A COMPUTATIONAL ANALYSIS OF BLOOD FLOW THROUGH PORTAL VEIN UNDER NORMAL AND EXTRAHEPATIC OBSTRUCTIONS

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ABSTRACT

Portal hypertension is an alarming disease globally whereas Extrahepatic portal vein obstruction (EHPVO) is an important cause of portal hypertension among children in most of the Asian countries. The frequent occurrence of this disease is due to the formation of thrombosis in the portal vein. To acquire insights into the behaviour of hemodynamics, EHPVO case is studied by the technique of computational fluid dynamics (CFD) by considering partial block formation with different sizes inside the main portal vein. On the basis of block sizes, three cases namely normal portal vein (without block in the portal vein) case, 40% block in the portal vein case and 85% block in the portal vein case are simulated by assuming a 2D steady, laminar and fully developed flow with no-slip wall condition using Ansys[TM] Fluent. Our study reveals that the flow distribution between the left and right branches of the portal vein is significantly influenced by the formation of the block in the main portal vein. It is concluded that Extrahepatic portal vein obstruction leads to the possibility of Hypertrophy and Atrophy complex to the liver and could heighten the risk of liver diseases. Our numerical examination will play a great role to the Liver Specialist for understanding the flow behavior with obstructed flow path as well as the diagnosis of associated liver disease and their treatments.

Key words: Portal hypertension, Extrahepatic portal vein obstruction, Computational Fluid dynamics, Hemodynamic, Hypertrophy, Atrophy

INTRODUCTION

The portal vein (PV) is formed by the confluence of the splenic vein and the superior mesenteric vein behind the neck of the pancreas. The portal vein is responsible for around two thirds of the blood flow that rich in oxygen, nutrients growth factors, hormones, among other elements. In the hilum of the liver, a portal vein divides into the left and right portal vein branches which supply blood to the left and right sides of the liver respectively.

Portal hypertension (PH) is caused by altered hemodynamics in the liver. One alteration is increased resistance to flow of blood through the liver and a second is a large increase in the amount of blood trying to enter the liver from the splanchnic circulation

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(Sherlock and Dooley 2002). PH is used to describe elevated pressures in the portal venous system. The elevated pressure is characterized by a gradient of greater than 10 mm Hg between the portal venous and central venous pressures (Bari *et al.* 2012). Causes of increased resistance of blood flow into the liver can be presinusoidal, intrasinusoidal, or postsinusoidal. Presinusoidal causes include blockage of the main portal vein or annular fibrosis of intrahepatic portal venules. Intrasinusoidal causes are mainly due to cirrhosis and include subendothelial deposition of collagen in the space of disse, distortion from regenerating nodules, constriction due to synthesis of NO and increased vasocontrictors (endothelin), and impaired hepatic removal and increased consumption of endotoxins or compression by tumors. Postsinusoidal causes include veno-occlusive disease, obstruction of small hepatic venules, Budd Chiari Syndrome, obstruction of the main hepatic vein, and severe right side congestive heart failure (see Sherlock and Dooley 2002) and Sherlock (1974) classified, an older classification, PH into two main groups – presinusoidal and intrahepatic. The presinusoidal is further divided into extrahepatic, in which the obstruction is in the main portal vein. Portal hypertension is one of the major complications in patients with diseases of the liver, such as liver cirrhosis, veno-occlusive disease, idiopathic extrahepatic portal vein obstruction and pre-hepatic portal idiopathic pathology (Denk 2004, British Liver Trust).

Portal vein thrombosis (PVT) is an important cause of portal hypertension. PVT refers to thrombosis that develops in the trunk of the portal vein or including its right and left intrahepatic branches and may even extend to the splenic or superior mesenteric veins or towards the liver involving intrahepatic portal branches. PVT occurs either in association with cirrhosis or malignancy of liver or may occur without an associated liver disease. Balfour and Stewart (1869) described the first case of PVT in 1868 in a patient with ascites, splenomegaly and variceal dilation. PVT is responsible for 5%-10% of all cases of PH in western countries. Of all cases of PH in developing countries, 40% are attributed to PVT (Ogran *et al.* 2006). Non-cirrhotic prehepatic PH is a consequence of portal vein thrombosis all over the world.

Orllof *et al.* (2002) determined the site of blood flow obstruction in 200 children and young adults. The authors observed that 67% of patients showed only portal vein obstruction, whereas 28% showed portal and splenic vein involvement, and 5% had portal vein and superior mesenteric thrombosis. Portal vein obstruction can occur at any site of the portal system.

