

Application of Navier-Stokes Equation in the Determination of the Velocity Profile of Constitutive Carrier Wave in a Visco-Elastic Blood Circulating System of HIV/AIDS Infected Person

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Abstract

The general understanding of the literature of clinical diseases is that the HIV attacks and feeds on the active cells that make up the human immune system. However, this process is associated with the gradual depletion and weakening of the active cells thereby resulting in a breakdown in the biological performance of the human system. This is a very correct statement but not a unique understanding. There is also a cause (vibration) that gives the HIV its own intrinsic characteristics, activity and existence. It is not the Human system that gives the HIV its life and existence, since the HIV itself is an active living organism and with its own peculiar wave characteristics even before it enters the system of Man. It is the vibration of an unknown force that causes life and existence. Therefore, for any active matter to exist it must possess vibration and vibration produces wave. The human heart stands as a transducer of this vibration. Fortunately, the blood stands as a means of conveying this vibration to all units of the human system. In this work, therefore, we primarily seek to determine the vibratory wave characteristics that are peculiar to both Man and the HIV. The spectrum of the velocity profile conveyed by the constituted carrier wave is characterised by the predominance of the radial velocity and with minimal contribution from the angular velocity. The characteristic spectrum of the velocities shows that within 21 hours after infection with HIV, the biological system of Man must imaginarily recognize and react to the interference of a strange velocity-like body whose influence may be destructive or constructive as the case may be. However, the recuperative inbuilt factor in the human system initially tends to annul this effect and renormalizes the frequency of vibration until it finally goes to zero after a specified time. It is also shown in this work that the radial and the angular velocity are oppositely related.

Key words: Newtonian, Navier-Stokes Equation, Constitutive Carrier Wave, Host Wave, Parasitic Wave, Raising Multiplier and HIV/AIDS.

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1.0. Introduction.

The role of Human-Immunodeficiency Virus (HIV) in the normal circulating blood system of man (host) has in general been poorly understood. However, its role in clinical disease has attracted increasing interest. Human immunodeficiency virus infection / acquired immunodeficiency syndrome (HIV/AIDS) is a disease of the human immune system caused by the human immunodeficiency virus (HIV). The human immunodeficiency virus (HIV) is among the most pressing health problem in the world today. Since its discovery, AIDS has caused nearly 30 million deaths as of 2009. It has been estimated that as of 2010 approximately 34 million people have contracted HIV globally and greater proportion of the population coming from Africa and Asian countries. AIDS is considered a pandemic—a disease outbreak which is present over a large area and is actively spreading [1, 2].

After the virus enters the body there is a period of rapid viral replication, leading to an abundance of virus in the peripheral blood. During primary infection, the level of HIV may reach several million virus particles per millilitre of blood. This response is accompanied by a marked drop in the number of circulating CD4⁺ T cells. The CD8⁺ T cell response is thought to be important in controlling virus levels, which peak and then decline, as the CD4⁺ T cell counts recover. A good CD8⁺ T cell response has been linked to slower disease progression and a better diagnosis, though it does not eliminate the virus [3, 4].

The HIV fatal effect stems from the attack on a person's CD4⁺ T cell counts. This attack of the HIV on the human system result to the progressive depletion of the CD4 cell counts which play a pivotal regulatory role in the immune response to infections and tumours. Ultimately, HIV causes AIDS by depleting CD4⁺T cells. This weakens the immune system and allows opportunistic infections. T cells are essential to the immune response and without them; the body cannot fight infections [5].

Two types of HIV have been characterized: HIV-1 and HIV-2. HIV-1 is the virus that was originally discovered. It is more virulent, more infective, and is the cause of the majority of HIV infections globally. The lower infectivity of HIV-2 as compared with HIV-1 implies that fewer people exposed to HIV-2 will be infected per exposure. Because of its relatively poor capacity for transmission, HIV-2 is largely confined to West Africa [6, 7].

In addition to the general understanding provided by the clinical literature about HIV/AIDS is that there is also a cause (vibration) that gives the HIV its own intrinsic characteristics, activity and existence. It is not the Human system that gives the HIV its life and existence, since the HIV itself is a living organism and with its own peculiar characteristics even before it entered the system of Man. It is the vibration of an unknown force that causes life and existence. Therefore, for any active matter to exist it must possess vibration. The human heart stands as a transducer of this vibration and fortunately the blood stands as a medium of conveying this vibration to all units of the human system.

