

THE PHARMA INNOVATION

A mathematical model for two phase hepatic blood flow in artery with special reference to hepatitis B

V. Upadhyay*, Om Prakash, P.N. Pandey

Submitted 10.01.2012. Accepted for publication 12.02.2012.

In this paper, we have presented a model of two phased arterial hepatic blood flow in Hepatic arteries remote from the heart and proximate to the Liver keeping in view the nature of Hepatic blood circulation in human body. As the hematocrit increases, the blood in the arteries remote from the heart shows power law model of non-Newtonian flow. P.N. Pandey and V. Upadhyay have considered that the blood flow has two phased, one of which that of red blood cells and other is plasma. They have also applied the power law model in Bio- fluid mechanical setup. We have collected a clinical data in case of Hepatitis B 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 drop in case of Hepatic disease Hepatitis B.

Keyword: Structure & function of the Liver and hepatic arteries, Hematocrit, Hepatic Blood Flow, Non-Newtonian power law model.

INTRODUCTION

(Description of Bio- Physical Problem)

Structure and function of Liver

The liver is one of the largest, anatomically and functionally most complex organ of the human body. The liver constitutes 2.5% of the human body weight and is the largest organ in the body. [1,2] The liver is a reddish-brown wedge-shaped organ with four lobes of unequal size and shape. Four lobes of man liver are right lobe, left lobe quadrats and caudate lobe. Liver lobes are surrounded by a thick capsule, mostly overlaid

with reflected periforinum. [3] A human liver normally weighs 1.44–1.66 kg (3.2–3.7 lb) [4] and has a width of about 15 cm. Located in the right upper quadrant of the abdominal cavity, it rests just below the diaphragm, to the right of the stomach and overlies the gallbladder. [5]

The liver is connected to two large blood vessels: the hepatic artery and the portal vein. The hepatic artery carries oxygen-rich blood from the aorta, whereas the portal vein carries blood rich in digested nutrients from the entire gastrointestinal tract and also from the spleen and pancreas. These blood vessels subdivide into small capillaries known as liver sinusoids, which then lead to a lobule. Lobules are the functional units of the liver.

Corresponding Author's Contact information:

V. Upadhyay*

Department of Physical Sciences, M.G.C. Gramodaya
Vishwavidhalay Chitrakoot, Satna, Madhya Pradesh, India

E-mail: drvirendra.upadhyay@gmail.com

Each lobule is made up of nutrients from the entire gastrointestinal tract and also from the spleen and pancreas fibroelastic connective tissue layer which extends into the structure of the liver, by accompanying the vessels (veins and arteries), ducts and nerves through the hepatic portal, as a fibrous capsule called Glisson's capsule.

The liver has a wide range of functions, including detoxification of various metabolites and toxic matter, regulation of glycogen storage, decomposition of red blood cells, which are responsible for carrying oxygen around the body, hormone production and the production of biochemical's necessary for digestion. The liver breaks down ammonia into urea as part of the urea cycle, and the urea is excreted in the urine. The liver is the only human internal organ capable of natural regeneration of lost tissue; as little as 25% of a liver can regenerate into a whole liver.^[6]

