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A mathematical study of two phase coronary blood flow in coronary arteries with special reference to angina

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Abstract
This mathematical study focuses on the behaviour of coronary blood flow in coronary arteries during Angina. Blood is modelled as non-Newtonian fluid. Here blood is represented as Power Law fluid model and flow model is shown by the equation of motion and the equation of continuity. Using appropriate boundary conditions, numerical expression for volumetric flow rate, pressure drop and wall shear stress have been derived. Affect the coronary circulation directly as an externally viscous force compressing the blood vessels or they may act indirectly by producing changes in the blood-pressure drop, that is, by altering the pressure perusing the coronary arteries. Any of these factors may alter the coronary blood flow or change the distribution of the flow between the two phases of the heart beat. The role of hematocrit is explicit in the determination of blood pressure in the case of angina. The hematocrit increase the blood pressure drop is also increase. The overall presentation is in tensorial form and solution technique adapted is analytical as well as numerical.

Keywords: Two phase blood flow, coronary blood flow, coronary circulation, angina, plasma, hematocrit, power law model, non-Newtonian, blood pressure drop, behaviour of blood

Introduction

Structure & Function of Heart

Heart is the pump-house of CVS. It composes of four chambers, viz., right atrium (RA), left atrium (LA), right ventricle (RV) and left ventricle (LV). There is a uni- directional valve between RA and RV known as tricuspid valve and the one between LA and LV is bicuspid valve. The pulmonary valve separates right ventricle and pulmonary artery whereas the aortic valve separates aorta and left ventricle. The tricuspid and bicuspid valves are collectively known as atrioventricular valves and the pulmonary and aortic valves are known as semi lunar valves. The deoxygenated blood from die rent parts of the body enters the RA, passes to RV through tricuspid valve. From LV, the blood is pumped to lungs and is oxygenated there. The oxygenated blood enters LA and passes to LV through bicuspid valve. From LV the oxygenated blood is pumped to all parts of the body. Figure1: The Human Heart (courtesy: Wikipedia) The pumping of heart generates sauciest pressure to circulate blood throughout the body. The contraction of atria happens rst, and then the ventricles contract. The contraction is triggered by depolarization of the plasma membrane which initially occurs at the Sino Arial (SA) node. The depolarization then spreads through the muscle cells of atria. Subsequently it spreads and reaches the Atrio Ventricular (AV) node which causes contraction of ventricles [1].

Structure and function of coronary Arteries

Coronary arteries supply blood to the heart muscle. Like all other tissues in the body, the heart muscle needs oxygen-rich blood to function. Also, oxygen depleted blood must be carried away. The coronary arteries wrap around the outside of the heart. Small branches dive into the heart muscle to bring it blood.

The 2 main coronary arteries are the left main and right coronary arteries.

1. Left main coronary artery (LMCA). The left main coronary artery supplies blood to the left side of the heart muscle (the left ventricle and left atrium). The left main coronary divides into branches: The left anterior descending artery branches off the left coronary artery and supplies blood to the front of the left side of the heart. The circumflex artery branches off the left coronary artery and encircles the heart muscle. This artery supplies blood to the outer side and back of the heart.
2. Right coronary artery (RCA). The right coronary artery supplies blood to the right ventricle, the right atrium, and the SA (sinoatrial) and AV (atrioventricular) nodes, which regulate the heart rhythm. The right coronary artery divides into smaller branches, including the right posterior descending artery and the acute marginal artery. Together with the left anterior descending artery, the right coronary artery helps supply blood to the middle or septum of the heart. Smaller branches of the coronary arteries include: obtuse marginal (OM), septal perforator (SP), and diagonals.

Since coronary arteries deliver blood to the heart muscle, any coronary artery disorder or disease can have serious implications by reducing the flow of oxygen and nutrients to the heart muscle. This can lead to a heart attack and possibly death. Atherosclerosis (a build up of plaque in the inner lining of coronary artery causing it to narrow or become blocked) is the most common cause of heart disease [2].