When portal hypertension occurs in the absence of cirrhosis, Extra Hepatic Portal Venous Obstruction (EHPVO) is most often indicated etiologically. Patients with EHPVO are generally young and belong mostly to Asian countries. EHPVO is an important cause of PH among children (Sarin SK and Kapoor D 2002). It is characterized mainly by PVT and is detected with upper gastrointestinal bleeding caused by esophageal

varices (Poddar *et al.* 2008). In Japan, 10%-20% of variceal bleeds and in the west, 2%–5% of variceal bleeds are due to EHPVO (Amitrano *et al.* 2004). In children, 70% of all variceal bleeds are due to EHPVO from the Indian subcontinent.

EHPVO can lead to severe bleeding from gastrointestinal varices, ascites, thrombocytopenia from hypersplenism, jaundice and other intrinsic liver diseases (Gibson *et al.* 1965). Decreased growth velocity and short stature is the complication of EHPVO and after shunt surgery, a medication of EHPVO, there is also declining hepatotropic growth factors and growth spurts usually observed (Kato T *et al.* 2000). Growth retardation has been seen in 51% of children in the authors' group of 500 patients due to EHPVO whereas other studies showed this association with decreased levels of IGF-1 and IGF BP-3. Also many kinds of literature explicate a hypothesis that the low supply of blood to the liver due to the formation of systemic collaterals in PVT patients may cause hepatotropic hormone deprivation, thus interfering with the child's global growth (Sarin SK and Agarwal SR 2002).

Computational Fluid Dynamics (CFD) has shown a great potential in computational biology and biomechanics lately due to the limitations of traditional measurement methods. Since CFD uses numerical methods and algorithms to solve and analyze problems that involve fluid flows so its applicability can be exploited for solving blood flow problems. This method can render relatively accurate results of the flow system.

In the study of portal system hemodynamic, Botara *et al.* (2010) implemented the Reynolds Stress model to describe hemodynamic in portal vein system. He used actual patient imaging of the portal vein in CFD modeling to obtain hemodynamic quantities such as velocities and wall shear stress. A very good correlation between simulation results and clinical data has been obtained by this study.

Petkova *et al.* (2003) examined blood flow in an idealized model of the portal vein with and without thrombosis using CFD with both a Newtonian and a Non-Newtonian blood viscosity model. The main focus of this study is that to evaluate the impact of thrombosis on the flow behavior.

Steenkiste *et al.* (2010) carried out a quantitative analysis of the Wall Shear Stress in portal vein in casts of different animal models of portal hypertension and cirrhosis using computational fluid dynamics. This study showed that vascular casting has an important role not only in the morphological evaluation of animal models of portal hypertension and cirrhosis, but also in defining the biological response of the portal vein wall to hemodynamic changes.

George *et al.* (2008) employed an idealized portal vein model to simulate how the blood coming from the splenic vein (SV) and superior mesenteric vein (SMV)

distributions between the left and right lobes of the liver. B. Chen *et al.* (2015) conducted in vitro experimentation and CFD simulation for both an idealized model and specific models based on the CT images acquired from four healthy subjects and two patients with liver cirrhosis. The analysis showed that right-lobe atrophy is significantly influenced by the distribution of blood from the SV. Moreover, the patients with liver cirrhosis had a significantly larger mass fraction of spleen-derived blood in the left portal vein branch (LPV) than healthy subjects, a result consistent with right-lobe atrophy and left-lobe hypertrophy.

It is reported that portal hypertension is one of the major complication in different liver disorders. The obstruction of blood flow in portal vein causes portal hypertension. The objective of the present study is to investigate the change of hemodynamic for the 40% block and 85% block in the portal vein even when the flow conditions are identified as a normal portal vein. A gradual reduction of blood pressure as well as the increment of blood velocity and wall shear stress is observed near the block region of the 40% block portal vein and 85% block portal vein cases respectively. According to this perspective we get a favorable scenario of portal hypertension for 40% blocked and 85% blocked portal vein cases.

MATERIALS AND METHODS

Problem Statement

Here we consider Extra Hepatic Portal Vein Obstruction (EHPVO) by forming the different size of the partial block in the main portal vein (without involving intrahepatic obstruction) to evaluate the hemodynamic quantities. We have used a simple 2D geometry according to Petkova *et al.* (2003) shown in Fig.1, Fig.2 and Fig.3 which has four branches with different flow rates.
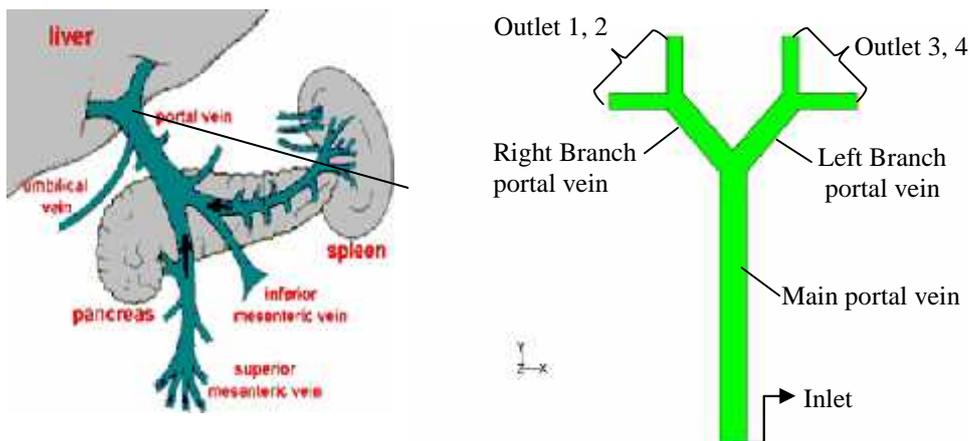

