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A mathematical modeling of two phase hepatic mean blood flow in arterioles during liver cirrhosis

Anil Kumar, V Upadhyay, AK Agrawal and PN Pandey

Abstract

Present paper visualize a model of two phased mean blood flow in hepatic arterioles remote from the heart and proximate to the Liver, Keeping in view the nature of hepatic blood circulation in human body. The viscosity increases in the arterioles due to formation of rouleaux along axis of red blood cells. We have applied the Herschel Bulkley Non-Newtonian model in Bio- fluid mechanical setup with the help of clinical data in case of Liver Cirrhosis for hemoglobin versus blood pressure. The overall presentation is in tensorial form and the solution technique adopted is analytical as well as numerical. The role of hematocrit is explicit in the determination of blood pressure in case of Hepatic disease Liver Cirrhosis the graphical presentation for particular parametric value is much close to the clinical observation.

Keywords: Structure of the Liver, Hematocrit, Liver Cirrhosis, Hepatic Blood Flow, Herschel Bulkley Non-Newtonian model etc

Introduction

Structure and function of the Liver

The liver is the largest organ of the human body, weighs approximately 1500 g, and is located in the upper right corner of the abdomen. The organ is closely associated with the small intestine, processing the nutrient-enriched venous blood that leaves the digestive tract. The liver performs over 500 metabolic functions, resulting in synthesis of products that are released into the blood stream (e.g. glucose derived from glycogenesis, plasma proteins, clotting factors and urea), or that are excreted to the intestinal tract (bile). Also, several products are stored in liver parenchyma (e.g. glycogen, fat and fat soluble vitamins). Almost all blood that enters the liver via the portal tract originates from the gastrointestinal tract as well as from the spleen, pancreas and gallbladder. A second blood supply to the liver comes from the hepatic artery, branching directly from the celiac trunc and descending aorta. The portal vein supplies venous blood under low pressure conditions to the liver, while the hepatic artery supplies high-pressured

Arterial blood. Since the capillary bed of the gastrointestinal tract already extracts most O_2 , portal venous blood has a low O_2 content. Blood from the hepatic artery on the other hand, originates directly from the aorta and is, therefore, saturated with O_2 . Blood from both vessels joins in the capillary bed of the liver and leaves via central veins to the inferior caval vein.

Total human liver blood flow represents approximately 25% of the cardiac output, up to 1500 ml/min. Hepatic flow is sub divided in 25-30% for the hepatic artery (500 ml/min) and the major part for the portal vein (1000 ml/min). The hepatic artery also plays an important role in liver blood vessel wall and connective tissue perfusion. It also secures bile duct integrity. Blood from the hepatic artery and the portal vein joins in the sinusoids. However, recent studies by others as well as our own observations, have revealed that there are both common and separate channels for arterial and portal blood. The hepatic artery perfuses the liver vascular bed in a 'spotty' pattern, while the portal vein perfuses the liver uniformly. The liver is able to regulate mainly arterial flow by means of so-called sphincters, situated at the in- and outlets of the sinusoids

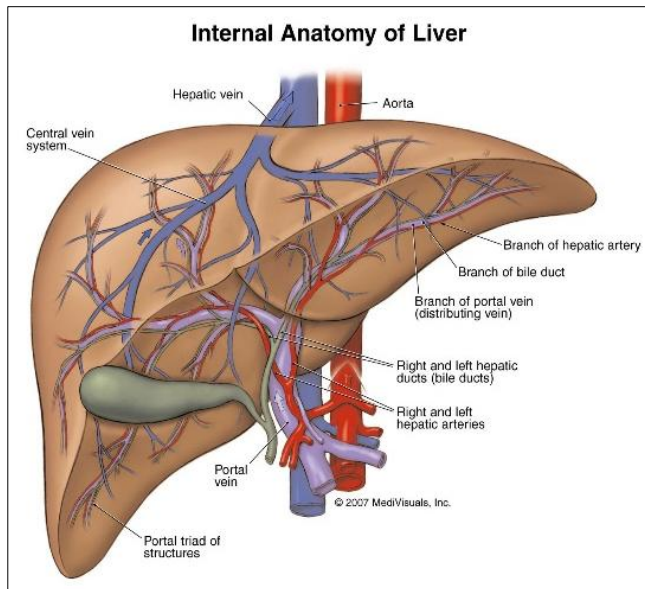


Fig 1

Structure and Functions of the Hepatic Arterioles

Blood is carried through the body via blood vessels. An artery is a blood vessel that carries blood away from the heart, where it branches into ever-smaller vessels. Eventually, the smallest arteries, vessels called arterioles, further branch into tiny capillaries, where nutrients and wastes are exchanged, and then combine with other vessels that exit capillaries to form venules, small blood vessels that carry blood to a vein, a larger blood vessel that returns blood to the heart.