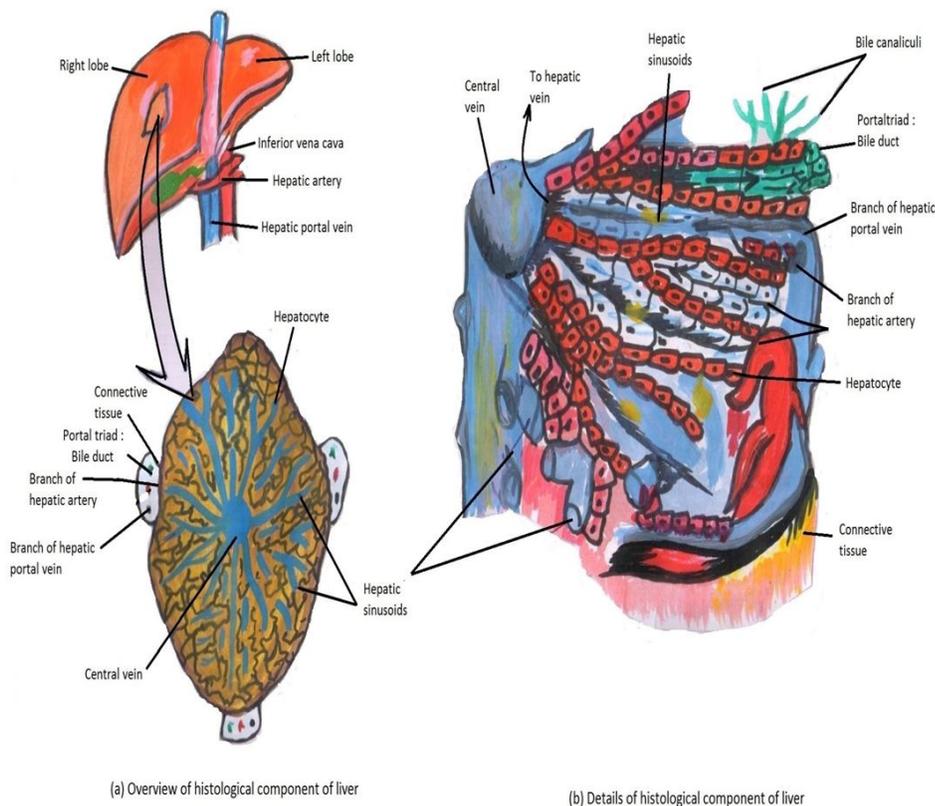


Fig 1: Liver and Its Components

Structure and function of Hepatic Arteries

The hepatic artery “normally” originates from the celiac trunk and branches close to the porta hepatis into the left hepatic artery (LHA) and right hepatic artery (RHA). The artery may be subdivided into the common hepatic artery (CHA) - from the celiac trunk to the origin of the gastroduodenal artery and the hepatic artery 'proper' (PHA) from that point to its bifurcation.^[7] The anatomical knowledge of different variations of hepatic artery is required to reduce the number of iatrogenic complications in traditional and laparoscopic hepatobiliarypancreatic surgery.^[8] In 2010, Ugurel *et al*,^[9] reported that the incidence of the variations of the celiac trunk and/or the hepatic arteries increased with the presence of the accessory renal arteries.

The common hepatic artery is a blood vessel that supplies oxygenated blood to the liver, pylorus of the stomach, duodenum and pancreas. 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.^[10] The liver

normally receives more oxygen than it requires, and it can extract more oxygen to compensate for reduced delivery. ^[11] 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. ^[12,13] Great differences have been found from one species to another ^[14,15] 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. ^[16]