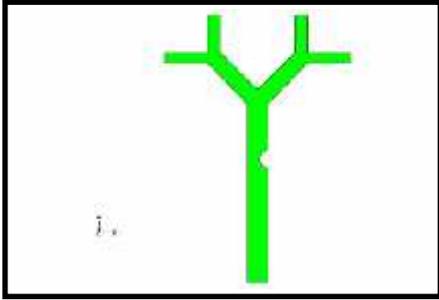Fig. 1. Portal vein with four branches
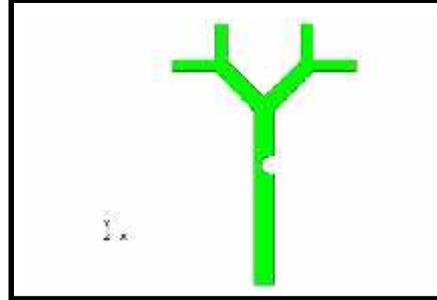
Fig.2: 40% partially blocked portal vein   Fig.3: 85% partially blocked portal vein

Table 1 shows the dimensions of the geometry used in this model.

Table 1: Model dimensions.

Description	Dimensions(mm)
Inlet diameter	10 mm
First branching diameter	8mm
Outlet diameter	6 mm
Total height	150 mm

Fig.1 is the normal portal vein where Fig.2 and Fig.3 have 40% block and 85% block in the main portal vein. The model geometry was constructed by using Gambit 2.2.30 a component of the fluent package. The meshing scheme is composed of elements and type. The elements selected were quadrilateral defines the shape of the element used to mesh and the type parameter defines the meshing algorithm which in our case was paved. After the geometry is successfully meshes, the zone types are specified. A mesh file is exported for use in Fluent 17.2.

GOVERNING EQUATIONS

Blood in the Portal vein manifests laminar flow with a Reynolds number ranging from 663 to 730. Because the Portal vein is far from the heart, the influence of the cardiac cycle can be disregarded. In our study, the 2D blood flow is assumed to be steady and laminar. The three basic laws of conservation of mass, momentum and energy are solved for incompressible flows of non-Newtonian fluid.

The 2D continuity equation or equation for conservation of mass for incompressible flow with steady state can be written as:

$$div\,\vec{V} = 0 \tag{1}$$

Where $\vec{V}$ is the velocity field.

The Navier-Stokes equation for the incompressible flow with steady state in vector form is described as the following form :

$$\rho(\vec{V}\cdot\nabla)\vec{V} = -\nabla P + \nabla\cdot(\bar{\tau}) + \rho\vec{g} \tag{2}$$

Where, ρ is the fluid density, $\vec{V}$ is the velocity field, P is the static pressure, $\bar{\tau}$ is the stress tensor and $\rho\vec{g}$ is the gravitational body force. Though the veins are narrow but the density of blood is very high so there is some gravitational effect which is taken into consideration for this model (Petkova *et al.* 2003, S.M. George 2008, Yin et al 2001).

The stress tensor $\bar{\tau}$ is given by

$$\bar{\tau} = \mu\left[\left(\nabla\bar{V} + \nabla\bar{V}^T\right) - \frac{2}{3}\nabla.\bar{V}I\right] \tag{3}$$

Where μ is the molecular viscosity, I is the unite tensor and the second term of the right hand side is the volumetric dilation.

For incompressible Newtonian fluids viscosity μ is independent of the rate of deformation. But for the non-Newtonian fluids μ is a function of the rate of deformation. Thus for some non-Newtonian fluids, the shear stress can be written in terms of a non-Newtonian viscosity η :

$$\bar{\tau} = \eta(\bar{D})\bar{D} \tag{4}$$

where $\bar{\tau}$ is the stress tensor and $\bar{D}$ is the rate of deformation tensor defined by

$$\bar{D} = \left(\frac{\partial u_j}{\partial x_i} + \frac{\partial u_i}{\partial x_j}\right) \tag{5}$$

The Energy equation is

$$\rho \, div(i\bar{V}) = -P \, div\bar{V} + div)\kappa \; grad \; T) \tag{6}$$

where i is the specific internal energy, $\bar{V}$ is the velocity field, κ is the thermal conductivity and T is the temperature of the fluid. In the case of incompressible flow there is no need to establish the linkage between the energy equation and the equations of continuity and momentum.