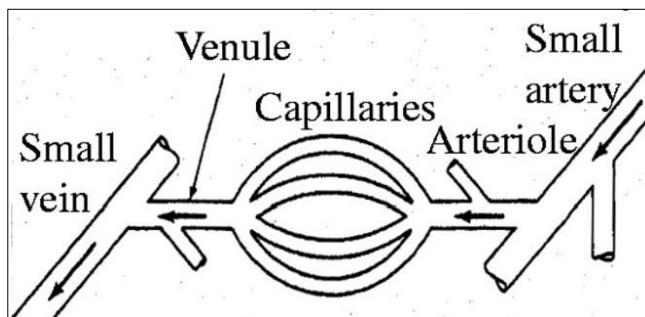


Fig 2

An arteriole is a very small artery that leads to a capillary. Arterioles have the same three tunics as the larger vessels, but the thickness of each is greatly diminished. The critical endothelial lining of the tunica intima is intact. The tunica media is restricted to one or two smooth muscle cell layers in thickness. The tunica externa remains but is very thin. With a lumen averaging 30 micrometers or less in diameter, arterioles are critical in slowing down—or resisting—blood flow and, thus, causing a substantial drop in blood pressure. Because of this, you may see them referred to as resistance vessels. The importance of the arterioles is that they will be the primary site of both resistance and regulation of blood pressure. The precise diameter of the lumen of an arteriole at any given moment is determined by vasodilation (the term vasodilation refers to the dilation or relaxation of the arterioles to allow more blood to an area) in the arterioles are the primary mechanisms for distribution of blood flow.

An arteriole lowers the resistance and results in an increase in flow through that particular arterioles. (Varun Mohan *et al.*, 2012) [1]. Arterioles are the blood vessels in the arterial side of the vascular tree that are located proximal to the capillaries and, in conjunction with the terminal arteries, provide the majority of resistance to blood flow. Consequently, arterioles are important contributors to the regulation of mean arterial pressure and tissue perfusion. Their wall consists of cellular and extracellular components that have been traditionally classified as conforming three layers: an intima containing endothelial cells sited on a basement membrane; a media made of an internal elastic lamina opposed by one or two layers of smooth muscle; and an adventitia composed mostly of collagen bundles, nerve endings and some fibroblasts. These components of the wall are dynamically interconnected, providing a level of plasticity to the arteriolar wall that blurs the traditional boundaries of a rigid layered classification (LuisA. Martinez - Lemus, 2011) [2]. We have visualize mathematical modeling with the help of two phase Herschal Bulkley Non Newtonian model we explain the blood flow in various arterioles in the human heart. But present work will focus on Hepatic circulatory system, hepatic circulatory system is a sub system of whole circulatory system, (Srivastava Manoj *et al.*, 2012) [3]. On the basis of previous work in present study be achieve new findings which are discussed separately below.

Constitution of Blood

Blood is a circulating tissue composed of fluid plasma and cells (red blood cells, white blood cells, platelets). Blood is the means and transport system of the body used in carrying elements (e.g. nutrition, waste, heat) from one location in the body to another, by way of blood vessels. Blood consists of many components (constituents). These include 55% plasma and 45% components 'Blood Cells' 99% are erythrocytes (red blood cells) and 1% are leucocytes (white blood cells) and thrombocytes (blood platelets).

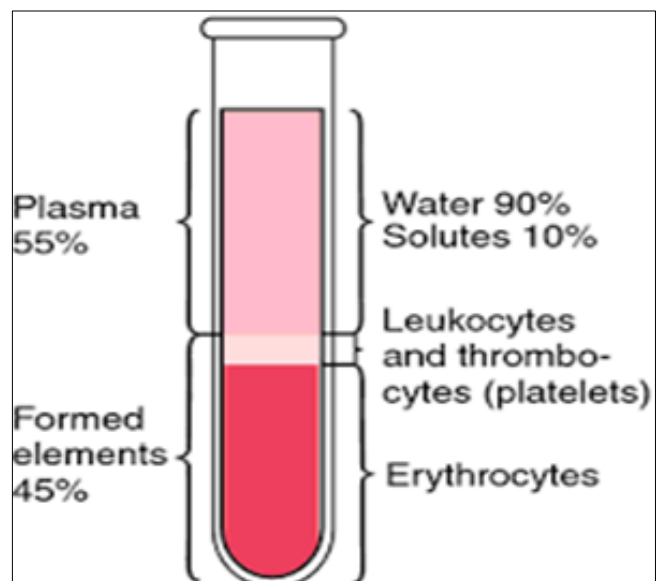


Fig 3

The human blood approximately 40 to 45% by volume of the normal human blood and more than 99% of all blood cells. The ratio between red blood cells volume to total volume of blood is called hematocrit. Thrombocytes are a