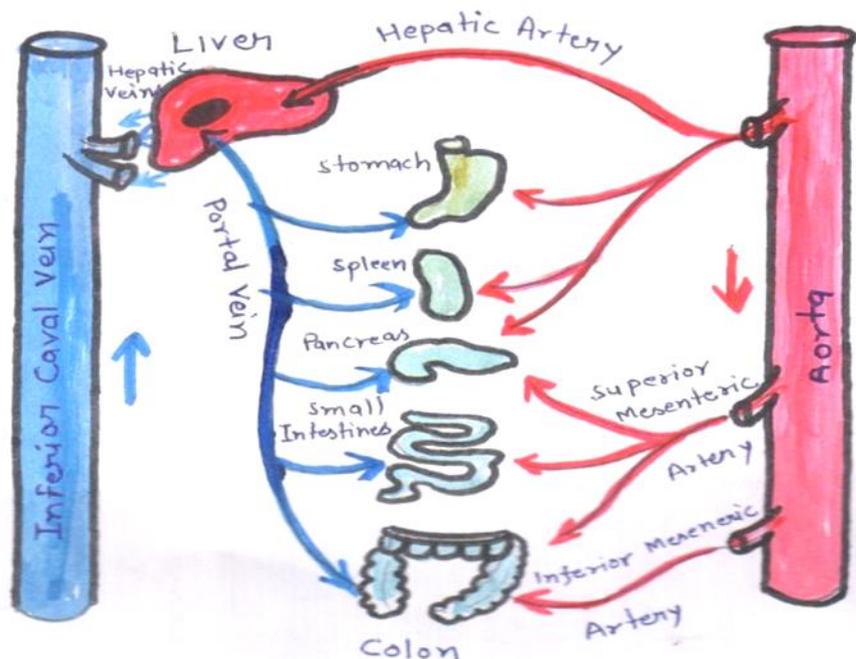


Fig 2: Hepatic Blood Flow

The blood has been supposed non – Newtonian. As the hematocrit(3 times of hemoglobin) increases, the blood in the hepatic arteries remote from the heart shows power law model of non – Newtonian flow. The previous researchers of the this field ^[17] have taken the blood to be single phased. In this model the role of hematocrit which plays vital role in the blood flow flux becomes negligible. The special feature of our two phased blood flow is that the foresaid error is removed and hence the related formula becomes realistic as well as useful. If the effective viscosity of blood is supposed to be zero, the non – Newtonian two phase model reduces to that of Newtonian.

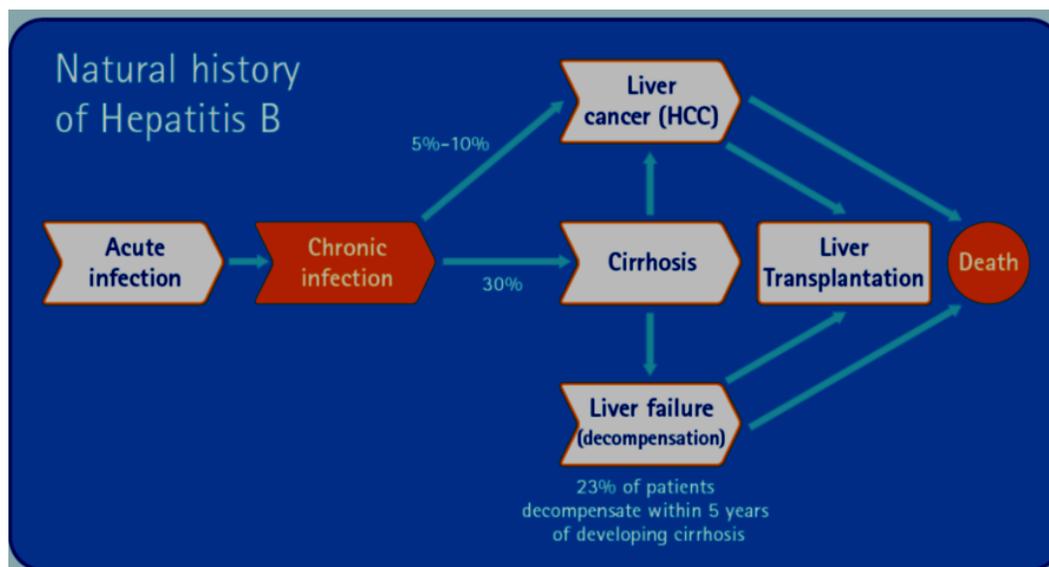
The pressure in the hepatic artery amounts to 100 mm Hg, with a pressure dependent auto regulation of the blood flow (increase of pressure decrease of blood flow, and vice versa).

Hepatitis B and Its Virus

In 1947 Mac Callum classified viral Hepatitis A & viral hepatitis B. Hepatitis B virus was discovered in 1965 by Dr. Baruch Blumberg.

In 1981 The FDA approved sophisticated Plasma, Derived Hepatitis vaccine are human use. In 2001, FDA approved a combination Hepatitis A and Hepatitis B vaccine (Twinrix, Glaxo Smithkline). During 1990-2004, incidence of acute Hepatitis B in the United States declined 75%. The greatest decline (94%) occurred among children and adolescent. People with chronic hepatitis B infection are at increased risk of dying from cirrhosis and liver cancer. Many Cancer Patients at Risk for Hepatitis B Virus Reactivation. According to an estimation of the World Health Organization (WHO), which says

that two to five percent of India's population may be affected by the virus. Each year, one lakh patients die of viral Hepatitis (including Hepatitis B and C) in India.



Adapted from Torresi *et al*, 2000 and Fattovich *et al*, 2003.