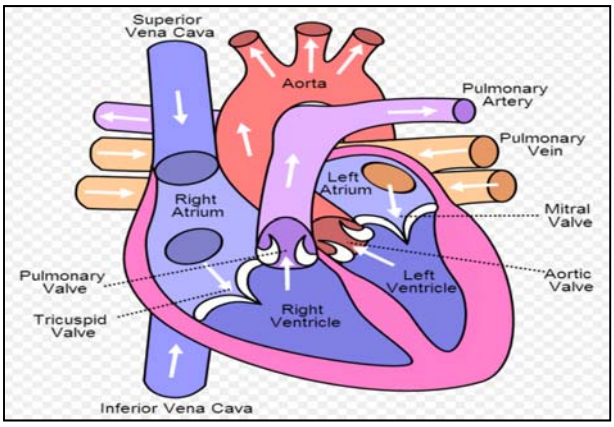


Fig 1: The Human Heart (courtsey: Wikipedia)

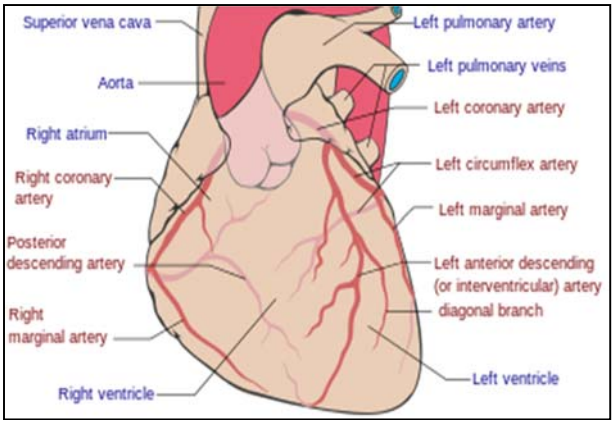


Fig 2: Structure and Function of coronary arteries

Constitution of blood

Blood carries oxygen and nutrients to approximately 1014 cells in the body. It is a heterogeneous solution of blood cells in plasma. The blood cells comprises of Red Blood Cells (RBC), White Blood Cells (WBC) and platelets. Each component has a septic functioning the body. The RBC aids transport of blood gases, WBC adenitis and disposes foreign substances, and platelets aid in clotting. Plasma is a uid which is almost twice as viscous as water. They contain the nutrient such as glucose, electrolytes, vitamins, minerals, enzymes etc. that has to be supplied to cells. In a normal human body there is around ve litres of blood which constitutes about 8% of body weight [1].

Angina Heart disease

Angina pectoris: Commonly known as angina – is chest pain due to ischemia of the heart muscle, generally due to obstruction or spasm of the coronary arteries [3]. The main cause of angina pectoris is coronary artery disease, due to atherosclerosis of the arteries feeding the heart. The term derives from the Latin angina.

Plaque Build-up in an Artery

Figure A shows a normal artery with normal blood flow. The inset image shows a cross-section of a normal artery. Figure B shows an artery with plaque build up. The inset image shows a cross-section of an artery with plaque build up. Figure A shows a normal artery with normal blood flow. The inset image shows a cross-section of a normal artery.

Figure B shows an artery with plaque build up. The inset image shows a cross-section of an artery with plaque build up. Plaque narrows and stiffens the coronary arteries. This reduces the flow of oxygen-rich blood to the heart muscle, causing chest pain. Plaque build up also makes it more likely that blood clots will form in your arteries. Blood clots can partially or completely block blood flow, which can cause a heart attack.