Man and the Human-Immunodeficiency Virus (HIV) are both active matter, as a result, they must have independent peculiar vibrations in order to exist. It is the vibration of the HIV that interferes with the vibration of Man (host) in the blood circulating system after infection. The resultant interference of the vibration is parasitically destructive and it slows down or makes the biological system of Man to malfunction since the basic intrinsic parameters of the resident host wave have been altered.

The cyclic heart contraction generates pulsatile blood flow and latent vibration. The latent vibration is sinusoidal and central in character, that is, it flows along the middle of the vascular blood vessels. It orients the active particles of the blood and sets them into oscillating motion with a unified frequency as it passes. Elasticity of the vascular blood vessels supports pulsatile blood flow, connectivity network of the blood circulating system, and not the latent vibration. The human blood carries oxygen and food nutrients to nourish the human cells in the course of its flow from one region to another within the human micro-vascular blood circulating system. Any negative alteration to the normal flow of blood results to starvation and weakening of the human cells in the system. However, if this anomaly is not corrected after a period of time then a total loss of intended signal and a phenomenon called 'death' occurs and the affected system ceases to exist [8].

Blood Viscoelasticity is a property of human blood that is primarily due to the elastic energy that is stored in the deformation of red blood cells as the heart pumps the blood through the body. The energy transferred to the blood by the heart is partially stored in the elastic structure, another part is dissipated by viscosity, and the remaining energy is stored in the kinetic motion of the blood. When the pulsation of the heart is taken into account, an elastic regime becomes clearly evident. It has been shown that the previous concept of blood as a purely viscous fluid was inadequate since blood is not an ordinary fluid. Blood can more accurately be described as a fluidized suspension of elastic cells. Other factors



contributing to the viscoelastic properties of blood is the plasma viscosity, plasma composition, temperature, and the rate of flow or shear rate. Together, these factors make human blood viscoelastic, non-Newtonian, and thixotropic [9, 10].

In fluid dynamics, an incompressible fluid is a fluid whose density is constant. It is the same throughout space and it does not change through time. It is an idealization used to simplify analysis. In reality, all fluids are compressible to some extent. The linear Newtonian friction law is expected to hold for small rates of strain because higher powers of elasticity are neglected. However, for common fluids such as air and water the linear relationship is found to be surprisingly accurate for most applications. Some liquids important in the chemical industry, on the other hand, display non-Newtonian behaviour at moderate rates of strain [11, 12]. These include: (i) solutions containing polymer molecules, which have very large molecular weights and form long chains coiled together in spongy ball-like shapes that deform under shear; and (ii) emulsions and slurries containing suspended particles, two examples of which are blood and water containing clay. These liquids violate Newtonian behaviour in several ways—for example, shear stress is a *nonlinear* function of the local strain rate, it depends not only on the local strain rate, but also on its *history*. Such a “memory effect” gives the fluid an elastic property, in addition to its viscous property. Most non-Newtonian fluids are therefore viscoelastic [13].

The Navier-Stokes equations are always solved together with the continuity equation. The Navier-Stokes equations represent the conservation of momentum, while the continuity equation represents the conservation of mass. In that case, the density is assumed to be constant and the continuity equation reduces to divergence $\nabla \cdot u = 0$. The motion of a non-turbulent, Newtonian fluid is governed by the Navier-Stokes equation [14, 15]. The Navier-Stokes equations are only valid as long as the representative physical length scale of the system is much larger than the mean free path of the molecules that make up the fluid. In that case, the fluid is referred to as a *continuum*. The ratio of the mean free path, λ , and the representative length scale, L , is called the Knudsen number, $K_n = \lambda/L$. The Navier-Stokes equations govern the motion of fluids and can be seen as Newton's second law of motion for fluids. The Navier-Stokes equations are a set of nonlinear partial differential equations that describe the flow of fluids. They model weather, the movement of air in the atmosphere, ocean currents, water flow in a pipe, as well as many other fluid flows [16].