In our model the non-Newtonian power law model has been used and the non-Newtonian viscosity η is considered to be a function of the shear rate $\dot{\gamma}$ only. $\dot{\gamma}$ is related to the second invariant of $\bar{D}$ and defined as

$$\dot{x} = \sqrt{\bar{D}:\bar{D}} \tag{7}$$

For our model, the non-Newtonian viscosity is calculated by using the following formula (FLUENT 6.0 Manual, Chapter 7.3.5)

$$y = Ky^{n-1}e^{\frac{T_0}{T}} \tag{8}$$

where K is the power law consistency factor, n is the flow behavior index, T_0 is the

reference temperature. It may be noted that if n = 1 then fluid corresponds to Newtonian behavior, if n < 1 then fluid corresponds to shear-thinning or pseuwwdo-plastics behavior and if n > 1 then shear-thickening or dilatants behavior.

BASIC ASSUMPTIONS

The portal vein or hepatic portal vein is a blood vessel that carries blood from the gastrointestinal tract and spleen to the liver. The blood from the portal vein supplies 80% of the liver's blood and contains nutrients that were absorbed from the digestive tract. As the heart pulsates blood at a certain frequency, the blood that flows from that organ has a pulsatile behavior. However as the blood flows to the rest of the body and the blood vessels get thinner, the blood flow assumes a steady state behavior. In a mathematical point of view two parameters are used to know whether the flow is closer to steady or pulsatile behavior. Those parameters are Reynolds number (Re) and the Womersely number (α). The following equations are used to determine the value of Re and α

$$\text{Re} = \frac{\rho V D}{\mu} \tag{9}$$

and $\qquad \alpha \frac{D}{2} \sqrt{\frac{\rho \omega}{\mu}}$ (10)

where, ρ is the blood density, $\overline{V}$ is the mean velocity, D is the diameter of the blood vessel, ω is the angular frequency, $\omega = 2\pi \times 0.5$ rad/s and μ is the blood viscosity. In our model, the Womersely number is $\alpha = 5.26$ according to equation (10). Since $\alpha < 10$, flow can be assumed steady. Reynolds number is Re=708 according to equation (9) so the flow is laminar.

Blood viscosity can be defined as the quantity that describes the blood's resistance to flow, which is being deformed by either shear stress or tensile stress. For different parts of the circulatory system, different behaviors of blood viscosity seem to happen. In healthy straight large blood vessels, where the blood flow is in high strain rate blood shows Newtonian behavior, however, in small blood vessels, the strain rate is low and the blood viscosity shows a shear-thinning behavior.

The difference in portal blood flow between normal subjects and patients remains unclear. Kayacetin *et al.* (2004) and Vyas *et al.* (2002) found a significant decrease in portal blood flow in patients but Yin *et al.* (2001) found a significant increase in portal blood flow. Several other studies found no significant difference between the two groups. Portal hypertension is considered present when the portal vein flow velocity is less than 21cm/sec (Haag *et al.* 1999).

Since the angles between the branches of the portal vein and their radius do not play any significant impact on the hemodynamic changes so we are not concerned about those.

BOUNDARY CONDITIONS

In every CFD model, apart from the geometry definition and the consequent mesh, there is the need of prescribing boundary conditions. Blood flows by entering and exiting blood vessels, which have walls that do not allow blood to pass through. Therefore it is clear that for the inlet and outlet as well as for the walls there must be boundary conditions. For the inlet a velocity inlet is used. As for the outlets an outflow condition is used. Rigid wall conditions were assumed according to the literature (Botara *et al.* 2010) and neglecting the elastic property (Vimmr *et al.* 2010).

For our study, we consider an average velocity of superior mesenteric vein (SMV) and splenic vein (SV) for the inlet velocity under portal hypertension condition. The operating pressure is 2731.1 Pascal and inlet velocity is 20cm/s. Since the left part of the liver is approximately two times smaller than the right part and therefore has a blood flow twice as high (Orloff *et al.* 2002). All outlets were classified as an outflow in which the flow split was taken with flow rate weighting 0.70 for each outlet 1 and 2 in the right branch and 0.50 for each of outlet 3 and 4 in left branch. Portal vein carries the rich blood of nutrients which has a large amount of water (approximately 70%). For this reason we consider alike-blood as the working fluid whose physical properties (specific heat and thermal conductivity) are similar to water with density $= 1060$ kg/m^3. As mentioned previously, blood shows a shear-thinning behavior and is often modeled as a Non-Newtonian fluid. This behavior is dependent on the strain rate of the fluid. However, a diseased portal vein with decreased blood flow will show a lower strain rate in the range of 1-200s^{-1} where the shear-thinning will be important (Wang *et al.* 2009).

The flow properties of blood used in this study is given in the following table.

Table 2. Flow properties.

