International Research Journal of Pure and Applied Physics, 11 (1),132-143, 2024
ISSN 2055-009X(Print)
ISSN 2055-0103(Online)
Website: https://www.eajournals.org/
Publication of the European Centre for Research Training and Development -UK

Mathematical Modelling and Numerical Simulation of Haematocrit and Pressure Effects on Blood Flow through Blood Vessel

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doi: : https://doi.org/10.37745/irjpap.13vol11n1132143 Published October 29, 2024

Citation: Bunonyo K.W. and Ndu R.I. (2024) Mathematical Modelling and Numerical Simulation of Haematocrit and Pressure Effects on Blood Flow through Blood Vessel, *International Research Journal of Pure and Applied Physics*, 11 (1),132-143

Abstract: *In this article, we derived mathematical models representing blood momentum and the haematocrit in dimensional forms. These models were further scald into dimensionless forms using derived dimensionless quantities. The governing models were converted from partial differential equations to ordinary differential equations considering dense and fully developed flow. In addition, the Laplace method was deployed to solve the dimensionless system and the blood velocity function was obtained with some pertinent parameters such as haematocrit and pressure parameters respectively. The numerical simulations were carried out using Wolfram Mathematica, version 12, varying pertinent parameters within a specific range on the blood velocity and volumetric flow rate profiles. The results depict the effects of haematocrit and pressure parameters on blood velocity as well as flow rate. Specifically, blood velocity varied significantly at different pressure levels; however, it decreased as blood pressure increased due to increased resistance to flow and the narrowing of blood vessels (vasoconstriction), which leads to an increase in boundary layer thickness. Furthermore, the analysis showed a positive correlation between Hct and blood pressure as higher Hct results in increased blood viscosity, potentially elevating peripheral resistance and blood pressure. We also discovered that the blood velocity attained different magnitudes at different haematocrit counts, indicating an indirect relationship between the haematocrit (Hct) and the blood velocity. This indicates that the blood velocity adapts a high altitude increment for an increasing haematocrit. The volumetric flow rate is equally seen to be affected by the aforementioned parameters. This study may serve as a guide in understanding blood flow challenges and offer management advice to patients particularly when hospital data are available for both mathematicians and clinicians alike.*

Keywords: blood, haematocrit, pressure, flow, blood vessel, numerical simulation, and mathematical modelling

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INTRODUCTION

Blood is a fluid that circulates through the blood vessels in the body. In humans, blood consists of plasma (the liquid portion), red and white blood cells, and cell fragments called platelets. Plasma is the main component, primarily made up of water, along with proteins, ions, nutrients, and waste products. Red blood cells are responsible for transporting oxygen and carbon dioxide. The right proportion of red blood cells (haematocrit) is crucial for overall health and is measured as a percentage. Normal haematocrit levels range from approximately 40.7% to 50.3%, depending on the sex. Abnormal haematocrit levels can lead to conditions such as dehydration, chronic lung disease, anaemia, blood loss, or bone marrow disorders, influenced by factors like fluid intake, health conditions, and physical activity. Blood pressure, the force exerted against the walls of blood vessels, plays a vital role in regulating blood flow throughout the circulatory system. It can either positively or negatively affect blood flow, and haematocrit levels are a significant factor influencing both blood pressure and viscosity. Recently, several researchers have carried out studies on blood flow. Among so many, Dhange *et al.* (2022) developed a model for blood flow in stenosed arteries with post-stenotic dilatation. While blood flow through narrow catheterized arteries was analysed by Kumar *et.al.* (2013) focusing on stenosis height and shape.Huo *et al.* (2009) investigated wall shear stress in the coronary arterial tree, considering compliance and haematocrit.Reviewing mathematical modelling of the human circulatory system, Khalid *et al.* (2021) used continuity and Navier-Stokes equations to solve the models. Johnson *et al.* (2011) applied 1D blood flow models to predict differential pressures in arterial networks. The modelling of blood flow in arteries with unsteady overlapping stenosis was done by Roy *et al.* (2013). Sriram *et al.* (2014) investigated haematocrit dispersion in bifurcating vascular networks with a discovery of response to shear stress stimuli with changes in haematocrit.Onitilo *et al.* (2020) researched on the effects of haematocrit on blood flow through stenosed carotid arteries with resistance upshoot as haematocrit increases. Following the work of Elhanafy *et al.* (2020), haematocrit variation's impact on blood flow in arterial segments with variable stenosis was carried out using finite element method. Kumawat *et al.* (2021) examined two-phase blood flow through stenosed curved arteries with haematocrit-dependent viscosity. While Lücker *et al.* (2017) studied the influence of haematocrit and RBC velocity on oxygen transport from capillaries to tissue. Jafarzadeh *et al.* (2020) evaluated haematocrit and nanoparticle diameter effects on haemodynamics and drug delivery. It is observed that increased haematocrit level increases the blood resistance to drug delivery in the artery .Elhanafy *et al.* (2021) went ahead to conduct a numerical simulation of viscoelastic blood flow with haematocrit variation in arterial segments. Furthermore, Lee *et al.* (2018) developed a computational model for blood flow in asymmetrically bifurcating micro-vessels. Gabryś *et al.* (2006) simulated blood flow through fractal models of the circulatory system. Hartung *et al.* (2018) predicted depth-dependent haematocrit distribution in the cerebral cortex. Next, Pralhad *et al.* (2004) modelled arterial stenosis and its effects on shear stress and flow resistance. Curcio *et al.* (2021) provided an overview of mathematical models for haemorrhagic shock. Duffin *et al.* (2020) investigated cerebral blood flow regulation in anaemia and hypoxia. Takahashi *et al*. (2009) modelled hemodynamic parameters in the retinal microvascular network. In this study, the mathematical

