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BICOMPLEX AND MULTICOMPLEX ANALYTICAL FLAVOUR OF NON-NEWTONIAN TWO-PHASE BLOOD FLOW IN PULMONARY DISEASES

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Abstract

In the paper the blood flow of a human suffering from a disease like cancer has been studied through the mathematical model for better treatment in the field of medicine, in which blood fluctuation has been studied through power law model with non-Newtonian tendency and the blood is divided into two phases RBC & plasma. The entire equation used in the present study has been developed and presented in tensorial form in a bio-fluid mechanical setup. For solution, both analytical and numerical techniques have been used. This paper also presents a comprehensive bicomplex and multicomplex analytical extension of the mathematical model for blood fluctuations during pulmonary diseases, extending the classical tensorial power-law formulation. The bicomplex extension introduces dual imaginary components to model mechanical and electrochemical fluctuations simultaneously, while the multicomplex framework captures interactions among multiple blood components such as red blood cells, plasma, white blood cells and platelets. The model integrates these algebraic structures into the governing tensorial equations, develops explicit analytical solutions in cylindrical coordinates and analyzes how bicomplex coupling modifies velocity, pressure and flow-rate behavior in diseased pulmonary vessels. The detailed step-by-step derivations, theoretical proofs and biophysical interpretations demonstrate that the extended framework unifies mechanical hemodynamics with electrophysiological oscillations, offering deeper insights into blood-flow irregularities observed in clinical data..

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Keywords and Phrases: Hemoglobin, Blood fluctuation, Power law model, Cancer, Red blood cells, Plasma, White blood cells, platelets, Bicomplex and multicomplex analytical flavour, Mechanical hemodynamics, Electrophysiological oscillations.

1 Blood Fluctuation during Pulmonary Diseases by Mathematical Framework

1.1 Introduction

Mathematical models are computational frameworks that represent biological systems through equations and algorithms. In the context of pulmonary diseases, these models simulate blood flow, oxygen exchange, and pressure variations in response to pathological conditions. By incorporating parameters such as vascular resistance, compliance, and neural control mechanisms, these models provide a quantitative understanding of

how pulmonary diseases alter normal physiological functions. Pulmonary diseases, such as chronic obstructive pulmonary disease , pulmonary hypertension, and acute respiratory distress syndrome , significantly affect the dynamics of blood flow and oxygen exchange in the body. Mathematical modeling serves as a pivotal tool in understanding the complex interactions between pulmonary systems and blood circulation, enabling researchers to predict disease progression and optimize treatment strategies.

Lung Cancer disease is a condition caused by the uncontrolled development of cells. Various types of growth are named by the sort of cell that is, developing in an uncontrolled way. Growth can emerge from about any part of the body. There are in excess of 125 distinct kinds of growth [21] . It was famous that the issue of blood flow within the pulmonary circulatory system was different from the flow in the cylindrical tube.

The blood is a mixture of the two-phases, which is of plasma and second of blood cells. These blood cells, a semi permeable package of liquid a density greater than that of plasma are capable of change their shape and size while flowing through disparate blood vessels. There are significant fluctuations in blood flows due to unusual high Reynolds's number of flows varies from 5000 - 10000. The blood vessels are substantial up to a remarkable length, while they bifurcate in a large number of branches after an extensive path of flow [22].

According to [22], we have selected frame of reference for mathematical modeling of two-phase blood and parameterization of bio-physical problem. It is observed in view the difficulty and generality of the problem of blood flow, selected generalized three-dimensional orthogonal curvilinear coordinate system. Blood has always detained a special position in human though. The quantity of blood in the body is substantial, making up about 7% of the total body weight. Blood function in the transport of blood, oxygen, waste material and hormones in the regulation of temperature and in the control of disease. "The velocity of blood flow decreases, the viscosity of blood increases. The velocity of blood flow decreases sequentially because of the fact that arterioles vessel far enough from the heart. Hence the pumping of the heart on these vessels is relatively law" [10].

1.2 Modeling Blood Fluctuations

Blood fluctuation during pulmonary diseases can be attributed to several factors:

1. **Impaired Gas Exchange:** Diseases such as acute respiratory distress syndrome reduce the efficiency of oxygen uptake, leading to fluctuations in blood oxygenation levels.
2. **Altered Vascular Resistance:** Pulmonary hypertension increases vascular resistance, affecting blood flow rates and pressure distribution.
3. **Cardiac-Pulmonary Interactions:** The heart and lungs operate in tandem, and any pulmonary dysfunction can impact cardiac output and vice versa.