Power law index (n)	0.4851
Consistency index κ (kg-s^{n-2}/m)	0.2073
Reference temperature (^{0}K)	310
Minimum viscosity limit η_{max} (kg/m-s)	0.00125
Maximum viscosity limit η_{min} (kg/m-s)	0.003

NUMERICAL METHOD

The pressure based solver was chosen due to the modeling of low-speed incompressible flow. FLUENT solves the governing equations for mass and momentum

in order to obtain the velocity field. To do this a control volume based technique is used. The domain is divided into discrete control volumes based on the computational grid. The governing equations are integrated over the individual control volumes to construct algebraic equations. The discretized equations are linearized and solved to yield values for unknown variables. The formulation for the solver was implicit. The velocity formulation was absolute and porous formulation was superficial velocity. The Green-Gauss Cell based gradient option was used. For the pressure-velocity coupling the SIMPLE (Semi-Implicit Method for Pressure-Linked Equations) algorithm was selected which uses the relationship between velocity and pressure correction to enforce mass conservation and obtain the pressure field. The computations are considered to be converged when the residues for continuity, momentum and energy are less than 10^{-5}.

VALIDITY AND VERIFICATION

To validate our model we simulated blood flow through a normal portal vein (without block) with the boundary conditions as was done by Petkova (2008). We compared velocity distribution with that of Petkova at y = 20mm. As displayed in Fig. 4, the results mostly agreed with Petkova model results except for minor differences which are due to 3D geometry used by Petkova. We proceed with the assumption that our model is a valid model.
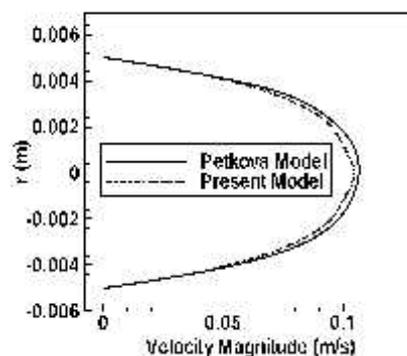
Fig. 4. The velocity distribution at y = 20mm

RESULTS AND DISCUSSION

In this section presents simulation results of blood flow in the normal portal vein as well as 40% block and 85% block in the main portal vein. The velocity was uniform at the inlet and was set 20cm/s. We compared velocity distribution of blood flow through the normal portal vein at three different positions, along with the streamwise direction,

which are at y = 50mm, y = 63mm and y = 75mm respectively. The velocity distributions are the same as expected for all the three positions which are displayed in the Fig.5.
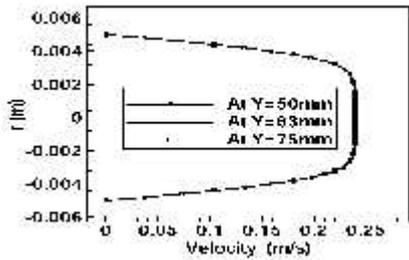

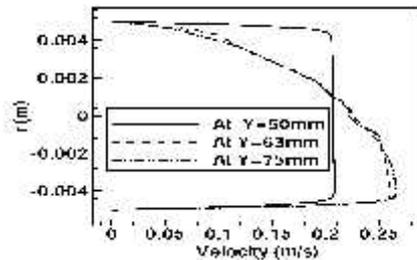Fig. 5. Velocity profiles for normal portal vein

Fig. 6. Velocity profiles for 40% partial blocked in the portal vein

The comparisons of velocity profile for 40% block and 85% block in the portal vein in the same region as mentioned above are represented by Fig.6 and Fig.7. With the comparison of Fig.5 as
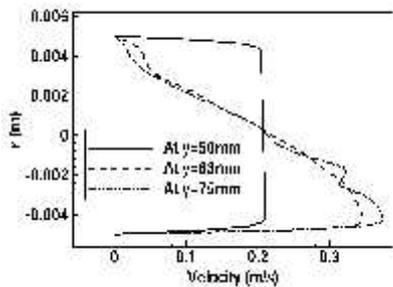

Fig. 7. Velocity profiles for 85% partial blocked in the portal vein
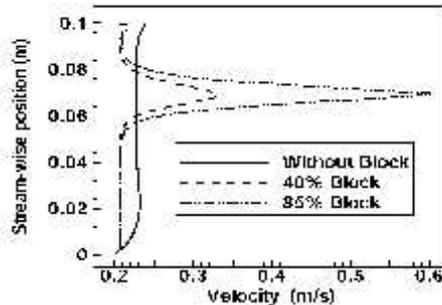
Fig. 8. Velocity profiles along the line at r = −1mm for the three cases