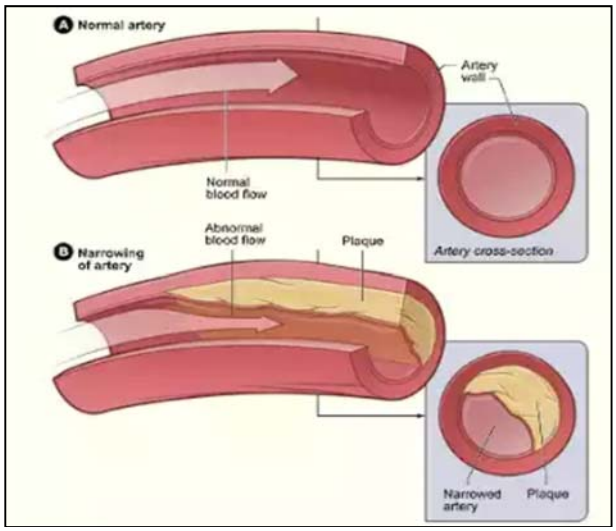


Fig 3: (A) Normal Artery [4].

(B) Narrowing artery

Angina also can be a symptom of coronary micro vascular disease (MVD). This is heart disease that affects the heart's smallest coronary arteries. In coronary MVD, plaque doesn't create blockages in the arteries like it does in CHD. Studies have shown that coronary MVD is more likely to affect

women than men. Coronary MVD also is called cardiac syndrome X and no obstructive CHD.

Types of Angina

The major types of angina are stable, unstable, variant (Prinzmetal's), and microvascular. Knowing how the types differ is important. This is because they have different symptoms and require different treatments.

Stable Angina

Stable angina is the most common type of angina. It occurs when the heart is working harder than usual. Stable angina has a regular pattern. ("Pattern" refers to how often the angina occurs, how severe it is, and what factors trigger it).

Unstable Angina

Unstable angina doesn't follow a pattern. It may occur more often and be more severe than stable angina. Unstable angina also can occur with or without physical exertion, and rest or medicine may not relieve the pain. Unstable angina is very dangerous and requires emergency treatment. This type of angina is a sign that a heart attack may happen soon.

Variant (Prinzmetal's) Angina

Variant angina is rare. A spasm in a coronary artery causes this type of angina. Variant angina usually occurs while you're at rest, and the pain can be severe. It usually happens between midnight and early morning. Medicine can relieve this type of angina [5-6].

Microvascular Angina

Microvascular angina can be more severe and last longer than other types of angina. The cause of Microvascular angina is unknown, but it appears to be the result of spasm in the tiny blood vessels of the heart, arms and legs [7, 8, 9].

Real Model

Choice of frame of Reference

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, [10] 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 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 ρ = ρ (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 [11].

Two Phase Description

Blood is a complex fluid consisting of particulate corpuscles suspended in a non-Newtonian fluid. The particulate solids are red blood cells (RBCs), white blood cells (WBCs) and platelets. 55% of the plasma and 45% of the blood cells in a whole blood and approximately 98% of RBCs in 45% of blood cells and there are a few parts (approximately 2%) of the other cells. Which are ignorable, so one phase of the bloods plasma and 2nd phase of blood is RBCs [12-13].

Constitutive Equations

Generally blood is non-homogeneous mixture of plasma and blood cells. Though for practical purposes it may be considered to be homogeneous two-phase mixture of plasma and blood cells. The constitutive equations proposed for whole blood mixture are as follows: [14]

Newtonian Power law equation

$\tau = \eta\ e^n$ when n = 1 then the nature of fluid is Newtonian [15].

The Non-Newtonian Power Law equation

$\tau = \eta\ e^n$

This is found to be comfortable for starin rate between 5 and 200^{-1}

Where η is viscosity coefficient. This is found to hold good in the broad blood vessels where there is low hematocrit.

It holds good when blood shows yield stress. We notice that the yield stress arise because blood cells form aggregates in the form of rouleaux at low strain rate.