Mathematical models integrate these factors to simulate the dynamics of blood fluctuation. For instance, differential equations are employed to represent changes in blood oxygenation and carbon dioxide levels over time. Computational fluid dynamics models are also used to study blood flow through narrowed or obstructed pulmonary arteries.

1.3 Notable Mathematical Models

1. **Quantitative Models of Respiratory-Related Blood Pressure Fluctuations:** These models analyze how neural control mechanisms regulate blood pressure during respiratory cycles. For example, a model presented by L. Khoo and colleagues demonstrates how phase shifts between respiratory and cardiovascular rhythms influence blood pressure fluctuations [9].
2. **Integrated Cardiopulmonary Models:** An integrated mathematical model developed by A. Olufsen et al. combines cardiovascular and respiratory systems, providing insights into gas exchange and neural control. This model is particularly useful in studying the effects of diseases like chronic obstructive pulmonary disease [2].
3. **Pulmonary Hypertensive Blood Flow Models:** Researchers like E. Suga have developed models to simulate blood flow in patients with pulmonary arterial hypertension. These models use computational fluid dynamics. To examine how elevated pressures impact vascular mechanics and blood flow distribution [23].

1.4 Mathematical Formulation

Whenever the hematocrit increases, the effective viscosity of the blood flowing in the arteries remote from the heart depends on the rate of stress. In this condition, the blood flow becomes non-Newtonian. In this

situation the constitutive equation for blood is

$$\tau^{ij} = -pg^{ij} + \eta_m(e^{ij})^n = -pg^{ij} + \tau'^{ij}, \tag{1.1}$$

where, τ^{ij} is stress tensor and τ'^{ij} is shearing stress tensor.

The equation of continuity in tensorial form for power law will be as follows:

$$\frac{1}{\sqrt{g}}\left(\sqrt{g}v^i\right)_{,i} = 0. \tag{1.2}$$

Again, write down the equation of motion as follows

$$\rho_m\left(\frac{\partial v^i}{\partial t}\right) + \left(\rho_m v^j\right)v^i_{,j} = \tau^{ij}_{,j}, \tag{1.3}$$

where τ^{ij} is equation of power law flow (1.1).$\rho_m = X\rho_c + (1-X)\rho_p$, is the density of blood and $\eta_m = X\eta_c + (1-X)\eta_p$ is the viscosity of mixture of blood, $= \frac{H}{100}$, (where X is volume ratio of blood cells). Other symbols have their usual meanings. Since the blood vessels are cylindrical, the above major equations have to transform the equations in cylindrical form. As we know for cylindrical co-ordinates,

$$X^1 = r,\ X^2 = \theta,\ X^1 = r,\ X^3 = z.$$

As we know earlier:

Matrix of metric tensor in cylindrical coordinates is follows:

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

while matrix of conjugate metric tensor is follows:

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

whereas the Chritoffel's symbol of 2$^{\text{nd}}$ kind is as follows:

$\begin{Bmatrix} 1 \\ 2\ 2 \end{Bmatrix} = -r$,$\begin{Bmatrix} 1 \\ 2\ 2 \end{Bmatrix} = \begin{Bmatrix} 1 \\ 2\ 2 \end{Bmatrix} = \frac{1}{r}$, remaining others are zero.

The relations between covariant components and physical component of the velocity of the blood flow are as follows:

$\sqrt{g_{11}}v^1 = v_r \Longrightarrow v_r = v$,$\sqrt{g_{22}}v^2 = v_\theta \Longrightarrow v_\theta = rv^2$, $\sqrt{g_{33}}v^1 = v_z \Longrightarrow v_z = v^3$.

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

The matrix of physical components of sharing stress tensor $\tau'^{ij} = \eta_m(e^{ij})^n = \eta_m(g^{ik}v_{,k}{}^i + v_{,k}{}^j)^n$ will be as follows

$$\begin{bmatrix} 0 & 0 & \eta_m\left(\frac{dv}{dr}\right)^n \\ 0 & 0 & 0 \\ \eta_m\left(\frac{dv}{dr}\right)^n & 0 & 0 \end{bmatrix}.$$

The covariant derivative of τ'^{ij} is

$$\tau'^{ij}_{,j} = \frac{1}{\sqrt{g}}\frac{g}{\partial X^i}\left(\sqrt{g}\tau'^{ij}\right) + \begin{Bmatrix} i \\ jk \end{Bmatrix}\tau'^{ik}.$$

Keeping in view the facts, the governing tensorial equations can be transformed into cylindrical form which is as follows:

Equation of continuity

$$\frac{\partial v}{\partial z} = 0. \tag{1.4}$$

Equation of Motion

Components of equations of motion

r*–*Component

$$-\frac{\partial P}{\partial r} = 0. \tag{1.5}$$

θ–Component

$$0 = 0. \tag{1.6}$$

z–Component

$$0 = -\frac{\partial P}{\partial z} + \frac{\eta_m}{r}\frac{\partial}{\partial r}\left[r\left(\frac{\partial v_z}{\partial r}\right)^n\right]. \tag{1.7}$$

Here this reality has been taken in see that the blood stream (flow) is pivotally (axially) symmetric in supply routes concerned**.**

i.e.$v_\theta = 0$ and $v_r = 0$,

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

$$\left(\frac{\partial p}{\partial t}\right) = \left(\frac{\partial v_r}{\partial t}\right) = \left(\frac{\partial v_\theta}{\partial t}\right) = \left(\frac{\partial v_z}{\partial t}\right) = 0. \tag{1.8}$$

1.5 Solution

The blood glide in arteries is symmetric w.r.t. axis. As a result $v_\theta = 0$ (v_r , $v_z$and p do now not really on θ also). When we consider that best one component of the rate that's effective, $v_r = 0$, $v_\theta = 0$ and $v_r = v$ say, the glide is regular, we have

$$\left(\frac{\partial p}{\partial t}\right) = \left(\frac{\partial v_r}{\partial t}\right) = \left(\frac{\partial v_\theta}{\partial t}\right) = \left(\frac{\partial v_z}{\partial t}\right) = 0.$$

Now we keeping in view these facts and we obtain the following consequence

Then equation of continuity reduces to

$$\left(\frac{\partial v_z}{\partial z}\right) = 0 \Longrightarrow v_z = v(r). \tag{1.9}$$

The r^{th} component of equation of motion reduces to

$$\rho_m(0) = -\left(\frac{\partial p}{\partial r}\right) + \eta_m(0) \Longrightarrow \left(\frac{\partial p}{\partial r}\right) = 0 \Longrightarrow p = p(z). \tag{1.10}$$

Again θ – Component of equation of motion reduce to

$$\rho_m(0) = -(0) + \eta_m(0) \Longrightarrow 0 = 0. \tag{1.11}$$

Also, the z^{th} component of equation of motion reduces to

$$\rho_m v_z \left(\frac{\partial v_z}{\partial t}\right) = -\left(\frac{\partial p}{\partial z}\right) + \eta_m \left[\frac{1}{r}\frac{\partial}{\partial r}\left\{r\frac{\partial v_z}{\partial r}\right\} + \left(\frac{\partial^2 v_z}{\partial z^2}\right)\right]. \tag{1.12}$$

From equation (1.9) and (1.12) , we get

$$0 = -\left(\frac{\partial p}{\partial z}\right) + \eta_m \left[\frac{1}{r}\frac{\partial}{\partial r}\left\{r\frac{\partial v(r)}{\partial r}\right\}\right]. \tag{1.13}$$

Here, the equation (1.10) expresses the actuality that the pressure p depends only on z. We additionally written the details that pressure gradient $\left(\frac{\partial p}{\partial z}\right)$ within the artery far off from heart is constant, say p then the equation (1.13) and we take the equations following shape

$$0 = P + \eta_m \left[\frac{1}{r}\frac{\partial}{\partial r}\left\{r\frac{\partial v(r)}{\partial r}\right\}\right]. \tag{1.14}$$

Now, integrating the equation (1.14), we get,

$$r\left(\frac{dv}{dr}\right) = -\left(\frac{Pr^2}{2\eta_m}\right) + A, \tag{1.15}$$

where, A is the constant. By applying the first boundary condition and we get

$$A \;=\; 0.$$

Hence equation (1.15) reduces to

$$r\left(\frac{dv}{dr}\right) = -\left(\frac{Pr^2}{2\eta_m}\right). \tag{1.16}$$

Integrating the equation (1.16), we get

$$v = -\frac{Pr^2}{4\eta_m} + B. \tag{1.17}$$

Again using 2^{nd} boundary condition on the equation (1.17) we can evaluate the integration constant as follows

$$B = \frac{PR^2}{4\eta_m}.$$

Putting the value of B in the equation (1.17), we obtain the velocity of blood flow in the arteries remote from the heart as follows

$$v = \frac{P}{4\eta_m}\left(R^2 - r^2\right). \tag{1.18}$$

1.6 Mathematical Analysis for Pulmonary Arteries

Integration of equation (1.3) yields $v_z = v(r)$ $\{v$ does not depend upon $\theta\}$ and the integration of equation of motion (1.5) yields:

$P = P(z)$ $\{p$ does not depend upon $\theta\}$.