vital component of the blood clotting mechanism. The total volume concentration of leukocytes and thrombocytes is only about 1% (N. Bessonov *et al.*, 2016) [4]. Then we have considered only two phases of blood. Which is one of red blood cells and other phase is plasma.

Description of disease

Cirrhosis is a complication of many liver diseases that is characterized by abnormal structure and function of the liver. The diseases that lead to cirrhosis do so because they injure and kill liver cells and the inflammation and repair that is associated with the dying liver cells causes scar tissue to form. The liver cells that do not die multiply in an attempt to replace the cells that have died. This results in clusters of newly-formed liver cells (regenerative nodules) within the scar tissue. Cirrhosis is considered to be the end stage of chronic hepatopathies which often leads to hepatocellular carcinoma. The diagnosis of the disease is best achieved by looking at the granular structure of the liver parenchyma and the aspects of the liver surface such as its unevenness and its contour as shown in Fig.4

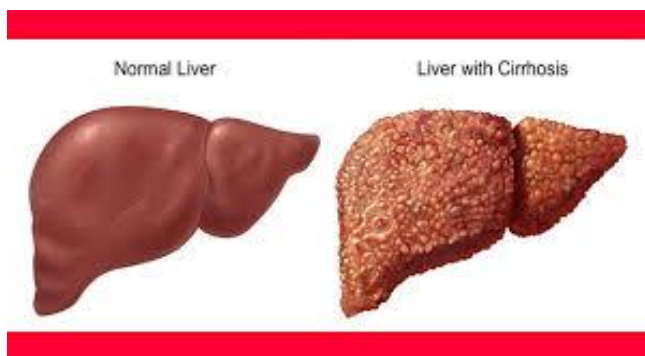


Fig 4

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. 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: $n = 155,000$) [5].

Real Modal

Choice of frame of reference

We have to selected a frame of reference for mathematical modeling of the state of a moving blood· keeping in view the difficulty and generality of the problem of blood flow, we select generalized three-dimensional orthogonal curvilinear co-ordinate system, briefly prescribed as E_3 , called as 3-dim Euclidean space, We interpret the quantities related to blood flow in tensorial form which is comparatively more realistic, The biophysical laws thus expressed fully hold good in any co-ordinate system, which is compulsion for the truthfulness of the law (Mishra *et al.*, 1965) [6]. Now, let the co-ordinate axes be OX_i , O denotes origin and subscript $i = 1, 2, 3$. Let X_i be the co-ordinates of any point P in space, The mathematical description of the state if a moving blood is affected by means of functions which give the distribution of the blood velocity $v_k = v_k(X_i, t)$, $k = 1, 2, 3$ and of any two thermodynamic quantities pertaining to the blood, for instance the pressure $p_i = p_i(X_i, t)$ and the density $\rho = \rho(X_i, t)$, As is well known, all the thermodynamic quantities are determined by the values of any two of them, together with the equate of state, Hence, if we are given five quantities, namely the three components of velocity v_k , the pressure p and the density ρ , the state of moving blood is completely determined· All these quantities are, in general, functions of the co-ordinates X_i , $i = 1, 2, 3$ and of the time t , We emphasize that $v_k(X_i, t)$, is the velocity of the blood at a given point X_i in space and at a given t , i.e. it refers to fixed points in space and not to fixed particles of the blood; in the course of time, the latter move about in space, The same remarks apply to p and ρ Blood is a mixed fluid, (Chandra, H. *et al.*, 2014) [7] The first phase is red blood cells, and other phase is plasma, while the other phase is that of blood cells· The blood cells are enclosed with a semi-permeable membrane whose density is greater than that of plasma, these blood cells are uniformly distributed in plasma, Thus blood can be considered as a homogeneous mixture of two-phases (Sherman *et al.* 1989) [8].