REAL MODEL

We have to select 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 E3, 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 (1990).^[18] Now, let the co-ordinate axes be OX^i where O is origin and superscript $i=1,2,3$ 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 $p = p (X^i, t)$ 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 , ie 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, Mainly there are two-phases in blood, The first phase is plasma, while the 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.^[19]

MATHEMATICAL MODELING

First of all the physician poisseuille in 1840 studied the flow of blood in the arteries remote from the heart. According to this study the flux of blood flow is directly proportional to the pressure gradient and

fourth power of the radius of blood vessel. The viscosity of blood can also be evaluated by Poisseuille formula. [20] Even today the physicians in the field of Hemodynamics use the Poisseuille formula.

The blood has been supposed to be non – Newtonian. As the hematocrit increases, the blood in arteries remote from the liver shows power law model of non – Newtonian flow. The previous researchers of this field [21] have taken the blood to be single phased. In this model the role of hematocrit which plays vital role in blood flow flux becomes negligible. The special future of our two phased blood flow is that the foresaid error is removed and hence the related formula becomes realistic as well as useful. If the effective viscosity of blood is supposed to be zero, the non – Newtonian two - phase model is reduces to that of Newtonian.

Whenever the hematocrit increases, the effective viscosity of blood flowing in arteries remote from the liver depends upon the strain rate. In this situation, the blood flow becomes non – Newtonian. When strain rate is in between 5 to 200 per second, the power law $T' = \eta_m e^n$, where $0.68 \leq n \leq 0.80$, holds good for blood flow. In this situation the constitutive equation of blood is as follows [22] :

$$T^{ij} = - p g^{ij} + \eta_m (e^{ij})^n = - p g^{ij} + T'^{ij} \tag{3.1}$$

Where T^{ij} is stress tensor and T'^{ij} is shearing stress tensor.

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

$$\frac{1}{\sqrt{g(\sqrt{g v^i}, i)}} = 0 \tag{3.2}$$

Again the equation of motion is as follows:

$$\rho_m \frac{\partial v^i}{\partial t} + \rho_m v^j v_{,j}^i = T_{,j}^{ij} \tag{3.3}$$

Where T^{ij} is taken from constitutive equation of power law flow.

$\rho_m = X \rho_c + (1 - X)\rho_p$ density of blood and $\eta_m = X \rho_c + (1 - X)\rho_p$ is the viscosity of mixture of blood.

$X = H/100$ is volume ratio of blood cells. H is hematocrit. Other symbols have their usual meanings.

Since the blood vessels are cylindrical, the above governing equation 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 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:

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

Whereas the Christoffel's symbols of 2^{nd} kind are as follows:

$$\left\{ \begin{matrix} 1 \\ 2 \end{matrix} \right\} = -r, \left\{ \begin{matrix} 2 \\ 2 \end{matrix} \right\} = \left\{ \begin{matrix} 2 \\ 1 \end{matrix} \right\} = \frac{1}{r}, \text{ remaining others are zero.}$$

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

$$\begin{aligned} \sqrt{g_{11}}v^1 &= v_r \Rightarrow v_r = v^1 \\ \sqrt{g_{22}}v^2 &= v_\theta \Rightarrow v_\theta = r v^2 \\ \text{and } \sqrt{g_{33}}v^3 &= v_z \Rightarrow v_z = v^3 \end{aligned}$$

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

The matrix of physical components of shearing stress – tensor

$T'^{ij} = \eta_m (e^{ij})^n = \eta_m (g^{jk} v_{,k}^i + g^{jk} v_{,k}^j)^n$ will be as follows:

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

The covariant derivative of T'^{ij} is

$$T'_{,j}{}^{ij} = \frac{1}{\sqrt{g}} \frac{\partial}{\partial x^j} (\sqrt{g} T'^{ij}) + \left\{ \begin{matrix} i \\ j \ k \end{matrix} \right\} T'^{kj}$$

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

$$\frac{\partial v}{\partial z} = 0 \tag{3.4}$$

The equation of motion -

r – component:

$$-\frac{\partial p}{\partial r} = 0 \tag{3.5}$$

θ – component:

$$0 = 0 \tag{3.6}$$

Z – component:

$$0 = -\frac{\partial p}{\partial r} + \frac{\eta_m}{r} \frac{\partial}{\partial r} \left(r \left(\frac{\partial v_z}{\partial r} \right)^n \right) \tag{3.7}$$

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, v_z and p do not depend upon θ . Also the blood flows steadily, i.e.