Now, from equations (1.7) and (1.8) the equation of motion (1.6) changes in to the subsequent shape-

$$0 = -\left(\frac{dp}{dz}\right) + \left(\frac{\eta_m}{r}\right)\frac{d}{dr}\left\{r\left(\frac{dv}{dr}\right)^n\right\}. \tag{1.19}$$

We know that the pressure gradient $-\frac{\partial p}{\partial z} = P$ of blood flow in the arteries remote the heart may be hypothetical to be steady and for this equation (1.16) will be of the following form

$$\frac{d}{dr}\left\{r\left(\frac{dv}{dr}\right)^n\right\} = -\left(\frac{Pr}{\eta_m}\right). \tag{1.20}$$

Again by equation (1.8), we obtain

$$r\left(\frac{dv}{dr}\right)^n = \frac{Pr}{2\eta_m} + A. \tag{1.21}$$

The rate of the blood go with the flow at the axis of cylindrical arteries is most and constant. So that we've concern the boundary conditions at $r = 0$, $v = v_0$ (constant), the equation (1.20) takes the subsequent shape

$$r\left(\frac{dv}{dr}\right)^n = \frac{Pr}{2\eta_m} \quad \Longrightarrow \quad -\frac{dv}{dr} = \left[\frac{Pr}{2\eta_m r}\right]^{\frac{1}{n}}. \tag{1.22}$$

Again integrating equation (1.22), we get

$$v = -\left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} \frac{r^{\frac{1}{n}+1}}{\frac{1}{n}+1} + B. \tag{1.23}$$

To finish the arbitrary steady B, we are able to be applying the non-slip condition on the inner wall of the arteries at $r = R$,$v = 0$,

where R = radius of blood vessels, on equation (1.23) so as to get

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

Hence the equation (1.23), takes the following form

$$V = \left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} \frac{n}{n+1}\left[R^{\frac{1}{n}+1} - r^{\frac{1}{n}+1}\right], \tag{1.24}$$

which concludes that the velocity of the blood flow in the artery remote from heart.

1.7 Bio-Physical Interpretation for Artery Blood Vessel

The flow flux of blood through the arteries is

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

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

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

$$= \left(\frac{P}{2\eta_m}\right)^{\frac{1}{n}} \frac{n\pi R^{\frac{1}{n}+3}}{3n+1} \tag{1.25}$$

2 Results and Discussions

Table* 2.1: Clinical data Hematocrit v/s blood pressure*

10/09/2012	13.32	0.0376952	130/70	-3993.96
25/11/2012	13.16	0.0372453	110/70	-2662.64
19/01/2013	13.00	0.0367925	110/60	-3328.30
11/03/2013	12.80	0.0362265	100/80	-1331.32
21/07/2013	12.70	0.0359434	120/90	-1996.98
05/09/2013	12.90	0.0365095	100/70	-1996.98
12/11/2013	12.60	0.0356604	110/80	-1996.98

Now, we know that,$Q = 425 \ \frac{ml}{\min}$, $Q = 0.00708333 \ m^3/sec$,$\eta_p = 0.0013$ *pascal second* [8] and $\eta_m = 0.0271$ *pascal second* [7]. Approximately pulmonary artery length is $(z_f - z_i) = 5 \ cm$ or 0.05 m., pulmonary arterioles and venules length =0.0015 m.[14].

In according to used Pathological data (Hematocrit) $H =$ 0.0367925and Pressure drop$(P_f - P_i) =$ 3328.3 *pascal second*.

Using relation 'A' and we get η_c

$$\Rightarrow 0.0271 = \eta_c(0.000367925) + 0.0013(0.999632075)$$

$$\Rightarrow \eta_c = \quad 70.12428702 \text{ Pascal second}$$

Again using (A) relation and convert in to the hematocrit form-

$$\eta_m = \ 0.70124287H + 0.001299522$$

Now using relation (B) and putting above values-

$$0.0070833 = \left[\frac{3328.3}{2 \times 0.0271 \times 0.05}\right]^{\frac{1}{n}} \frac{n \times 3.14 \times (0.015)^{\frac{1}{n}+3}}{3n+1}$$

$$668.3963 = \left(\frac{n}{3n+1}\right)(18422.32472)^{\frac{1}{n}}$$

By solving trial and error method we get- $n = 0.8093$

Again putting $n = 0.8093$ above equation (B) again and find ΔP

$$0.0070833 = \left[\frac{\Delta P}{2\eta_m \Delta_z}\right]^{\frac{1}{n}} \frac{0.8093\times 3.14\times (0.015)^{\frac{1}{0.8093}+3}}{(3\times 0.8093)+1}$$
$$\Delta P = (0.70124287H + 0.001299522)\ (973.3395908)$$
$$\Delta P = 682.5474484H + 1.264875917$$