Fig. 8. represents the velocity profile along the line in the position at r = −1mm in the main portal vein for the three cases. This figure depicts the scenario of onward velocity distribution beside the region where the block has been created. The average velocity in the main portal vein are 20cm/s for the normal portal vein, 22.19cm/s for the 40% blocked portal vein and 24.95 cm/s for the 85% blocked portal vein respectively. There is a high blood velocity through the 40% blocked portal vein and is a much higher blood velocity through the 85% blocked portal vein compared with the normal portal vein in consequence of stuffing flow through the narrow flow path.
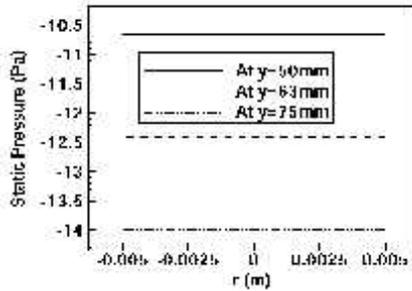
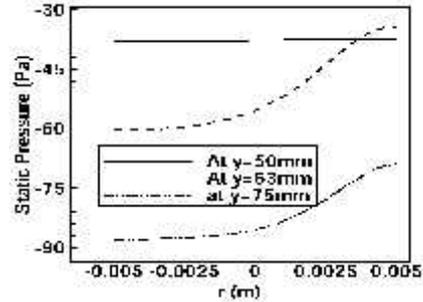Fig. 9. Pressure distribution for normal portal vein

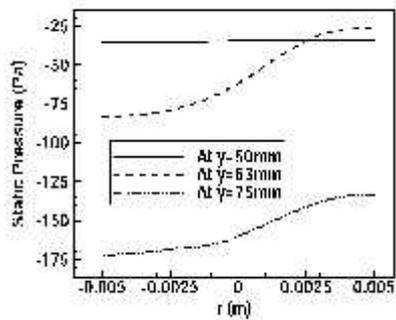Fig. 10. Pressure distribution for 40% block portal vein

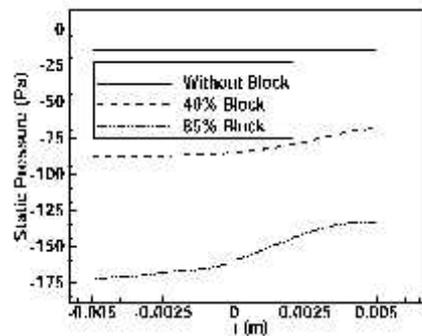Fig. 11. Pressure distribution for 85% block portal vein

Fig. 12. Comparison of static pressure distribution after the block region for the three cases

Fig. 9, Fig. 10 and Fig. 11 show the assimilation of static pressure distribution at three different positions which are at y = 50mm, y = 63mm and y = 75mm for normal portal vein, 40% blocked and 85% blocked portal vein successively. Pressure distribution at these regions, in Fig.9, does not vary significantly for normal portal vein. But a noticeable variation of pressure distribution, in Fig.10 and Fig.11, is observed for 40% blocked and 85% blocked portal vein. Along the line at y = 50mm gives a stagnant pressure distribution for the three cases because of there is no variation of velocity distribution in that position. But along the line at y = 63mm, before the block region, gives an inequitable pressure distribution for the 40% blocked and 85% blocked portal vein as shown in Fig.10 and Fig.11. The same visualization has been observed along the line at y = 75mm, after the block region. High velocity magnitude at these regions has been responsible for this environment of pressure distributions. This observation has been granted by the statement of Bernoulli equation.

Fig.12 shows the comparison of static pressure distribution at the region of the block for the three cases respectively. Since near the block region the velocity is very high and

thereby the pressure is low. This may cause more shrinkage of the vein which stops blood circulation gradually. Its gives the most favorable picture of portal hypertension.
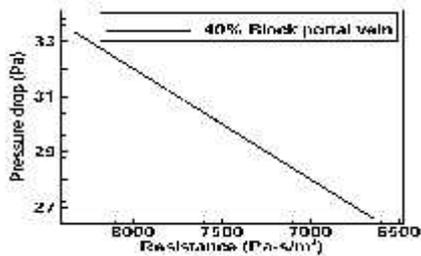

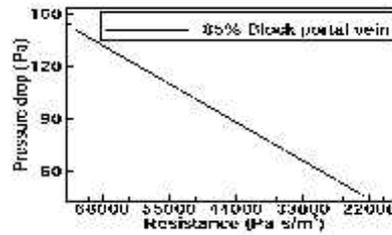Fig. 13. Resistance of flow in the block region for 40% block portal vein.

Fig. 14. Resistance of flow in the block region for 85% block portal vein.