In this work, we primarily seek to determine the vibratory wave characteristics that are peculiar to both Man and the HIV, also to compare our present work of Navier-Stokes equation approach with the previous work where we used Newtonian mechanics approach [17]. In another development we also want to use the information regarding the independent vibrations to investigate how it affects the velocity profile of blood in the blood circulating system of HIV/AIDS infected person.

The organization of this paper is as follows. In section 1, we discuss the nature of wave and interference associated with fluid. In section 2, we show the mathematical theory of fluid dynamics and 2D Navier-Stokes equation. The results emanating from this study is shown in section 3. The discussion of the results of our study is presented in section 4. Conclusion of this work is presented in section 5. The paper is finally brought to an end by a few lists of references.

1.1 Research methodology

We first use simple algebra to derive the constitutive carrier wave which is the resultant of the superposition of the HIV vibration (parasitic wave) on the Human vibration (host wave). We also use the information regarding the independent vibrations to investigate how it affects the velocity and pressure of blood in the blood circulating system of HIV/AIDS infected person by using Navier-Stokes equation.

2.0. Mathematical theory and scientific research procedure.

- The activity of the HIV which is resident within the human system is parasitic in nature and the condition is synonymous with the coexistence of the parasitic plant Mistletoe (*Viscum album*) with another plant.
- The HIV transforms the latent vibration of the host to become equal to its own form and quality. The Mistletoe plant also transforms the vibratory characteristics of the host plant to its own form and quality.
- The HIV saps the energy and nutritive substances of the resident host vibration (host wave) the same way the Mistletoe plant saps the energy and nutritive substances of the resident host plant vibration.
- The HIV dies off the moment the resident host (Man) is dead since it does not have the requirements for independent sustenance and existence.



- The Mistletoe plant also withers and gradually dies off once the resident host plant is dead since there are no more nutritive substances left with the host plant to be sapped by the parasitic plant.
- If the wave function of any given active system is known, then its characteristics can be predicted and altered by means of anti-vibratory component.
- That the HIV kills slowly with time shows that the wave-functions of the HIV and that of the host were initially incoherent. As a result, the basic features of the Human vibration were initially greater than those of the HIV.
- The wave properties of HIV are independent of intrinsic variables such as the number, size, mass and of course mutation.
- Since the immune system of AIDS patient is almost zero, the measured wave function shall depend entirely on the vibrating property of the HIV only as every other active wave characteristics of the Human blood system would have been completely eroded.
- The wave characteristic of HIV infected candidate is the same everywhere within the resident host (Man). That is, irrespective of the occupation of the HIV in the host system, the activity is the same.
- The wave properties of HIV cannot be directly measured since it does not have its own independent existence outside the host system. As a result, the wave function of HIV can only be deductively measured.
- If HIV exists it must have its own peculiar vibration which must be independent of the vibration of the Human (host) system.
- The wave and vibrating characteristics of blood in the circulating system of a normal individual free from HIV/AIDS infection shall be assumed to be measured and the four independent variables following the observations about the wave recorded function are: (i) the amplitude, a (ii) the phase angle, ε (iii) the angular frequency, n and (iv) the wave number, k . Note that a , ε , n and k are assumed to be constant with time in a normal human system, except for some fluctuating factors, e.g. illness, which of course can only alter them slightly and temporarily.
- The wave and vibrating characteristics of blood in the circulating system of HIV/AIDS infected candidate, whose immune count rate is very low or almost zero is also assumed to be measured and the four independent variables following the observations of the recorded wave functions are: (i) the amplitude, b (ii) the phase angle, ε' (iii) the angular frequency, n' and (iv) the wave number, k' .
- Now, suppose we consider the wave function of the human vibration as the 'host wave' which can be described by the cosine sinusoidal function

$$y_1(\vec{r}, t) = a \beta \cos(\vec{k} \beta \cdot \vec{r} - n \beta t - \varepsilon \beta) \quad (2.1)$$

Where $\vec{k} = k_i + k_j$ and the position vector $\vec{r} = xi + yj$ are two dimensional (2D) vectors in Cartesian coordinate system and t is the time. Although, in polar coordinate system $x = r \cos \theta$ and $y = r \sin \theta$. The equation contains an inbuilt lowering or raising multiplier β which is capable of lowering or raising the intrinsic wave characteristics of the host wave, but the lowering is slightly and temporary during illness. The wave characteristics of the host wave renormalizes to its initial values after the illness.