2.2 Choice of parameters

Blood is the Non- Newtonian fluids, then using this constitutive equation for fluids.

$$\tau = \eta e^n$$

If $n = 1$ then the nature of fluid is Newtonian and if $n \neq 1$ then the nature of fluid is Non-Newtonian fluids. Where τ is denoted by stress, e is denoted by strain rate this constitutive equation is called Herschel - Bulkily Non Newtonian law, and n is denoted by the parameter, these equation uses equation of motion. In present study there are five parameter are used but three parameter are frequently used namely velocity v , pressure p and density ρ (manoj Srivastava *et al.*, 2012) [3]

2.3 Choice of constitutive equation:

We have using in two phase blood flow through arterioles and whose constitutive equation is as follows:

$$T^* = \eta_m e^n + T_p \quad (T^* \geq T_p)$$

where T_p is yield stress

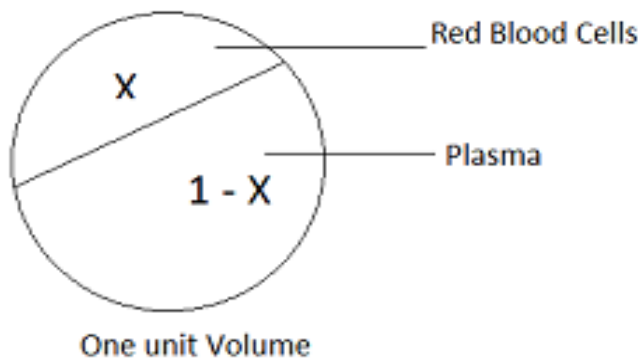
When strain rate. $e = 0 (T^* < T_p)$ A core region is formed which flow just like a plug. (Upadhyay, 2000) [9].

2.4 Constitution of two phase blood volume:

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 [10]. 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.

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

Where ρ_c and ρ_p are densities of blood cells and blood plasma respectively. usually this mass ratio is not a constant, even then this may be supposed to constant in present context.



Mathematical Model / Formulation

We have recommended that blood flow in vessels is a peristaltic transport system because they thought blood is having two layers of fluid while in the peripheral reasons of vessels blood flow is a Newtonian phenomenon. Blood is in the liquid form and it is non-Newtonian. Though blood is not an ideal fluid, even to develop the equation of motion. We start with a model of ideal fluid. The second important principle of fluid dynamics is that of conservation of momentum. The equation of motion is based on this principle.

According to this principle, the total momentum of any fluid system is conserved in absence of external force.

$$\frac{dp}{dt} + P - F_{v(viscosity)} = 0 \text{ (External force)}$$

The blood can be considered as homogeneous mixtures of two phases. We derive the fundamental equation of continuity, which is a mathematical expression of principal of conservation of matter.

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 [10]. 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

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

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. 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

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

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

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 $[(1-X)\rho_p v^i]_{,i}$ 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 ρ_m as follows

$$\frac{1+r}{\rho_m} = \frac{r}{\rho_c} + \frac{1}{\rho_p} \tag{4}$$

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

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

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) [12]. 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:

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

The equation of motion for plasma will be as follows:

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

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:

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

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 decreases 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 [9]. 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 [13].

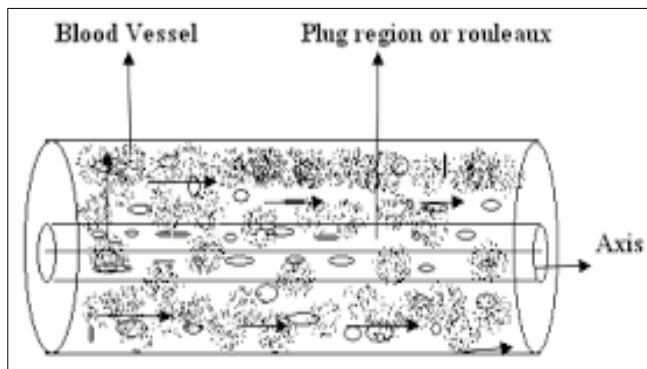
The Herschel Bulkley law holds good on the two phase blood flow through veins arterioles, veinules and whose constitutive equation is as follows: $T^* = \eta_m e^n + T_p$ ($T^* \geq T_p$) and

$e = 0$ ($T^* < T_p$) 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

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

$$\Rightarrow r_p = 2 \frac{T_p}{P} \tag{9}$$



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

and generalized form will be as follows.

$$T^{ij} = -Pg^{ij} + T_e^{ij}$$

Where $T_e^{ij} = \eta_m (e^{ij})^n$ 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:

Equation of continuity-

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

Equation of motion-

$$\rho_m \frac{\partial v^i}{\partial t} + (\rho_m v^j) v_{,j}^i = -T_{e,j}^{ij} \tag{10}$$

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:

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

zero.