model and numerical solutions of blood flow is formulated capturing the haematocrit and the pressure.

Mathematical Formulation

A mathematical model that represents the circulation of blood through an arteriole by the pumping action of the heart will be formulated. Also, we shall look at the volumetric flow rate and shear stress for different values of pressure and haematocrit.

In this study a mathematical model will be formulated to represent the circulation of blood through an arteriole, driven by the pumping action of the heart. Additionally, the volumetric flow rate and shear stress will be analysed for different values of pressure and haematocrit.

Model Formulation

The first system deals with mathematical formulation representing blood flow through a cylindrical vessel caused by the pumping action of the heart that generates a pressure gradient. First, we shall consider the following assumptions before formulating the models:

Assumptions

In order to formulate the mathematical model that the represents the flow of blood through an arteriole with the impact of haematocrit and blood pressure, we assume that:

1. The blood is non-Newtonian, fully developed, and incompressible.
2. The flow is in the axial direction, that is $\vec{w}^* = \left(0, 0, w^*\right)$.
3. The flow is steady $\frac{\partial w^*}{\partial t^*} = 0$.
4. The body is negligible, $F_b = 0$
5. The velocity of the blood is maximum at the centre and zero at the walls.
6. The pressure gradient is constant, that is $-\frac{\partial P}{\partial x} = P_0$

Governing Model

Following the above assumptions and considering the continuity equation and momentum equation, according to Butter *et al.* (2024) and Verma and Parihar (2010), we shall present the flow equations as follow:

Continuity Equation

$$\frac{1}{r^*}\frac{\partial\left(r^* u^*\right)}{\partial r^*} + \frac{\partial w^*}{\partial x^*} = 0 \qquad (1)$$

Momentum Equation

$$\rho_b\left(\frac{\partial w^*}{\partial t}+u^*\frac{\partial w^*}{\partial r^*}+w^*\frac{\partial w^*}{\partial x^*}\right)=\sum F_b-\frac{\partial P^*}{\partial x^*}+\mu_b\left(\frac{\partial^2 w^*}{\partial r^{*2}}+\frac{1}{r^*}\frac{\partial w^*}{\partial r^*}+\frac{\partial^2 w^*}{\partial x^{*2}}\right) \quad (2)$$

Following the above assumptions, equation (2) is reduced to:

$$\frac{\partial P^*}{\partial x^*}=\mu_b\left(\frac{\partial^2 w^*}{\partial r^{*2}}+\frac{1}{r^*}\frac{\partial w^*}{\partial r^*}\right) \quad (3)$$