- Also, suppose we consider the wave function of HIV vibration as the 'parasitic wave' which we can also described by the cosine sinusoidal function

$$y_2(\vec{r}, t) = b \lambda \cos(\vec{k}' \lambda \cdot \vec{r} - n' \lambda t - \varepsilon' \lambda) \quad (2.2)$$

As it is from the equation, the 'parasitic wave' has an inbuilt raising multiplier λ ($\lambda = 0, 1, 2, \dots, \lambda_{\max}$). The inbuilt multiplier is dimensionless and as the name implies, it has the ability of gradually raising the basic intrinsic parameters of the HIV 'parasitic wave' with time.



When equation (2.2) is superposed on equation (2.1) after some lengthy algebra we get a resultant wave equation given by,

$$y(\vec{r}, t) = y_1(\vec{r}, t) + y_2(\vec{r}, t) = a\beta \cos(\vec{k}\beta \cdot \vec{r} - n\beta t - \varepsilon\beta) + b\lambda \cos(\vec{k}'\lambda \cdot \vec{r} - n'\lambda t - \varepsilon'\lambda) \quad (2.3)$$

$$y = \sqrt{a^2\beta^2 + b^2\lambda^2 + 2a\beta b\lambda \cos((\beta\varepsilon - \varepsilon'\lambda) + (n\beta - n'\lambda)t)} \times \cos((\vec{k}\beta - \vec{k}'\lambda) \cdot \vec{r} - n\beta t - E) \quad (2.4)$$

$$E = \tan^{-1} \left(\frac{a\beta \sin \beta \varepsilon + b\lambda \sin(\varepsilon'\lambda - (n\beta - n'\lambda)t)}{a\beta \cos \beta \varepsilon + b\lambda \cos(\varepsilon'\lambda - (n\beta - n'\lambda)t)} \right) \quad (2.5)$$

Hence (2.4) is the resultant wave function which describes the superposition of the 'parasitic wave' on the 'host wave'. However, without loss of dimension we can recast (2.4) with subtractive terms as

$$y = \sqrt{(a^2\beta^2 - b^2\lambda^2) - 2(a\beta - b\lambda)^2 \cos((n\beta - n'\lambda)t - (\varepsilon\beta - \varepsilon'\lambda))} \cos(\vec{k}_c \cdot \vec{r} - (n\beta - n'\lambda)t - E) \quad (2.6)$$

Here the wave number and the position vector are 2D in character and respectively given as $\vec{k} = k_x i + k_y j$, $\vec{k}' = k'_x i + k'_y j$ and $\vec{r} = r(\cos \alpha i + \sin \alpha j)$, where $\alpha = (\varepsilon - \varepsilon'\lambda)$ and eventually $\vec{k}_c \cdot \vec{r} = (k\beta - k'\lambda) r(\cos \varphi + \sin \varphi)$, $\alpha = (\varepsilon\beta - \varepsilon'\lambda)$ where $k_x = k_y = k$. However, with the assumption that the effects of the resultant waves are subtractive and with the view that the basic parameters of the 'host wave' are constant with time, that is, $\beta=1$ and leave its variation for future study, then we get

$$y(\vec{r}, t) = \sqrt{(a^2 - b^2\lambda^2) - 2(a - b\lambda)^2 \cos((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \cos(\vec{k}_c \cdot \vec{r} - (n - n'\lambda)t - E(t)) \quad (2.7)$$

$$E(t) = \tan^{-1} \left(\frac{a \sin \varepsilon + b\lambda \sin(\varepsilon'\lambda - (n - n'\lambda)t)}{a \cos \varepsilon + b\lambda \cos(\varepsilon'\lambda - (n - n'\lambda)t)} \right) \quad (2.8)$$