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

On integrating equation (3.4) we get

$$v_z = v(r) \text{ because } v \text{ does not depend upon } \theta. \tag{3.8}$$

The integration of equation of motion (3.5) yields:

$$P = p(z), \text{ since } p \text{ does not depend upon } \theta. \tag{3.9}$$

Now, with the help of the equation (3.8) and (3.9), the equation of motion (3.7) converts in the following form:

$$0 = -\frac{\partial p}{\partial z} + \frac{\eta_m}{r} \frac{d}{dr} \left(r \left(\frac{dv}{dr} \right)^n \right) \quad (3.10)$$

ANALYSIS (SOLUTION)

The pressure gradient $-\left(dp/dz\right) = P$ of blood flow In the arteries remote from liver can be supposed to be constant and hence the equation (3.10) takes the following form:

$$\frac{d}{dr} \left(r \left(\frac{dv}{dr} \right)^n \right) = -\frac{Pr}{\eta_m} \quad (4.1)$$

On integrating equation (4.1), we get

$$r \left(\frac{dv}{dr} \right)^n = -\frac{Pr^2}{2\eta_m} + A \quad (4.2)$$

We know that the velocity of blood flow on the axis of the cylindrical arteries is maximum and constant. So that we apply the boundary condition, at $r = 0, v = V_0$ (constant), on equation (4.2) to get the arbitrary constant $A = 0$. Hence the equation (4.2) takes the following form:

$$r \left(\frac{dv}{dr} \right)^n = -\frac{Pr^2}{2\eta_m} \Rightarrow -\frac{dv}{dr} = \left(\frac{Pr}{2\eta_m} \right)^{\frac{1}{n}} \quad (4.3)$$

Integrating equation (4.3)once again, we get

$$v = -\left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{r^{\frac{1}{n+1}}}{(n+1)/n} + B \quad (4.4)$$

To determine the arbitrary constant B, we apply the no - slip condition on the inner wall of the arteries: at $r = R, v = 0$, where $R =$ radius of vessel, on equation (4.4) so as to get

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

Hence the equation (4.4) takes the following form:

$$v = \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{n}{n+1} \left[R^{\frac{1}{n+1}} - r^{\frac{1}{n+1}} \right] \quad (4.5)$$

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

RESULT & DISCUSSION (BIO-PHYSICAL INTERPRETATION)

The total flow – flux of blood through the transverse section of the arteries is ^[23] :

$$\begin{aligned} Q &= \int_0^R v \cdot 2\pi r \, dr = \int_0^R \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{n}{n+1} \left(R^{\frac{1}{n+1}} - r^{\frac{1}{n+1}} \right) 2\pi r \, dr \\ &= \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{2\pi \cdot n}{n+1} \left(\frac{R^{\frac{1}{n+1}} \cdot r^2}{2} - \frac{n \cdot r^{\frac{1}{n+1}+1}}{3n+1} \right)_0^R \\ &= \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{2\pi \cdot n}{n+1} \frac{(n+1)R^{\frac{1}{n+1}+3}}{2(3n+1)} \\ Q &= \left(\frac{P}{2\eta_m} \right)^{\frac{1}{n}} \frac{\pi n R^{\frac{1}{n+1}+3}}{(3n+1)} \text{ where } P = -\frac{dp}{dz} \\ Q &= \left[\frac{P_i - P_f}{2\eta_m(Z_f - Z_i)} \right]^{\frac{1}{n}} \frac{\pi n R^{\frac{1}{n+1}+3}}{(3n+1)} \end{aligned} \quad (5.1)$$

Observations: Hemoglobin Vs. Blood pressure is taken from Lala Lajpat Rai and Associated Hospital, Kanpur by Dr S.K. Gautam.