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

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

$$\sqrt{g_{22}} v^2 = v_\theta \Rightarrow v_\theta = r v^2$$

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

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

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

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

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

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

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

From equation no $\tag{9}$

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

Substituting the value of $\frac{dp}{dz}$ from (7) into (11), we get

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

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

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

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

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:

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

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

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

Using (12) and (14)

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

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

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

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

Now $Q=1000 \text{ ml/min}$ [$\Rightarrow 0.016661 \text{ lit/sec}$]

The flow flux of two phased blood flow in arterioles is

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

$\eta_m = 0.035$ (pascal – sec) According to Glenn Elert (2010)

According to Gustafson, Daniel R. (1980)

$\eta_p = 0.0015$ (pascal – sec)

$\eta_m = 0.035$ (pascal – sec) According to Glenn Elert (2010)

$H= 27.6$ and $P_i - P_f=4437.7333$ Pascal second

Then

Result (Bio-Physical Interpretation)

Observations: Hematocrit vs. Blood pressure is taken from

Gastro liver Hospital swaroop nagar Kanpur UP

By Dr. V.K. Mishra.

Patient Name: Mrs. Shiv Devi Age: 62 yr.

Diagnosis- Liver Cirrhosis

Hemoglobin in gm. /dl and blood pressure in mmhg

Date	B.P. mmhg	Hemoglobin
10-01-2017	100/60	9.2
11-01-2017	110/60	9.4
13-01-2017	120/60	9.8
15-01-2017	130/70	10.5
17-01-2017	125/60	10.0

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

$$\frac{27 * 0.01666}{2 * 3.14} = \left(\frac{4437.7333}{3 * 0.035} \right)^{\frac{1}{n}} \left[\frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1} \right]$$

$$0.071627 = (42264.1267)^{\frac{1}{n}} \left[\frac{26n^3 + 33n^2 + 9n}{6n^3 + 11n^2 + 6n + 1} \right]$$

Solve by numerical method, we get

$$n = -2.427414$$

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

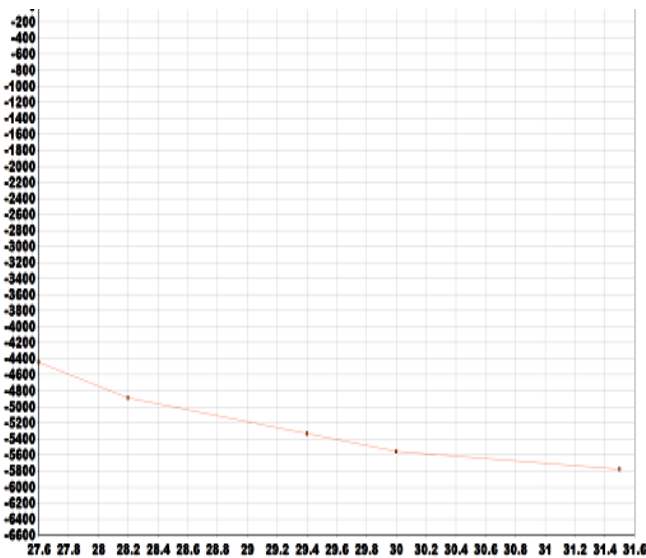
$$\Rightarrow z_f - z_i = 0.00005 \text{ meter}$$

By using relation $\eta_m = X\eta_c + (1 - X)\eta_p$ where $X = \frac{H}{100}$, we get

$\eta_c = 0.1228768$ And again using same above relation

$$\eta_m = 0.0012138H + 0.0015$$

$$\text{Now let } A = \frac{26n^3+33n^2+9n}{6n^3+11n^2+6n+1}$$



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

$$P = -\frac{dP}{dz} \Rightarrow dP = -PdZ$$

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

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

$$P_i - P_f = 0.00769H + 0.003169$$

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)
27.6	-4444.9554
28.2	-4888.9177
29.4	-5332.8800
31.5	-5778.1754
30	-5555.5200

Conclusion

A simple investigation of the graph 27.6 to 28.2 down convex and 28.2 to above graph straight line between blood pressure drop and hematocrit in suffering liver cirrhosis patient and confirmations that when hematocrit increase then blood pressure drop also increased. That is hematocrit inversely proportional to blood pressure drop.

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