Equation (2.7) is regarded as the **constitutive carrier wave (CCW)** necessary for our study. It is the equation that governs the dynamical behaviour of the coexistence of the HIV parasite in the human micro-vascular blood circulating system. It is obvious and readable from the equation that once the dynamic constituent parameters of the HIV parasite become equal to those of the Man the host, as a result of the raising multiplier λ , then the CCW goes to zero and the resident host (Man) ceases to exist. As the equation stands, it is a corrupt wave function, in which it is only the variation in the intrinsic parameters of the 'parasitic wave' that determines the life span of the physically active system which it describes. This equation describes a propagating carrier wave with non-stationary and frequency dependent amplitude modulated by a spatial oscillating cosine function. Thus, the reliability and the life span of most active systems are determined by the reluctance and willingness of the active components of the 'host wave' to the destructive influence of the 'parasitic wave'.

- The wave mechanics of HIV in the Human Blood circulating system is two dimensional 2D in character since it is a transverse wave, the position vector of the whole blood (particles and fluid) in motion can be represented as $\vec{r} = r(\cos \theta i + \sin \theta j)$ and hence the motion is constant with respect to the z -axis. $\vec{k}_c = (k - k'\lambda) i + (k - k'\lambda) j$.

- While on interpretation $\vec{k}_c \cdot \vec{r} = (k - k'\lambda) r(\cos \varphi + \sin \varphi)$ is the coordinate of two dimensional (2D) position vector, $\theta = \pi - (\varepsilon - \varepsilon'\lambda)$, the total phase angle of the CCW is represented by $E(t)$. By definition: $(n - n'\lambda)$ is the modulation angular frequency, the modulation propagation constant is $(k - k'\lambda)$, the phase difference δ between the two interfering waves is $(\varepsilon - \varepsilon'\lambda)$, and of course we have that the interference term is $2(a - b\lambda)^2 \cos((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda))$, while waves out of phase interfere destructively according to $(a - b\lambda)^2$, however, waves in-phase interfere constructively according to $(a + b\lambda)^2$.



- Driving forces in anti-phase ($\varepsilon - \varepsilon' = \pm\pi$) provide full destructive superposition and the minimum possible amplitude; driving forces in phase ($\varepsilon = \varepsilon'$) provides full constructive superposition and maximum possible amplitude.

The total phase angle of the CCW given by (2.8) is not constant with time. The variation as a function of time is

$$\frac{dE(t)}{dt} = \left(1 + \left(\frac{a \sin \varepsilon - b\lambda \sin((n - n'\lambda)t - \varepsilon'\lambda)}{a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda)} \right)^2 \right)^{-1} \times \frac{d}{dt} \left(\frac{a \sin \varepsilon - b\lambda \sin((n - n'\lambda)t - \varepsilon'\lambda)}{a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda)} \right) \quad (2.9)$$

$$\frac{dE(t)}{dt} = \left\{ \frac{(a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda))^2}{(a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda))^2 + (a \sin \varepsilon - b\lambda \sin((n - n'\lambda)t - \varepsilon'\lambda))^2} \right\} \times$$

$$\frac{d}{dt} \left(\frac{a \sin \varepsilon - b\lambda \sin((n - n'\lambda)t - \varepsilon'\lambda)}{a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda)} \right) \quad (2.10)$$

$$\frac{dE(t)}{dt} = -Z(t) \quad (2.11)$$

where we have introduced a new variable defined by the symbol $Z(t)$ as

$$Z(t) = (n - n'\lambda) \left(\frac{b^2\lambda^2 + ab\lambda \cos((n - n'\lambda)t + (\varepsilon - \varepsilon'\lambda))}{a^2 + b^2\lambda^2 + 2ab\lambda \cos((n - n'\lambda)t + (\varepsilon - \varepsilon'\lambda))} \right) \quad (2.12)$$

This is the characteristic angular velocity of the constitutive carrier wave. It has the dimension of $rad./s$. Note that we used the trigonometric identity $\cos(x \pm y) = \cos x \cos y \mp \sin x \sin y$ to reduce the work. Also, for the purpose of further application we may also determine the variation of the characteristic angular velocity of the constitutive carrier wave $Z(t)$ with respect to time. Thus

$$\frac{dZ(t)}{dt} = Q(t) = \frac{(n - n'\lambda)^2 (ab^3\lambda^3 - a^3b\lambda) \sin((n - n'\lambda)t + (\varepsilon - \varepsilon'\lambda))}{\left(a^2 + b^2\lambda^2 + 2ab\lambda \cos((n - n'\lambda)t + (\varepsilon - \varepsilon'\lambda)) \right)^2} \quad (2.13)$$

Hence $Q(t)$ is the characteristic group frequency of the CCW and it has the dimension of rad/s^2 .