The corresponding boundary conditions are:

$$\left.\begin{aligned} w^*\neq 0 \quad \text{at } r^*=0\\ w^*=0 \quad \text{at } r^*=R\end{aligned}\right\} \quad (3)$$

Haematocrit

The haematocrit is the percentage of red blood cells in the blood volume, it can be stated according to Verma and Parihar (2010), as:

$$\mu_b=\frac{\mu_0}{\left(1+2.5H\right)} \quad (4)$$

Dimensionless Quantities

We considered the dimensionless quantities below in reducing equations (1)-(3), according to Bunonyo *et al*. (2021) and Bunonyo *et al*. (2024), we have.

$$\left.\begin{aligned} x^*=x\lambda, r^*=rR_0, \upsilon_b=\frac{\mu_b}{\rho_b} w^*=\frac{w\upsilon_b}{R_0}, t^*=\frac{R_0^2 t}{\upsilon_b}, P=\frac{R_0^3 P^*}{\upsilon_b\mu_0\lambda}, P_0=\frac{\partial P}{\partial x}\end{aligned}\right\} \quad (5)$$

Following the aforementioned assumptions and applying the dimensionless quantities, equations (1)-(3) are reduced to:

$$\frac{\partial w}{\partial x}=0 \quad (6)$$

$$\frac{\partial^2 w}{\partial r^2}+\frac{1}{r}\frac{\partial w}{\partial r}=\left(1+2.5H\right)\frac{\partial P}{\partial x} \quad (7)$$

Solving equation (5), we obtained:

$$w=w\left(r\right) \quad (8)$$

Simplifying equation (6), we have:

$$\frac{\partial^2 w}{\partial r^2}+\frac{1}{r}\frac{\partial w}{\partial r}=P_0\left(1+2.5H\right) \quad (9)$$

The corresponding boundary and initial conditions are:

$$\left.\begin{aligned} w\neq 0 \quad \text{at } r=0\\ w=0 \quad \text{at } r=h<\infty\end{aligned}\right\} \quad (10)$$

Laplace Method of Solution (LMS)

We shall adopt the Laplace method of solution of equation (8), the Laplace of different functions can be stated as follows:

$$L\{w(r)\} = \int_0^\infty w(r)e^{-sr}dr = w(s) \tag{11}$$

$$L\{r^n\} = \int_0^\infty r^n e^{-sr}dr = \frac{n!}{s^{n+1}} = w(s) \tag{12}$$

$$L\left\{\frac{\partial^2 w}{\partial r^2}\right\} = \frac{d^2}{dr^2}\int_0^\infty w(r)e^{-sr}dr = \frac{d^2 w}{dr^2} \tag{13}$$

$$L\left\{\frac{\partial w}{\partial r}\right\} = \frac{d}{dr}\int_0^\infty w(r)e^{-sr}dr = \frac{dw}{dr} \tag{14}$$

$$L\left\{r\frac{d^2 w}{dr^2}\right\} = -\frac{d}{ds}L\left\{\frac{d^2 w}{dr^2}\right\} = -\frac{d}{ds}\left(s^2 w(r) - sw(0) - \dot{w}(0)\right) \tag{15}$$

$$L\{rw(r)\} = -\frac{d}{ds}L\{w(r)\} = -\frac{d}{ds}\int_0^\infty w(r)e^{-sr}dr \tag{16}$$

$$L\left\{\frac{dw}{dr}\right\} = \int_0^\infty \frac{dw}{dr}e^{-sr}dr = sw(s) - w(0) \tag{17}$$

Applying the Laplace techniques in equations (14)-(16) on equation (8), we have:

$$L\left\{r\frac{d^2 w}{dr^2}\right\} + L\left\{\frac{dw}{dr}\right\} = P_0 L\{r\} \tag{18}$$

Simplifying equation (17), we have:

$$-\frac{d}{ds}\left(s^2 w(s) - sw(0) - \dot{w}(0)\right) + sw(s) - w(0) = -\frac{P_0(1+2.5H)}{s^2} \tag{19}$$

Simplifying equation (18), we have:

$$-\frac{d}{ds}\left(s^2 w(s)\right) + sw(s) = -\frac{P_0(1+2.5H)}{s^2}$$
$$\frac{dw}{ds} + \frac{1}{s}w = \frac{P_0(1+2.5H)}{s^4} \tag{20}$$