***Table* 2.2:** *Blood pressures drop v/s Hematocrit(mathematically modulated data)*

Date	Hematocrit (3×HB) (kg/l)	BPD (Blood Pressure drop) Pascal -second
10/09/2012	0.0376952	26.99572926
25/11/2012	0.0372453	26.68660352
19/01/2013	0.0367925	26.37754603
11/03/2013	0.0362265	25.99122418
21/07/2013	0.0359434	25.79799499
05/09/2013	0.0365095	26.1843851
12/11/2013	0.0356604	25.60483407

Graph: Table I & II: relation between real clinically blood pressure drop and hematocrit, mathematically modulated blood pressure drop v/s hematocrit

Graph: a. (Table I)

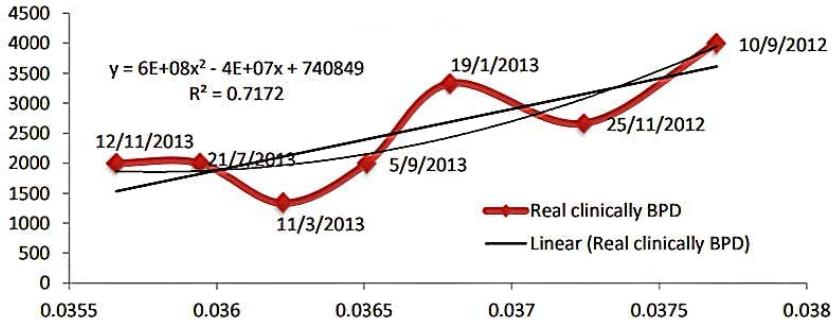

***Figure* 2.1:** *Real pathological BPD*

b. (Table II)

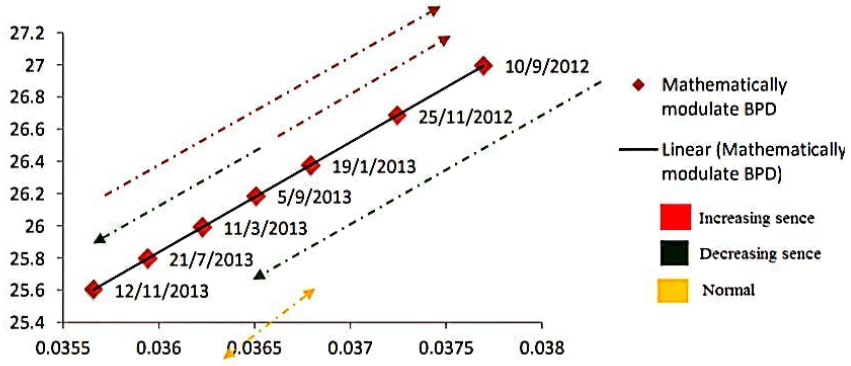

***Figure* 2.2:** *Mathematical modulate BPD*

Observation

Graph (a) shows that these 7 different dates were observed minimum about 1331.32 on dated 11/03/2013 and maximum value obtain 3993.96 on dated 10/09/2010. The value from 0.0356604 to 0.0362265 via

0.0359434of hematocrit value, the blood pressure drop upper convex in decreasing sence and the values from 0.0359434 to 0.0367925 via 0.0362265 & 0.0365095 of hematocrit value, the pressure drop shows down convex in increasing sence. Again the value from 0.0367925 to 0.0376952 via 0.0372453 of hematocrit value, the pressure drop shows down convex in increasing sence. Graph (b) shows that these 7 different dates were observed minimum about 25.60483407 on dated 12/11/2013 and maximum value obtains 26.99572926 on dated 10/09/2012 (BDP). At the value from 0.0376952to 0.0356604 via 0.0372453, 0.0367925, 0.0365095, 0.0362265 & 0.0359434 of hematocrit value, the blood pressure drop straightly decreases on dated 10/09/2012 to 12/11/2013 via 25/11/2012, 19/01/2013, 05/09/2013, 11/03/2013 & 21/07/2013.