2.1. The Pressure-force law obeyed by the CCW in 3D Navier – Stokes Equation.

The Navier-Stokes equation can be viewed as an application of Newton's second law, $f = ma$, which states that force is the product of the mass of an object times its acceleration. (Note, we will now be using f to represent forces, not scalar or vector fields). The Navier-Stokes equations are the fundamental partial differentials equations that describe the flow of incompressible fluids. Using the rate of stress and rate of strain tensors, it can be shown that the components of a viscous force f in a nonrotating frame are given by the Navier-Stokes equation

$$\rho \left(\frac{\partial u}{\partial t} + u \cdot \nabla u \right) = \nabla \cdot \sigma + f \quad (2.14)$$

where ρ denotes the density of the fluid and is equivalent to mass, $\frac{\partial u}{\partial t} + u \cdot \nabla u$ is the acceleration and u is velocity, and $\nabla \cdot \sigma + f$ is the total force, with $\nabla \cdot \sigma$ being the shear stress and f being all other forces. The Navier-Stokes equations are always solved together with the continuity equation:



$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = 0 \quad (2.15)$$

Now consider the irrotational Navier-Stokes equations in particular coordinate systems. In Cartesian coordinates with the components of the velocity vector given by $\mathbf{u} = (u, v, w)$, the continuity equation is

$$\left(\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} + \frac{\partial w}{\partial z} \right) = 0 \quad (2.16)$$

In cylindrical coordinates with the components of the velocity vector given by $\mathbf{u} = (u_r, u_\phi, u_z)$, the continuity equation is

$$\left(\frac{\partial U_r}{\partial r} + \frac{U_r}{r} + \frac{1}{r} \frac{\partial U_\phi}{\partial \phi} + \frac{\partial U_z}{\partial z} \right) = 0 \quad (2.17)$$

In spherical coordinates with the components of the velocity vector given by $\mathbf{u} = (u_r, u_\theta, u_\phi)$, the continuity equation is

$$\left(\frac{\partial U_r}{\partial r} + \frac{2U_r}{r} + \frac{1}{r} \frac{\partial U_\theta}{\partial \theta} + \frac{U_\theta \cot \theta}{r} + \frac{1}{r \sin \theta} \frac{\partial U_\phi}{\partial \phi} \right) = 0 \quad (2.18)$$

We may also write equation (2.13) as

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u} \cdot \nabla \mathbf{u} \right) = -\nabla p + \eta \nabla^2 \mathbf{u} + \mathbf{f} \quad (2.19)$$

where p is pressure and η is dynamic viscosity. *Viscosity* is defined as the measure of the resistance of a fluid which is being deformed by the shear stress. The different terms correspond to: (i) the inertial forces (ii), pressure forces (iii), viscous forces and (iv), external forces applied to the fluid. The Navier-Stokes equations were derived by Navier, Poisson, Saint-Venant, and Stokes between 1827 and 1845. In unidirectional flows such as blood, all nonlinear terms in the Navier-Stokes equations vanish: the convective term $\mathbf{u} \cdot \nabla \mathbf{u} = 0$ [15, 16]. Navier-Stokes explicitly models changes in the directional velocity using four components: (i) The first of these is $-(\mathbf{u} \cdot \nabla) \cdot \mathbf{u}$, which is the divergence on a velocity, or in simpler terms, it is how the divergence affects the velocity. (ii) The factor $-\nabla p$ may be thought of as how the particles move as pressure changes, specifically, the tendency to move away from areas of higher pressure. (iii) Next we consider the term $\eta \nabla^2 \mathbf{u}$. The two key parts are viscosity (η) and Laplacian ∇^2 . It may be a little hard to make sense of this part, but think of it as the difference between what a particle does and what its neighbours do. (iv) finally we have \mathbf{f} , which again, is any other forces acting on the substance.