Simplifying equation (18), we have

$$s^2\frac{dw(s)}{ds} + sw(s) = \frac{P_0(1+2.5H)}{s^2} \tag{21}$$

Simplifying equation (20), we have:

$$\frac{dw}{ds} + \frac{1}{s}w = \frac{P_0(1+2.5H)}{s^4} \tag{22}$$

We apply the integrating factor method to resolve equation (21), that is:

$$\text{I.F} = e^{\int\frac{1}{s}} = e^{\log s} = s \tag{23}$$

Multiplying equation (22) and (21), we have:

$$s\frac{dw}{ds}+w=P_0\left(1+2.5H\right)s^{-3} \tag{24}$$

Simplifying equation (23), we have:

$$d\left(sw\right)=P_0\left(1+2.5H\right)s^{-3}ds \tag{25}$$

Integrating both sides of equation (24), we have:

$$w(s)=-\frac{P_0\left(1+2.5H\right)}{2s^3}+\frac{c}{s} \tag{26}$$

Note that, $w(r)=L^{-1}\left\{w(s)\right\}$ (27)

Apply the inverse Laplace of equation (25), which is,

$$w(r)=-\frac{P_0\left(1+2.5H\right)}{2}L^{-1}\left\{\frac{1}{s^3}\right\}+cL^{-1}\left\{\frac{1}{s}\right\} \tag{28}$$

Following equation (11), we have, $L^{-1}\left\{\frac{1}{s}\right\}=1, L^{-1}\left\{\frac{1}{s^2}\right\}=r,$ and $L^{-1}\left\{\frac{1}{s^3}\right\}=\frac{r^2}{2}$,

then equation (27) reduces to:

$$w(r)=-\frac{P_0\left(1+2.5H\right)r^2}{4}+c \tag{29}$$

Applying the boundary conditions in equation (7) on equation (28), we have:

$$c=\frac{P_0\left(1+2.5H\right)h^2}{4} \tag{30}$$

Substituting equation (29) into equation (28), we have:

$$w(r)=\frac{P_0\left(1+2.5H\right)h^2}{4}-\frac{P_0\left(1+2.5H\right)r^2}{4} \tag{31}$$

Simplifying equation (30), we have:

$$w(r)=\frac{P_0\left(1+2.5H\right)}{4}\left(h^2-r^2\right) \tag{32}$$

Volumetric Flow Rate

The volumetric flow rate can be stated mathematically as:

$$Q=2\pi\int_{r=0}^{r=h}wdr \tag{33}$$

Substituting equation (32) into equation (33), and then integrate the result, we have:

$$Q=2\pi\frac{P_0\left(1+2.5H\right)h^2}{4}\int_{r=0}^{r=h}\left(h^2-r^2\right)dr \tag{34}$$

Simplifying equation (34), we have:

$$Q=2\pi\frac{P_0\left(1+2.5H\right)h^2}{4}\left(h^2r-\frac{r^3}{3}\right)_{r=0}^{r=h} \tag{35}$$

Resolving equation (36), we have:

$$Q=2\pi\frac{P_0\left(1+2.5H\right)}{4}\left(h^5-\frac{h^5}{3}\right) \tag{36}$$

Shear Stress at the wall

The shear stress can be calculated mathematically as:

$$\sigma_r = \left.\frac{dw}{dr}\right|_{r=h} \tag{37}$$

Simplifying equation (37) using equation (32), we have:

$$\sigma_r = \left.\frac{dw}{dr}\right|_{r=h} = -\frac{P_0\left(1+2.5H\right)h^3}{2} \tag{38}$$

RESULTS AND DISCUSSION

We performed numerical simulation using Wolfram Mathematica, version 12, where the haematocrit, blood pressure parameters were varied on the blood velocity and the flow rate respectively. The values of the parameters were derived from Bunonyo *et al*. (2021).