Patient's Name : Mr Ajay Kumar Age/Sex : 65 Years/Male

Clin. : Dr S.K. Gautam Anu. No : 9477

S.No	Date	B.P. (In mm hg)	Hemoglobin (gm / dl)	Hematocrit H	B.P. (In Pascal)
1	12.01.12	100/60	8.9	26.7	13332.0/7799.2
2	14.01.12	100/70	7.5	22.5	13332.0/9332.4
3	16.01.12	110/70	8.1	24.3	14665.2/9332.4
4	18.01.12	100/70	8.7	26.1	13332.0/9332.4
5	20.01.12	120/80	9.2	27.6	15998.4/10665.6
6	22.01.12	120/80	7.0	21.0	15998.4/10665.6

Average Systolic Pressure = 14443.0 pa

Average Diastolic Pressure = 9554.6 pa

H = Average Hematocrit = 24.7

P_i = Pressure on Artery = Average Systolic Pressure
= 14443.0 pa

P_f = Pressure on Arterioles = $\frac{S+D}{2} = \frac{14443.0 + 9554.6}{2}$
= 11998.8 pa

According to Glenn Elert (2010)

η_m = Viscosity of Mixture = 0.0035 p.s.

According to Gustafson, Daniel R. (1980)

η_p = Viscosity of Plasma = 0.0015 p.s. [24]

Length of common hepatic Arteries = 0.0347 m

We know that

$$\eta_m = \eta_c X + \eta_p (1 - X)$$

$$\text{or } \eta_m = \eta_c \frac{H}{100} + \eta_p \left(1 - \frac{H}{100}\right) \text{ Where } X = \frac{H}{100}$$

$$0.0035 = \eta_c \frac{24.7}{100} + (0.0015) \left(1 - \frac{24.7}{100}\right)$$

$$\eta_c = \frac{0.0023705}{0.2470} = 0.009597$$

or η_c = Viscosity of Cells = 0.009597 p.s.

Now putting the value of η_c in η_m , We have

$$\eta_m = 0.009597 \frac{H}{100} + 0.0015 \left(1 - \frac{H}{100}\right)$$

$$\eta_m = (8.097 \times 10^{-5}) H + 0.0015 \quad (5.2)$$

Now from equation (5.1), Flow Flux is given as

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

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

$$0.01666 = \left[\frac{14443.0 - 11998.8}{2 \times 0.0035 \times 0.0347}\right]^{\frac{1}{n}} \frac{(3.14)n [0.0025]^{\frac{1}{n}+3}}{(3n+1)}$$

$$0.01666 = (25156.44298)^{\frac{1}{n}} \left(\frac{n}{3n+1}\right) (4.90625 \times 10^{-8}) 339566.879 = (25156.44298)^{\frac{1}{n}} \left(\frac{n}{3n+1}\right)$$

On solving above equation, we get

$$n = 0.712692$$

Now again from equation (5.1), We have

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

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

Substituting values in above equation, We have

$$\Delta P = \left[\frac{\{(3(0.712692)+1)\}(0.01666)}{(3.14)(0.712692)(0.0025)^3} \right]^{0.712692} \left[\frac{2\eta_m (0.0347)}{0.0347} \right]$$

$$= (27.76)[(8.097 \times 10^{-5}) H + 0.0015]$$

η_m from equation

(5.2)