Fig.13 and Fig.14 show the resistance of flow near the block region for 40% blocked and 85% blocked portal vein. Resistance controls the blood flow rate of the blood vessels. Resistance of blood vessel is calculated by using Ohm's law. This law can be stated as - the higher the resistance, the slower the flow rate. The resistance of flow in case of normal portal vein is very minimal which is neglected here. On the other hand, the resistance of flow in case of 85% blocked portal vein is higher than that of 40% blocked portal vein. This is due to the larger amount of reduction of cross-sectional area in 85% blocked portal vein compared to 40% block portal vein. In small width of the vessels, it is hard to push a lot of blood through it. It implies increased the pressure difference and decreased flow rate through the vessel.
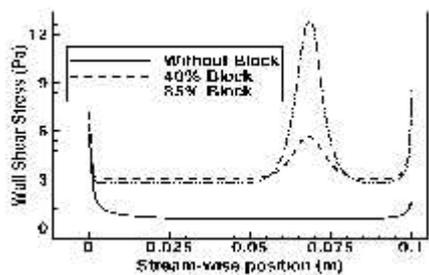

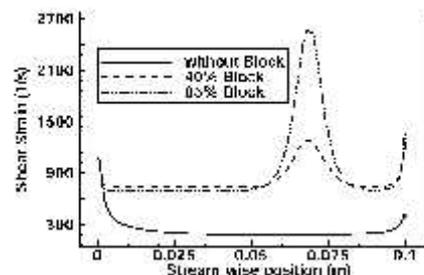Fig. 15. Shear stress on the wall in the main portal vein for three cases

Fig. 16. Shear strain on the wall in the main portal vein for three cases

Fig. 15 is the combined stage of shear stress on the wall for the normal portal vein as well as 40% blocked and 85% blocked portal vein respectively. Also a collection of shear strain on the wall from the three cases is shown by Fig. 16. It can be seen that the wall shear stress and the wall shear strain is almost constant in the flow path for the normal portal vein but a distinguishable depictures for the remainder two cases. The blockage in the main portal vein decreases the available cross section area. This reduction of available

cross section area introduces higher strain rates and shear stress for 40% blocked and 85% blocked portal vein. With an increase of strain rate may contribute to change of viscoelasticity property of blood.

Under the normal circumstances, blood flow through the portal vein is split between the right branch of the portal vein (RPV) and left branch of the portal vein (LPV) in such way that the conservation of mass flow through the liver is maintained. Since the right lobe of the liver is twice than left lobe of the liver so the RPV carries much blood than LPV. The average velocity for RPV and LPV are 16.02cm/s and 10.13cm/s respectively for the normal portal vein. The average velocity for RPV and LPV are 15.95cm/s and 11.38cm/s is observed in the 40% blocked portal vein case. Moreover the average velocities for RPV and LPV in the 85% blocked portal vein case are 15.58 cm/s and 11.46 cm/s.

Table 3 gives the flow rate of RPV and LPV for the normal portal vein, 40% blocked portal vein and 85% blocked portal vein. It is noted that, the flow rate through the RPV

Table 3. The RPV and LPV Flow rate

	Flow rate (m^3/s)	
	RPV	LPV
Normal portal vein	0.00121748	0.000782621
40% Block portal vein	0.00121721	-0.000782531
85% Block portal vein	0.00121735	-0.00078258

and LPV for normal portal vein case, 40% blocked and 85% blocked portal vein cases is almost constant. The constancy is due to the maintenance of conservation of mass through the portal vein. But the flow rate in LPV contains a minus sign for 40% blocked and 85% blocked portal vein cases. This minus sign indicates a decrease of pressure in the direction of flow through the LPV. The decreases of blood pressure through LPV may cause atrophy of the left lobe of liver.

To get the effect of irregular blood flow to the liver, this model is designed with different sizes of block in the portal vein under healthy liver condition and results are presented by considering block sizes effects. This kind of investigation is rare in the literature. The nearest one which is similar to that of ours in some respects is done by Petkova *et al.* (2003) where they considered not a healthy liver but a liver affected by liver cirrhosis.

A vortex flow near the blocked region for the 40% blocked and 85% blocked portal vein is pointed out by Fig. 17 and Fig. 18.
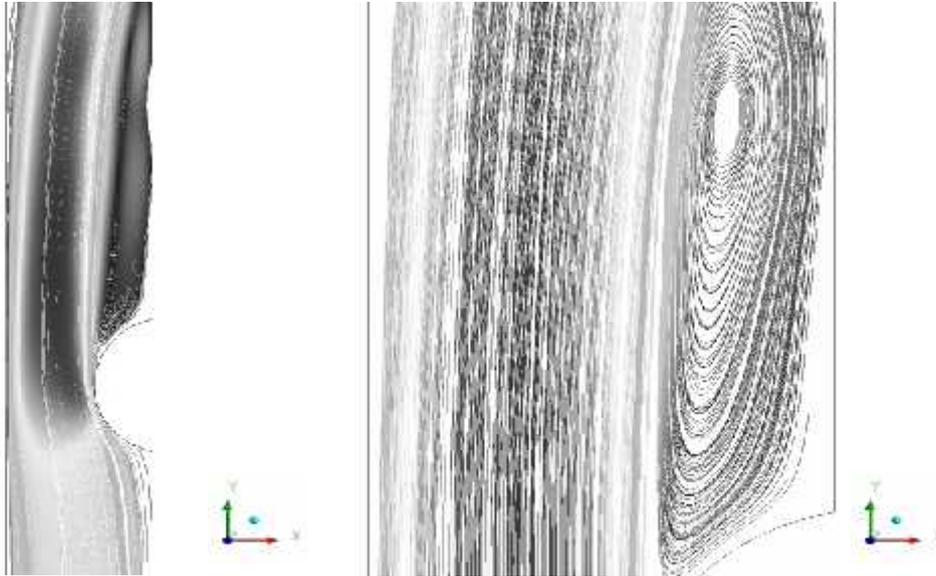