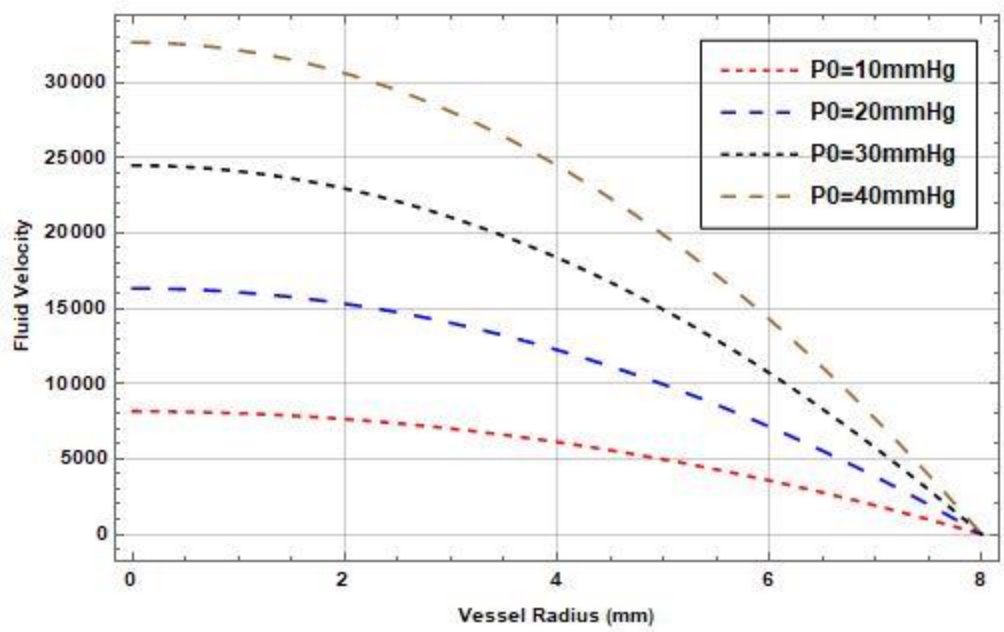

Figure 1: The effect of different values of blood pressure on blood velocity

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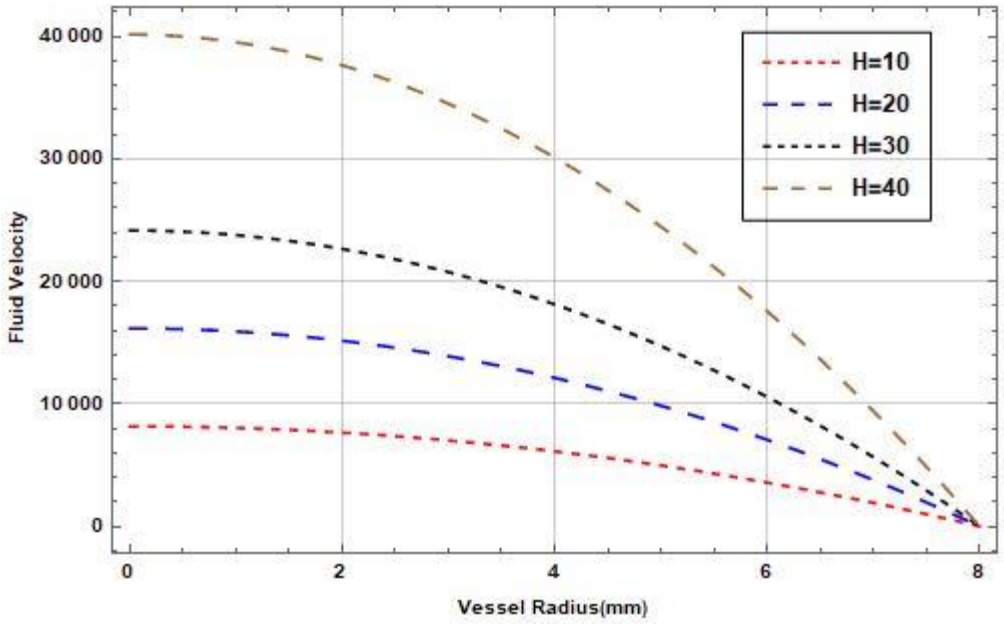