Boundary Conditions are as follows

1. The velocity of blood flow on the axis of arteries at r=0 will be maximum and finite, say V_0 = maximum velocity, V = V_0then A = 0
2. The velocity of blood flow on the wall of coronary artery at r=R, where, R is the radius of coronary artery, will be zero. This condition is well known as no-slip condition. V = 0 at r = R

Mathematical Modeling

Equation of continuity for power law flow will be as follows:

$$\frac{\mathbf{1}}{\sqrt{\mathbf{g}}(\sqrt{\mathbf{g}}\mathbf{V^i})_{,\mathbf{i}}} = 0 \quad \text{..... (3.1)}$$

$$\rho_m \frac{\partial V^i}{\partial t} + (\rho_m V^i)\, V^i_{,j} = T^{ij}_{,j} \quad \text{..... (3.2)}$$

Where T^{ij} is taken from constitutive equation of per law flow. $\rho_m = X\rho_c + (1-X)\rho_p$ is the density of blood and $\eta_m = X\eta_c + (1-X)\eta_p$ is the viscosity of mixture of the blood. X = H/100 is volume ratio of the blood cell; H is the Hematocrit. Other symbols have their usual meanings.

Since the blood vessels are cylindrical, the above governing equations have to be transformed into cylindrical co-ordinates. As we know earlier:

Now we have to transform the equations (3.1) and (3.2) in cylindrical form. As we know. For cylindrical

$X^1 = r$, $X^2 = \Theta$, $X^3 = z$

Matrix of metric tensor in cylindrical co-ordinates is as follows:-

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

While matrix of conjugate matrix tensor is as follow-

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

Whereas the chritoffel's symbol of 2nd kind are as follow:-

$\{{}^{1}_{2\,2}\} = -r$, $\{{}^{1}_{2\,2}\} = \{{}^{1}_{2\,2}\} = \frac{1}{r}$

Remaining others are zero.

Relation between contra variant physical components of the blood flow will be as follows:

$$\sqrt{g_{11}}\, V^1 = V_r \implies V_r = V^1$$
$$\sqrt{g_{22}}\, V^2 = V_\theta \implies V_\theta = rV^2$$
$$\sqrt{g_{33}}\, V^1 = V_z \implies V_z = V^3$$

Again the physical components of $p_{,j}g^{ij}$ is $-\sqrt{g_{ii}}\; p_{,j}g^{ij}$
The matrix of the physical components of shearing stress-tensor
$T^{ij} = \eta_m(e^{ij})^n = \eta_m(g^{ik}V^i_{,k} + g^{jk}V^j_{,k})^n$ Will be as follows

$$\begin{bmatrix} 0 & 0 & \eta_m(\frac{dV}{dr})^n \\ 0 & 0 & 0 \\ \eta_m(\frac{dV}{dr})^n & 0 & 0 \end{bmatrix}$$

The covariant derivative of T^{ij}

$$T^{ij}_{,j} = \frac{1}{\sqrt{g}}\frac{\partial(\sqrt{g}T^{ij})}{\partial x^j} + \begin{Bmatrix} i \\ j\ k \end{Bmatrix} T^{ij}$$

Keeping in view the above fact, the governing tensorial equation can be transformed into cylindrical form which Are as follows; the equation of continuity

$$\frac{\partial v}{\partial z} = 0 \quad \text{.....} \quad (3.3)$$

Equation of motion
r – Component

$$-\frac{\partial p}{\partial r} = 0 \quad \text{.....} \quad (3.4)$$

$$\Theta = \text{component}, 0 = 0 \quad \text{.....} \quad (3.5)$$

$$\text{z component} 0 = -\frac{\partial p}{\partial z} + \frac{\eta_m}{r}[r(\frac{\partial Vz}{\partial r})^n] \quad \text{.....} \quad (3.6)$$

Here, this fact has been taken in view that the blood flow is axially Symmetric in arteries concerned, i.e.

$$V_\Theta = 0 \text{ and } V_r = 0, \quad \text{.....} \quad (3.7)$$

V_z and p do not depend upon Θ. Also the blood flow steadily, i.e,

$$\frac{\partial p}{\partial t} = \frac{\partial V_r}{\partial t} = \frac{\partial V_\theta}{\partial t} = \frac{\partial V_z}{\partial t} = 0 \quad \text{.....} \quad (3.8)$$