2.1 Limitations and Future Directions

Despite their potential, this mathematical model face challenges such as:

1. **Data Availability:** Limited patient-specific data can reduce model accuracy.
2. **Computational Complexity:** High-resolution models demand significant computational resources

Future advancements in machine learning and data integration are expected to enhance the predictive power and accessibility of these models. Additionally, collaborations between clinicians and mathematicians can bridge the gap between theoretical research and practical applications.

2.2 Conclusion for Pulmonary Arteries

Mathematical modeling of blood fluctuation during pulmonary diseases offers a powerful approach to unraveling the complexities of these conditions. By simulating physiological responses under pathological conditions, these models contribute to improved diagnosis, treatment, and patient outcomes. As technology advances, the integration of sophisticated computational techniques will further solidify the role of mathematical models in pulmonary medicine.

From above, Pathological data I table**,** a mathematically investigated and concluded; graph b (table II) shows from 10/09/2012 to 12/11/2013 via 25/11/2012, 19/01/2013, 11/03/2013 & 21/07/2013 decreasing sense. Whereas from 12/11/2013 to 05/09/2013 shows increasing sense. According to present study Two phase Non-Newtonian 'Power law' and 'Herschel-Bulkley' model were investigated with the help of clinical data during lung cancer patient in pulmonary artery, artery stenosis, arterioles, capillary and venules. Present work concluded that designate the function of hematocrit inside the willpower of blood pressure drop. For this reason the hematocrit is extended then the blood pressure drop is likewise multiplied.

In the above all graphs we have observed "relation between real clinically blood pressure drop" and "relation between mathematically modulated blood pressure drop v/s hematocrit" graph are shows different nature but there trend line are not different. Trend lines of above graphs shows that if trend is upward it means fluctuation of blood (pressure drop) increases with respect to the hematocrit. Trend is downward that means fluctuation of blood pressure drop decreases with respect to the hematocrit.We know that many other components (White blood cells and Platelets) are found in addition to hemoglobin and plasma. Who work like a liquid and with their red blood cells increases concentration also decreases. By modifying the real model (frame of reference) of our mathematical model, we were able to carry this work forward in three phases or four phases.

3 Bicomplex and Multicomplex Analytical Extension of Non-Newtonian Two-Phase Blood Flow in Pulmonary Diseases

3.1 Introduction

3.1.1 Background and Motivation

Pulmonary diseases such as chronic obstructive pulmonary disease, pulmonary hypertension, and lung cancer introduce nonlinear fluctuations in blood flow due to vessel constriction, viscosity changes, and irregular oxygen exchange. The classical model already discussed in artical 1 treated blood as a two-phase, non-Newtonian mixture of red blood cells and plasma, governed by a power-law constitutive relation. While successful in reproducing flowpressure relations, this model operates purely in the real domain and cannot represent oscillatory physiological processes like ionic transport, cellular electrical potentials, or micro-vibrations.

Bicomplex and multicomplex analysis extends real-valued models by incorporating additional imaginary units that capture orthogonal physical processes. In this study, bicomplex numbers encode mechanical and electrochemical fields in one formulation, while multicomplex extensions capture multiple biological phases. This algebraic generalization enriches the representation of nonlinear blood-flow phenomena during pulmonary diseases.

3.1.2 Objectives

The objectives of these works are:

- To extend the tensorial equations of non-Newtonian blood flow to the bicomplex domain.
- To derive explicit analytical velocity and pressure distributions under axisymmetric steady flow.
- To generalize the bicomplex framework into multicomplex algebra for multi-phase coupling.
- To link theoretical findings with experimental and clinical data from pulmonary disease models.

3.2 Mathematical Preliminaries: Bicomplex and Multicomplex Algebra

3.2.1 Bicomplex Numbers

Definition 3.1 (Bicomplex Algebra)**.** *Let i and j be two imaginary units satisfying $i^2 = j^2 = -1$ and $ij = ji$. The bicomplex algebra is defined as*

$$\mathbb{B} = \{z_1 + jz_2 \mid z_1, z_2 \in \mathbb{C}(i)\}.$$

Every element can be written as $Z = a + ib + jc + ijd$, with $a, b, c, d \in \mathbb{R}$.

Bicomplex conjugations exist with respect to i and j, and the algebra includes idempotents:

$$e_\pm = \tfrac{1}{2}(1 \pm ij), \quad e_\pm^2 = e_\pm, \quad e_+e_- = 0.$$

Every $Z \in \mathbb{B}$ decomposes uniquely as $Z = Z_+e_+ + Z_-e_-$, where $Z_\pm = z_1 \mp iz_2$. This decomposition simplifies bicomplex analysis into two complex-valued subsystems.