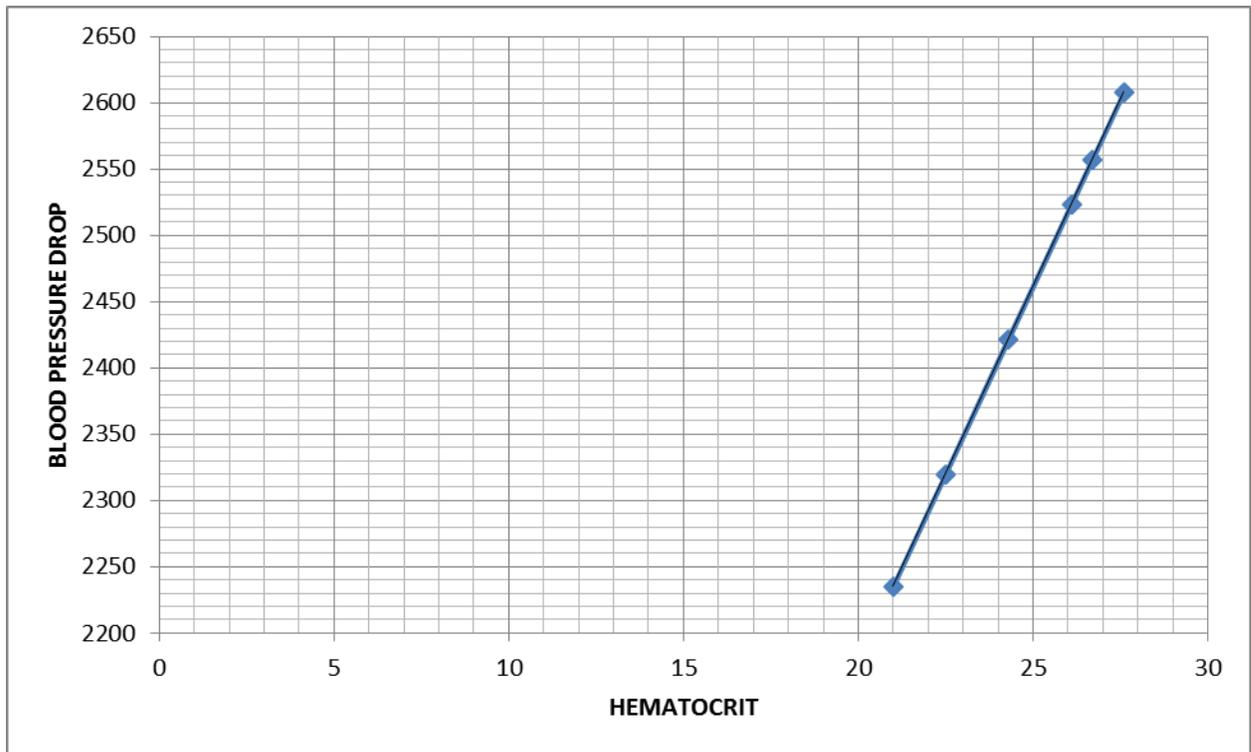
$$\Delta P = 56.5449 H + 1047.516$$

(5.3)

Putting values of H in above equation (5.3), We get the following table of blood pressure drop

Table 1: Hematocrit v/s blood pressure drop

Date	12.01.12	14.01.12	16.01.12	18.01.12	20.01.12	22.01.12
Hematocrit (H)	26.7	22.5	24.3	26.1	27.6	21.0
Blood Pressure Drop (ΔP)	2557.3	2319.8	2421.6	2523.3	2608.2	2234.9



GRAPH 1

Observation of Graph

A simple survey of the graph between blood pressure drop and hematocrit in Hepatitis B patient shows day wise fluctuation in blood pressure drop. We observe that graph shows straight line in decreasing

sense between 12.01.12 to 14.01.12, straight line in increasing sense between 14.01.12 to 20.01.12 via 16.01.12 and 18.01.12, but straight line in decreasing sense between 20.01.12 to 22.01.12.

CONCLUSION

Since when blood pressure drop is increased, then we cannot suggest for operation of liver, But when Blood pressure drop is decreased then operation of liver is suggested because hematocrit is directly proportional to blood pressure drop. Above data are not taken during operation, but it is possible according to nature of graph between 12.01.12 to 14.01.12 and between 20.01.12 to 22.01.12 successful operation is suggested otherwise not.

Acknowledgement

I give my sincere thanks to Dr S.K. Gautam, physician of Lala Lajpat Rai and Associated Hospital, Kanpur (U.P.).

Remark

If this would have been possible to get blood Pressure on the particular tissue (Liver) during operation of Hepatitis B patient then the graph between blood pressure drop and hematocrit will show more accurate information about operation. That is suppose that the graph shows the decreasing sense meaning thereby the blood pressure drop that is fluctuation in blood pressure is decreasing with decrease of hematocrit. In this situation /duration the successful operation is suggested.