Figure 2: The effect of different values of Haematocrit count on blood velocity

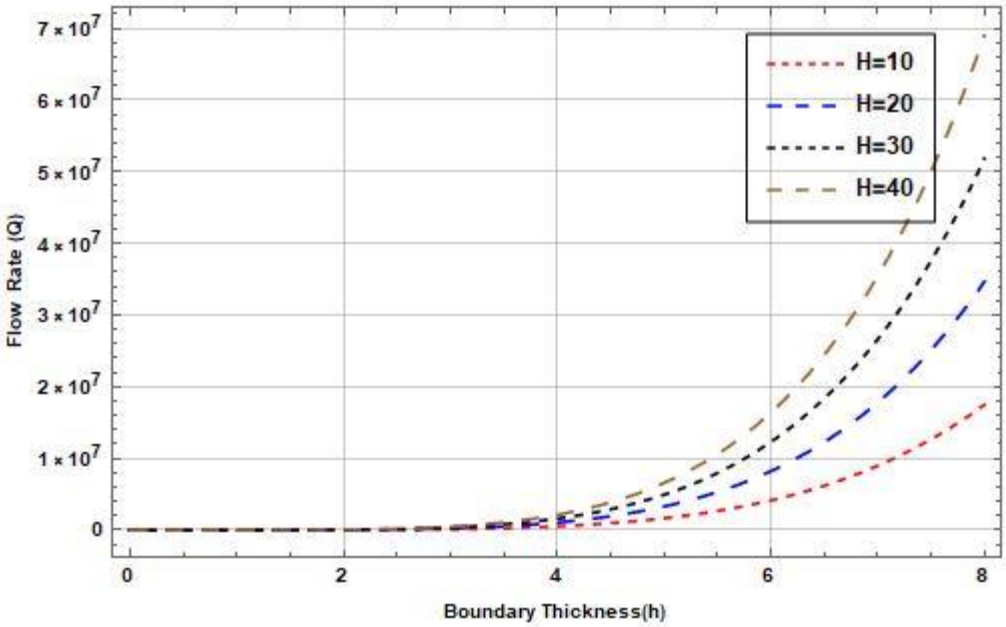

Figure 3: The effect of Haematocrit count on volumetric flow rate

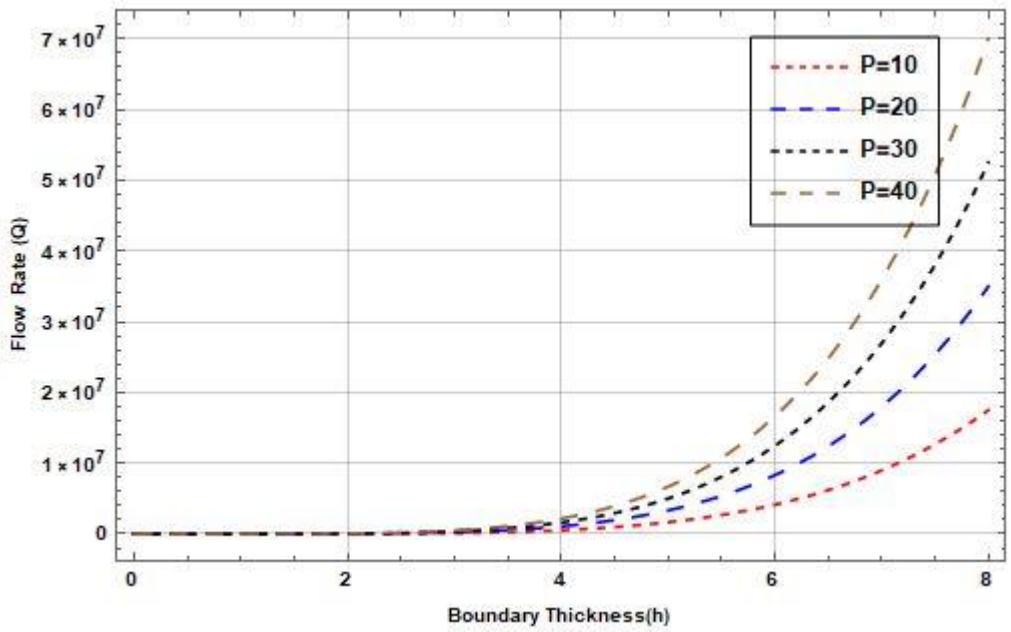

Figure 4: The effect of blood pressure values on volumetric flow rate

DISCUSSION OF RESULTS

Figure 1 illustrates the effect of the pressure level at $P0 = 10, 20, 30, 40$ in mmHg on blood velocity. The result indicates that blood velocity increases to different magnitude at different pressure levels. While the velocity decreases with increasing blood pressure due to increased flow resistance and narrowing of blood vessel (vasoconstriction), which causes the boundary layer thickness to increase from $0 \le r \le 8$. The analysis further showed a positive correlation between Hct and blood pressure, as higher Hct leads to increased blood viscosity, leading to increase in both peripheral resistance and blood pressure.