Fig. 17: Streamlines (a) with magnified view of vortex flow (b) for 40% blocked portal vein

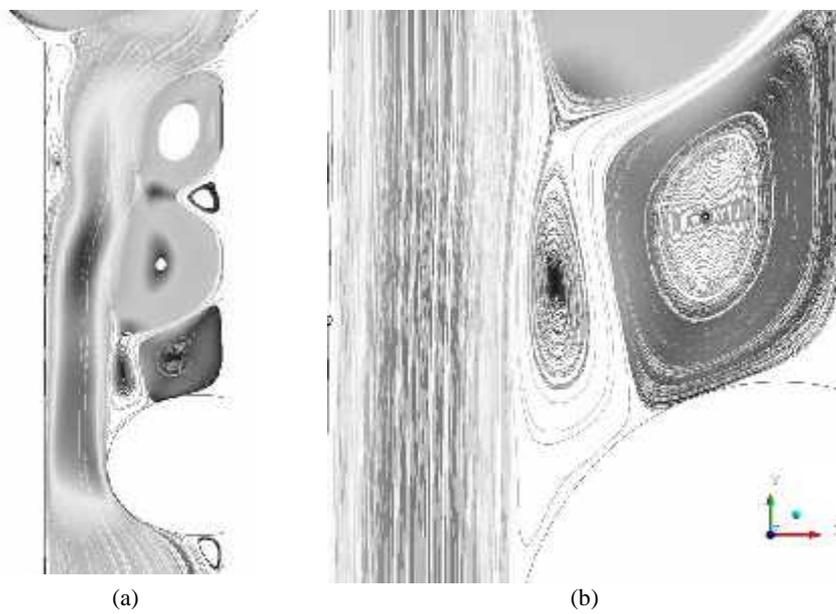(a)   (b)

Fig. 18. Streamlines (a) with magnified view of vortex flow (b) for 85% blocked portal vein

Fig.19 and Fig.20 represent the reverse flow through the RPV and LPV of the portal vein. This vortex and the reverse flows are an indication of advance portal hypertension stage. It can be said that corrosion is possible to happen in such locations. This may leads to form thrombosis in the main portal vein, RPV and LPV of the portal vein.
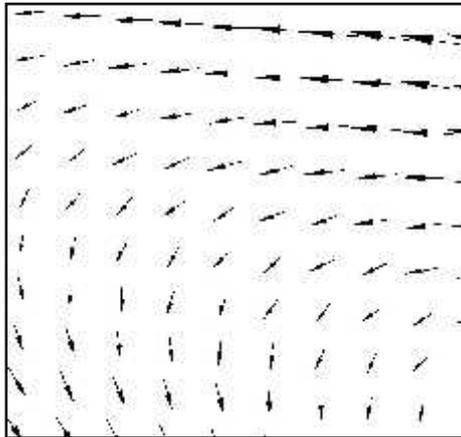

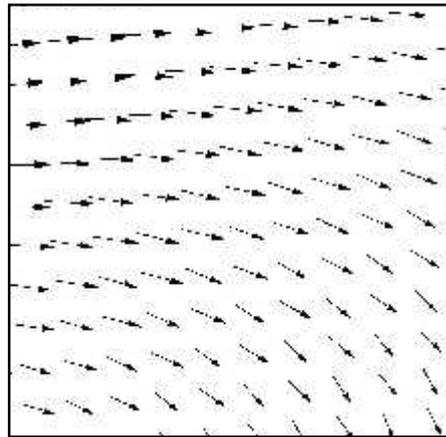Fig.19. Reversed flow through RPV   Fig. 20. Reversed flow through LPV

CONCLUSION

The simulated results of blood flow through portal vein models confirmed that the presence of block in the main portal vein have an effect on the variations of hemodynamic behavior. It is observed that there is a back flow through the blocked portal vein which may haphazard the systemic circulation and can be responsible for the enlargement of spleen and varicose veins in the stomach. Moreover the effect of blockage leads to produce the possibility of forming new thrombosis in the left and right branches of the portal vein which may cause the Hypertrophy and Atrophy complex to the liver. Thus the liver function and its architecture may be affected by the obstructions.

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