References

1. Vollmar B, Menger MD. The hepatic microcirculation: mechanistic contributions and therapeutic targets in liver injury and repair. *Physiol. Rev.* 1269-1339, 2009.
2. Hwang S. Microcirculation of the liver. Venous embolization of the liver. DOI 10.1007/978-1-84882-122-4_2, 2011.
3. Berry AK, Kapoor AS, Nagabhushanam R. *Animal physiology*, 1981.
4. Cotran Ramzi S, Kumar Vinay, Fausto, Nelson, Nelso Fausto, Robbins Stanley L, Abbas, Abul K., Robbins and Cotran pathologic basis of disease (7th ed.). St. Louis, MO: Elsevier Saunders. p. 878. 2005.
5. Tortora Gerard J, Derrickson Bryan H. *Principles of Anatomy and Physiology* (12th ed.). John Wiley & Sons. 2008, 945,
6. Häussinger, Dieter, *Liver Regeneration*. Berlin: De Gruyter. 2011, 1.
7. Sinnatamby CS. *Last's anatomy: regional and applied*. 11th ed. Edinburgh: Elsevier. 2006, 273.
8. Sheldon GF, Rutledge R. Hepatic trauma. *Adv Surg*; 22: 179-93, 1989.
9. Ugurel MS, Battal Bozlar, Nural Tasar, Saglam Karademir. Anatomical variations of the hepatic arterial system, the coeliac trunk and the renal arteries: an analysis with multidetector CT angiography. *The British Journal of Radiology*. 2010; 83(992):661-7.
10. Vollmar B, Menger MD. The hepatic microcirculation, mechanistic contributions and therapeutic target in liver injury and repair. *Physiol Rev*, 2009; 89:1269-1339.
11. Bredfeldt JE, Riley EM, Groszmann RJ. Compensatory mechanisms in response to an elevated hepatic oxygen consumption in chronically ethanol-fed rats. *Am J Physiol*. 1985; 248:G507-G511.
12. Burton-Opitz R. The vascularity of the liver. I. The flow of blood in the hepatic artery. *Quart. J. exp. Physiol*, 1910; 3:297.
13. McMichael J. The oxygen supply of the liver. *Quart. J. exp. Physiol*, 1938; 27:73.
14. Child CG. III. *The Hepatic Circulation and Portal Hypertension*. Philadelphia, Saunders, 1954, 196.
15. Markowitz S, Rappaport A, Scott AC. Prevention of liver necrosis following ligation of hepatic artery. *Proc. Soc. exp. Biol.* (N.1949; 70:305.

16. Graham RR, Cannell D. Accidental ligation of the hepatic artery. Report of one case, with a review of the cases in the literature. *Brit. J. Surg.* 1933; 20:566.
17. Poiseuille J. Recherches expérimentales sur le mouvement des liquides dans les tubes de très petits diamètres, *Comptes Rendus*, II, 961 and 1041, 1840; 12(112):1841.
18. Mishra RS. *Tensors and Riemannian Geometry*, Pothishala Pvt. Ltd. Allahabad, 1990.
19. Sherman IW, Sherman VG. *Biology – A Human Approach* Oxford Univ. press, New York, Oxford, 1989, 278-79.
20. Kapur JN. *Mathematical Model in Biology & Medicine*, EWP New Delhi, 1992; 358:346-47.
21. Ruch TC, Patton HD. (Eds) ; *Physiology and Biophysics*, Vols. II and III; W.B.S. 1973.
22. Kapur JN, Gupta RC. Power-law fluid flow in the inlet length of a circular pipe; *The Math. Seminar.* 1963; 3:55-67.
23. Upadhyay V, Pande PN. A Power law model of two phase blood flow in arteries remote from the heart, Accepted in *Proc. of third Con. Of Int. Acad. Of Phy. Sci.* 1999.
24. Gustafson, Daniel R. *Physics: Health and the Human Body*, Wadsworth, 1980.

Corresponding Author: V. Upadhyay

Journal: The Pharma Innovation

Website: www.thepharmajournal.com

Volume: 1

Issue: 1

Year: 2012

Page no.: 82-92