Figure 2 illustrates the effect of haematocrit count at $H = 10, 20, 30, 40$ unit on blood velocity. This result shows that the blood velocity varies significantly at different values of haematocrit. Specifically, the result depicts that there is an indirect relationship between haematocrit (Hct) and blood velocity. Blood velocity tends to increase in high altitude adaptation for an increasing haematocrit, thereby reaching its highest velocity at $H = 40$. However, the blood velocity decreases as a result of increase in red blood cell count and blood viscosity, due to an increase in boundary layer thickness from $0 \le r \le 8$ millimeter, hence narrowing the flow channel.
The effect of haematocrit at $H = 10, 20, 30, 40$ unit on blood flow rate was investigated with result presented in **Figure 3**. The relationship between haematocrit and flow rate is complex and influenced by some factors. It is within this physical principle that increased haematocrit leads to increased blood viscosity and a decreased blood flow rate. However, in exceptional cases such as high altitude adaptation, a rise in haematocrit can lead to an increase in red blood

count and improve oxygen delivery, potentially resulting in an increased flow rate. The investigation also considered the role of narrowing vessel on flow rate in some exceptional cases and found out that, the flow rate still increases for narrowing vessel diameter, from $0 \le r \le 8$ due to vasoconstriction, which redirects flow to more critical areas. Hence, increasing the flow rate in those areas with the heart pumping more blood (increased cardiac output).Finally, local vasodilation can occur in response to increased blood flow, allowing flow rates to increase despite the narrowing upstream vessels.

Figure 4 depicts the effect of the pressure level at $P0 = 10, 20, 30, 40$ in mmHg on the volumetric flow rate of blood through blood vessels. The figure illustrates the close relationship between blood pressure and flow rate,showing that increased blood pressure leads to an increased flow rate, as shown by direct pressure-flow curves, vascular resistance and auto-regulation cases. However, this investigation also looks at an exception cases such as vasoconstriction in small vessels, increased cardiac output, and local vasodilation, where flow rate still increase for narrowing vessel diameter, from $0 \le r \le 8$. In the case of vasoconstriction, blood flow can be redirected to more critical areas enhancing flow rates in those areas. Similarly within creased cardiac output, if the heart pump more blood (increased cardiac output), narrowing vessels can lead to higher flow rates in specific areas. Finally, local vasodilation can occur in response to increased blood flow, allowing the flow rate to increase despite narrowing upstream vessel.

CONCLUSION

In this study, we have systematically derived and solved a mathematical model representing blood momentum and haematocrit adopting the Laplace method to solve and obtain the blood velocity flow profile, imploring numerical simulation using Wolfram Mathematica, version 12. We can conclude as follows:

The haematocrit and blood velocity are complex and influenced by various factors. While an increased haematocrit can increase blood viscosity and decrease blood velocity. It also improves oxygen delivery and increase blood velocity in certain situations.

The haematocrit effect on flow rate depends on various factors. High haematocrit could increase blood viscosity and potentially reduce flow rate. Low haematocrit can increase blood flow in certain situation due to decreased blood viscosity. However, severe anaemia could have opposite effects which put other contributing factors into considerations.

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Nomenclature

w^*	Dimensional fluid velocity
w	Dimensionless fluid velocity
w_0	Perturbed fluid velocity
x^*	Dimensional displacement variable
x, r	Dimensionless distance variables
Q	Volumetric flow rate
H	Haematocrit parameter
P_0	Constant blood pressure
t	Dimensionless time

Greek letter

μ_b	Dynamic viscosity of blood
υ	Kinematic viscosity parameter
λ	Retardation time parameter
ω	Oscillatory frequency