr} = \left(\frac{pr}{2\eta_m}\right)^{\frac{1}{n}} \qquad (11)$$

Where, pressure gradient $\frac{dp}{dz} = P$

Replace $r\ to\ r - r_p$ for non-plug region

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

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

From equation no (9)

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

Substituting the value of from (7) into (11), we get

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

$$\frac{dv}{dr} = -\left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}}(r - r_p)^{\frac{1}{n}} \qquad (12)$$

Integrating above equation (12) under the no slip boundary condition: $v = 0\ at\ r = R$ so as we get:

$$v = -\left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} \frac{n}{n+1}\left[(R - r_p)^{\frac{1}{n}+1} - (r - r_p)^{\frac{1}{n}+1}\right] \qquad (13)$$

This is formula for velocity of blood flow in arterioles, veinules and veins.

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

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

Where the value $r_p$of is taken from (7).

Result (Bio-Physical Interpretation)

Observations:

Hematocrit vs. Blood pressure is taken from Gastro liver research Institute Kanpur UP

By Dr. Bijendra Singh

Patient Name: Mrs Chandra Prabha Age: 58 yr.

Diagnosis- Liver Cirrhosis

Hemoglobin in gm. /dl and blood pressure in mmhg

Date	Hemoglobin gm./dl	B.P. mmhg	Hematocrit
24-12-16	9	100/60	27
26-12-16	9.8	110/80	29.4
27-12-16	10.5	115/60	31.5
30-12-16	10	105/70	30
31-12-16	10.2	112/60	30.6

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

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

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

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

Using (12) and (14)

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

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

Now let $R = 1, and\ r_p = \frac{1}{3}\ and\ we\ get$

$$Q = \frac{\pi n}{(n+1)}\left[\frac{p}{2\eta_m}.\frac{2}{3}\right]^{\frac{1}{n}}\frac{2}{27}\left[\frac{26n^2+33n+9}{6n^2+5n+1}\right]$$

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

Now $Q = 1000\ ml/\min \Longrightarrow 0.016661\ lit/sec$
According to Gustafson, Daniel R. (1980)
$\eta_p = 0.0015\ (pascal - sec)$
$\eta_m = 0.035\ (pascal - sec)$ According to Glenn Elert (2010)
H= 30.0 and $P_i - P_f$=4666.27 Pascal
Then

$$\frac{27*0.01666}{2*3.14} = \left(\frac{4666.27}{3*0.035}\right)^{\frac{1}{n}}\left[\frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}\right]$$

$$0.071627 = (44440.6667)^{\frac{1}{n}}\left[\frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}\right]$$

Solve by numerical method, we get

$$n = -2.439886969$$

Terminal hepatic arterioles length $= 50\ \mu m$[20].

$$\Longrightarrow\ z_f - z_i = 0.00005\ meter$$

By using relation $\eta_m = X\eta_c + (1-X)\eta_P$ where $X = \frac{H}{100}$, we get
$\eta_c = 0.113167$ And again using same above relation $\eta_m = 0.001117H + 0.0015$
Now let $A = \frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}$

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

$$P = -\frac{dP}{dZ} \Longrightarrow dP = -PdZ$$

$$\Longrightarrow -\int_{p_f}^{p_i} dP = -\int_{Z_i}^{Z_f} 3\eta_m\left(\frac{27Q}{2\pi A}\right)^n dZ$$

Where $P_i - P_f$ pressure drop and
$Z_f - Z_i$ Length of hepatic arterioles

$$P_i - P_f = 3\eta_m\left(\frac{27Q}{2\pi A}\right)^n\ (Z_f - Z_i)$$

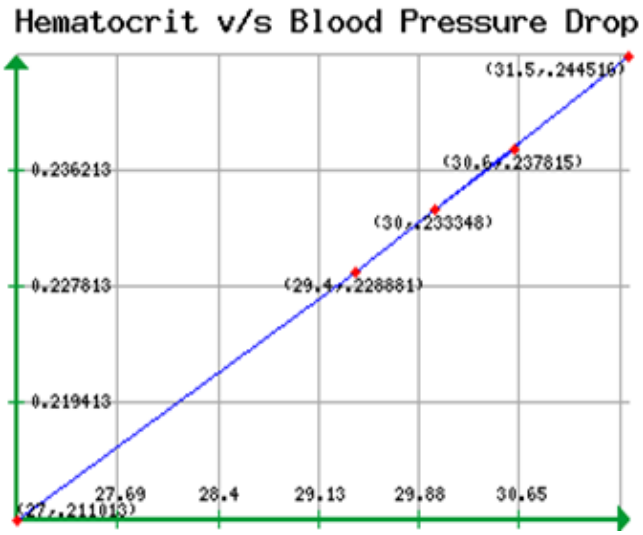
Substituting the value of $Q, \eta_m, Z_f - Z_i, and\ n$, we get

$$P_i - P_f = 0.007445H + 0.009998$$

We get, values of blood pressure drop if hematocrit is known by using above relation.

Relation between blood pressure drop and Hematocrit

Hematocrit (gm./dl)	Pressure Drop(Pascal)
27.0	0.211013
29.4	0.228881
31.5	0.244516
30.0	0.233348
30.6	0.237815



Conclusion

A simple survey of the graph between hematocrit and blood Pressure-drop in Liver Cirrhosis patient shows that a straight line. Which shows that hematocrit is directly proportional to blood pressure-drop.

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