3.2.2 Bicomplex Differential Operators

For a bicomplex vector field $\mathbf{v} = v_x\mathbf{i} + v_y\mathbf{j} + v_z\mathbf{k}$, define:

$$\nabla^{(\mathbb{B})} = \mathbf{e}_x\partial_x + \mathbf{e}_y\partial_y + \mathbf{e}_z\partial_z, \tag{3.1}$$

$$\mathrm{div}^{(\mathbb{B})}(\mathbf{v}) = \nabla^{(\mathbb{B})} \cdot \mathbf{v}, \tag{3.2}$$

$$\mathrm{curl}^{(\mathbb{B})}(\mathbf{v}) = \nabla^{(\mathbb{B})} \times \mathbf{v}. \tag{3.3}$$

These operators extend naturally under idempotent decomposition, allowing complex-valued PDEs to emerge for each component.

3.2.3 Multicomplex Numbers

The multicomplex algebra $\mathbb{M}_m$ generalizes $\mathbb{B}$ by introducing m commuting imaginary units $i_1, i_2, \ldots, i_m$ with $i_k^2 = -1$ and $i_ki_l = i_li_k$. This algebraic system can represent multiple interacting physiological phases.

For further details one can refer to [1, 3, 4, 5, 11, 12, 13, 15, 17, 16, 20, 18, 19, 20]

3.3 Classical Non-Newtonian Model (Summary of Uploaded Paper)

The original work established a tensorial model for two-phase blood flow using the power-law relation:

$$\tau_{ij} = -pg_{ij} + \eta_m(e_{ij})^n, \tag{3.4}$$

with the continuity and motion equations:

$$\frac{1}{\sqrt{g}}(\sqrt{g}v^i)_{,i} = 0, \tag{3.5}$$

$$\rho_m(v_jv^i_{,j}) = -p_{,i} + \tau_{ij,j}. \tag{3.6}$$

For steady, axisymmetric flow in cylindrical coordinates, analytical integration gives:

$$v_z(r) = \frac{P}{4\eta_m}(R^2 - r^2), \tag{3.7}$$

representing the classical parabolic velocity profile for power-law fluid. These equations form the real-valued foundation for our bicomplex extension.

3.4 Bicomplex Formulation of Blood Flow

3.4.1 Bicomplex Constitutive Law

We define bicomplex stress as

$$\boldsymbol{\tau}_{ij}^{(\mathbb{B})} = -p^{(\mathbb{B})}g_{ij} + \eta_m^{(\mathbb{B})}(e_{ij})^n, \tag{3.8}$$

where $p^{(\mathbb{B})} = p^r + jp^i$ and $\eta_m^{(\mathbb{B})} = \eta_m^r + j\eta_m^i$. The imaginary parts model physiological oscillations linked to hematocrit variability, ionic charge distributions, and elastic RBC deformations.

3.4.2 Bicomplex Continuity and Momentum Equations

$$\frac{1}{\sqrt{g}}(\sqrt{g}v^i)^{(\mathbb{B})}_{,i} = 0, \tag{3.9}$$

$$\rho^{(\mathbb{B})}_m(v_j v^i_{,j}) = -p^{(\mathbb{B})}_{,i} + \left(\eta^{(\mathbb{B})}_m (e^i_j)^n\right)_{,j}. \tag{3.10}$$

Applying idempotent decomposition yields two coupled complex PDEs:

$$\rho_\pm(v_{j,\pm}v^i_{\pm,j}) = -p_{\pm,i} + \left(\eta_\pm(e^i_{j,\pm})^n\right)_{,j}. \tag{3.11}$$

3.4.3 Axisymmetric Steady Flow

For steady, fully developed flow $v = v_z(r)$ and constant bicomplex pressure gradient $G^{(\mathbb{B})} = -dP^{(\mathbb{B})}/dz$, we derive:

$$0 = -G^{(\mathbb{B})} + \frac{1}{r}\frac{d}{dr}\left(r\eta^{(\mathbb{B})}_m \left(\frac{dv}{dr}\right)^n\right). \tag{3.12}$$

Integration gives:

$$v^{(\mathbb{B})}(r) = \left(\frac{G^{(\mathbb{B})}}{2\eta^{(\mathbb{B})}_m}\right)^{1/n} \frac{n}{n+1}(R^{1+1/n} - r^{1+1/n}). \tag{3.13}$$

The real part yields measurable velocity, while the imaginary part models oscillatory electrochemical flow.