Solution
On integrating equation (3.3) we get
$V_z = V(r)$ because V does not depend upon Θ
The integrating of equation of motion (3.5) yields:
P = p (z) since p does not depend upon Θ
Now, with the help of equation (3.7) and (3.8) the equations of motion (3.6) convert in the following form

$$0 = -\frac{dp}{dz} + \frac{\eta_m}{r}\frac{d}{dr}\left\{r\left(\frac{dV}{dr}\right)^n\right\} \quad \text{.....} \quad (3.9)$$

The pressure-gradient $-\frac{\partial p}{\partial z} = P$ of blood flow in the arteries remote the heart can be supposed to be constant and hence the equation (3.8) takes the following form

$$\frac{d}{dr}\left\{r\left(\frac{dV}{dr}\right)^n\right\} = -\frac{pr}{\eta_m} \quad \text{.....} \quad (3.10)$$

On integrating equation (3.8), we get

$$r\left(\frac{dV}{dr}\right)^n = \frac{pr}{2\eta_m} + A \quad \text{.....} \quad (3.11)$$

We know that the velocity of the blood flow on the axis of cylindrical arteries is maximum and constant. So that
We apply the boundary condition at r = 0, V = V_0 (constant), on equation (3.10) takes the following form

$$r\left(\frac{dV}{dr}\right)^n = \frac{pr}{2\eta_m} \implies \frac{-dV}{dr} = [\frac{pr}{2\eta_m}]^{\frac{1}{n}} \quad \text{.....} \quad (3.12)$$

Integrating equation (3.12) once again, we get

$$V = -[\frac{P}{2\eta_m}]^{\frac{1}{n}} \frac{r^{\frac{1}{n}+1}}{n+\frac{1}{n}} + B \quad \text{.....} \quad (3.13)$$

To determine the arbitrary constant B, we will apply the non-slip condition on the inner wall of the arteries at r = R, V = 0, where R = radius of vessel, on equation (3.13) so as to get

$$B = [\frac{P}{2\eta_m}]^{\frac{1}{n}} \frac{nR^{\frac{1}{n}+1}}{n+1}$$

Hence the equation (2.51) takes the following form

$$V = [\frac{P}{2\eta_m}]^{\frac{1}{n}} \frac{n}{n+1}[R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}] \quad \text{.....} \quad (3.14)$$

Which determine the velocity of the blood flow in the artery remote from heart where, P is gradient of blood pressure? And η_m is the velocity of blood mixture.

Bio-Physical Interpretation
The total flow of blood through the transverse section of the arteries is [16]

$$Q = \int_0^R v.2\pi r dr = \int_0^R \left[\frac{P}{2\eta_m}\right]^{\frac{1}{n}} \frac{n}{n+1}(R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1})$$

$$Q = \left[\frac{P}{2\eta_m}\right]^{\frac{1}{n}} \frac{n2\pi}{n+1}[\frac{R^{\frac{1}{n}+1}.r^2}{2} - \frac{nr^{\frac{1}{n}+3}}{3n+1}]_0^R$$

$$Q = \left[\frac{P}{2\eta_m}\right]^{\frac{1}{n}} \frac{n2\pi}{n+1}\frac{(n+1).R^{\frac{1}{n}+3}}{2(3n+1)}$$

$$Q = \left[\frac{P}{2\eta_m}\right]^{\frac{1}{n}} \frac{n\pi R^{\frac{1}{n}+3}}{3n+1} \quad \text{.....} \quad (3.15)$$

The pattern of blood flow can be shown by fig.