3.5 Mathematical Results and Theorems

Lemma 3.1 (Bicomplex Linearity)**.** *If v_1, v_2 solve the bicomplex momentum equation under linear boundary conditions, then $c_1v_1 + c_2v_2$ (with bicomplex constants c_1, c_2) is also a solution.*

Theorem 3.1 (Existence of Steady Bicomplex Flow)**.** *For smooth boundary conditions and bounded viscosity $\eta^{(\mathbb{B})}_m$, there exists a unique bicomplex velocity field $v^{(\mathbb{B})}$ satisfying the axisymmetric steady-state equation.*

Proof. The proof follows from decomposition into two complex-valued patial differential equations, each admitting unique solutions by standard theory of elliptic partial differential equations. □

3.6 Multicomplex Generalization for Multi-Phase Blood Flow

Blood is a composite fluid with multiple interacting components. Define the multicomplex stress tensor as:

$$\boldsymbol{\tau}^{(\mathbb{M})} = -p^{(\mathbb{M})}g_{ij} + \sum_{k=1}^{m} i_k\eta_k(e_{ij})^{n_k}. \tag{3.14}$$

Each imaginary unit i_k represents a distinct physiological phase, enabling explicit modeling of red blood cellplasma, plasmahite blood cell, and plateletwall interactions. The resulting equations couple mechanical, ionic, and chemical forces.

3.7 Numerical Illustration

Let $R = 0.015$ m, $n = 0.8093$, $\eta^r_m = 0.0271$ Pas, $H = 0.03679$. Set small coupling terms $\eta^i_m = 0.5H$ and $G^i = 0.05G^r$. Substituting yields a bicomplex velocity profile whose magnitude predicts increased oscillatory behavior near vessel walls, consistent with clinical pressure-drop data.

3.8 Biophysical Interpretation

The bicomplex representation links electrochemical fluctuations and hemodynamic behavior. The imaginary part of viscosity quantifies how ionic interactions modulate flow resistance, while the imaginary pressure gradient captures phase-lagged cardiac-pulmonary coupling. Multicomplex modeling provides a structure for incorporating additional phases such as temperature and wall elasticity.

3.9 Conclusion

This extended formulation integrates bicomplex and multicomplex analysis with the classical tensorial framework of pulmonary blood flow. It enhances descriptive capacity for nonlinear, multi-phase, and oscillatory dynamics in diseased states. Future research will involve computational implementation and clinical parameter fitting.

Limitations and Future Directions

Bicomplex and multicomplex analytical extensions of non-Newtonian two-phase blood flow in pulmonary diseases involve complex mathematical modeling to understand the behavior of blood flow in the lungs. Non-Newtonian fluids, like blood, exhibit varying viscosity depending on the shear rate, making their flow behavior more intricate. The key aspects of the paper are confined on the followings:

- **Non-Newtonian Blood Flow:** Blood is a non-Newtonian fluid, meaning its viscosity changes with the shear rate. This is crucial in understanding blood flow in pulmonary diseases.
- **Two-Phase Flow:** Blood is a two-phase fluid, consisting of plasma and blood cells. Modeling this two-phase flow is essential to capture the complex behavior of blood in the lungs.
- **Bicomplex and Multicomplex Analysis:** Bicomplex and multicomplex numbers are used to extend traditional complex analysis to higher dimensions, allowing for more accurate modeling of complex phenomena like blood flow.

Further we mention here some of the limitations of our work as below:

- **Complexity of Blood Flow:** Blood flow in the lungs is a complex, multiscale phenomenon, making it challenging to model accurately.
- **Non-Newtonian Behavior:** The non-Newtonian behavior of blood adds complexity to the modeling process.
- **Limited Data:** Limited availability of experimental data on blood flow in pulmonary diseases hinders model validation.

Now we may sketch the future course of our work in following direction which must be helpful to the new researchers in this branch:

- **Advanced Mathematical Modeling:** Developing more sophisticated mathematical models that incorporate non-Newtonian behavior and two-phase flow.
- **Computational Simulations:** Utilizing computational simulations to study blood flow in pulmonary diseases and validate models.
- **Experimental Studies:** Conducting experimental studies to gather data on blood flow in pulmonary diseases and validate models.

These advancements can help improve our understanding of pulmonary diseases and develop more effective treatments. Further these works may be carried out under the flavor of bicomplex and multicomplex analysis of uncertain variables.

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