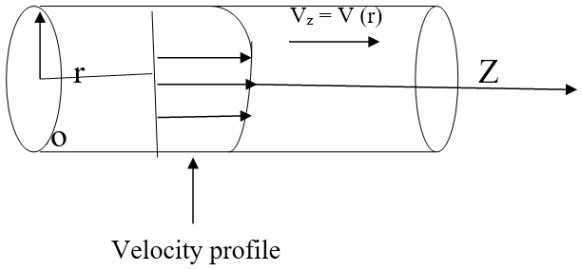


Fig 4: Pattern of blood flow

Observation-Hematocrit vs. blood pressure from an authorized Jabalpur Hospital &Research centre Jabalpur. By Dr Deepak Bahlani Patient case history(Age-76 years old)

Patient case history

S. No	Blood Pressure (mmhg)	Hemoglobin	Hematocrit
1.	120/70	11.9	35.7
2.	120/60	12.1	36.3
3.	100/70	11.8	35.4
4.	120/70	12.5	37.5

Hematocrit (H) = 35.7
$\eta_m = 0.035 pas.sec.$ [17]
$\eta_p = 0.0015 pas.sec.$ [18]

We know that
$\eta_m = \eta_c X + \eta_p(1 - X)$, where X = H/100
0.035 = η_c (0.357) +0.0015 (1- 0.357) $\Longrightarrow$ η_c = 0.095337535pas.sec.

Again using this relation and change in to the hematocrit
$\eta_m = \eta_c X + \eta_p(1 - X) \Longrightarrow \eta_m$ = 0.000938H + 0.0015
From equation (3.15)
P = -dp/dz
We get

$$Q = \left[\frac{\Delta P}{2\eta_m \Delta Z}\right]^{\frac{1}{n}} \frac{n\pi R^{\frac{1}{n}+3}}{3n+1} \quad \text{.....} \qquad (3.16)$$

Q = 250 ml/m = 0.004166 m^3/s [19]
Length of coronary artery $\Delta Z = 50cm. = 0.5m$ [20]
Radius of coronary artery R = 0.2 cm. = 0.002 m [20]
Pressure drop $\Delta P = S - \frac{S+D}{2} = 3328.3 pas.sec.$
Put the value of Q, ΔP, ΔZ and R in equation (3.16)

$$0.004166 = \left[\frac{3328.3}{2 \times 0.035 \times 0.5}\right]^{\frac{1}{n}} \frac{n \times 3.14 \times (0.002)^{\frac{1}{n}+3}}{3n+1}$$

$$0.004166 = [95094.285714]^{\frac{1}{n}}\left(\frac{n}{3n+1}\right)3.14 \times (0.002)^3 \times (0.002)^{\frac{1}{n}}$$

$$165843.949 = \left(\frac{n}{3n+1}\right) \times (190.1886)^{\frac{1}{n}}$$

By using trial method, we get the value of n is
n = 0.3818135702

Again using from equation no. (3.16)

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

$$0.004166 = \left(\frac{\Delta P}{\eta_m}\right)^{\frac{1}{n}} \left(\frac{1}{2 \times 0.5}\right)^{\frac{1}{n}} \left[\frac{0.3818135702 \times 3.14 \times (0.002)^{\frac{1}{0.3818135702}+3}}{3 \times 0.3818135702 + 1}\right]$$

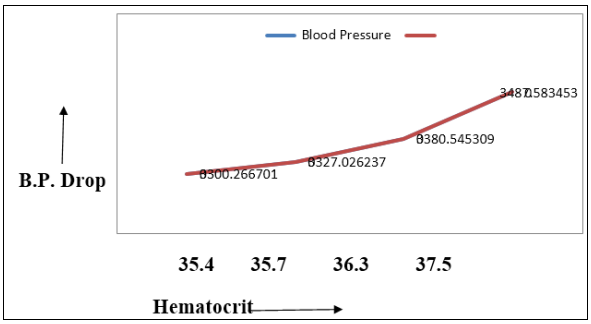
$$\Delta P = 0.000938H + 0.0015) \times (95094.30002)$$

$$\Delta P = 89.19845342H + 142.64145$$

Table for Haematocrit v/s Pressure drop

Hematocrit	35.7	35.4	36.3	37.5
Blood pressure drop	3327.026237	3300.266701	3380.545309	3487.583453

Bio-physical interpretation (Graphical Presentation of clinical data)



Conclusion

A simple survey of the graph between blood pressure drop and hematocrit in cardiac patient shows that when hematocrit is increased the blood pressure drop is also increased.

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