Here, u and p are the time-averaged velocity and pressure respectively. However, in this work we shall only focus on the cylindrical coordinate system since we assume that the Human blood vessels have a similar geometry with that of the cylinder. Now in cylindrical coordinate system, the radial pressure and the angular pressure in Navier-Stokes representation are respectively given by

$$\rho \left\{ \frac{\partial U_r}{\partial t} + \left(U_r \frac{\partial U_r}{\partial r} + \frac{U_\phi}{r} \frac{\partial U_r}{\partial \phi} + U_z \frac{\partial U_r}{\partial z} - \frac{U_\phi^2}{r} \right) \right\} = -\frac{\partial P}{\partial r} + \eta \left(\frac{\partial^2 U_r}{\partial r^2} + \frac{1}{r} \frac{\partial U_r}{\partial r} + \frac{1}{r^2} \frac{\partial^2 U_r}{\partial \phi^2} - \frac{U_r}{r^2} + \frac{\partial^2 U_r}{\partial z^2} - \frac{2}{r^2} \frac{\partial U_\phi}{\partial \phi} \right) + F_r \quad (2.20)$$

$$\rho \left\{ \frac{\partial U_\phi}{\partial t} + \left(U_r \frac{\partial U_\phi}{\partial r} + \frac{U_r U_\phi}{r} + \frac{U_\phi}{r} \frac{\partial U_\phi}{\partial \phi} + U_z \frac{\partial U_\phi}{\partial z} \right) \right\} = -\frac{1}{r} \frac{\partial P}{\partial \phi} +$$



$$\eta \left(\frac{\partial^2 U_\varphi}{\partial r^2} + \frac{1}{r} \frac{\partial U_\varphi}{\partial r} - \frac{U_\varphi}{r^2} + \frac{1}{r^2} \frac{\partial^2 U_\varphi}{\partial \varphi^2} + \frac{\partial^2 U_\varphi}{\partial z^2} + \frac{2}{r^2} \frac{\partial U_r}{\partial \varphi} \right) + F_\varphi \quad (2.21)$$

Since our work is restricted to 2D we have to ignore the z - axes or assume that the motion of the CCW is constant with respect to the z - axes. We also take the body forces $F_r = F_\varphi = 0$. Consequent upon this the Navier-Stokes equation becomes

$$\rho \left(\frac{\partial u}{\partial t} \right) = -\nabla p + \eta \nabla^2 u + f \quad (2.22)$$

Both equations (2.20) and (2.21) eventually reduces to

$$P_r = \frac{\partial P}{\partial r} = \eta \left(\frac{\partial^2 U_r}{\partial r^2} + \frac{1}{r} \frac{\partial U_r}{\partial r} + \frac{1}{r^2} \frac{\partial^2 U_r}{\partial \varphi^2} - \frac{U_r}{r^2} - \frac{2}{r^2} \frac{\partial U_\varphi}{\partial \varphi} \right) - \rho \left(\frac{\partial U_r}{\partial t} \right) \quad (2.23)$$

$$P_\varphi = \frac{\partial P}{\partial \varphi} = \eta \left(r \frac{\partial^2 U_\varphi}{\partial r^2} + \frac{\partial U_\varphi}{\partial r} - \frac{U_\varphi}{r} + \frac{1}{r} \frac{\partial^2 U_\varphi}{\partial \varphi^2} + \frac{2}{r} \frac{\partial U_r}{\partial \varphi} \right) - \rho \left(r \frac{\partial U_\varphi}{\partial t} \right) \quad (2.24)$$

Thus the bulk pressure of the CCW as it propagates in the human micro-vascular blood circulating system is the addition of the radial pressure and the angular pressure. Hence

$$\begin{aligned} \nabla P = \left(\frac{\partial P}{\partial r} + \frac{\partial P}{\partial \varphi} \right) &= \eta \left(\frac{1}{r} \frac{\partial U_r}{\partial r} + \frac{\partial^2 U_r}{\partial r^2} + \frac{1}{r^2} \frac{\partial^2 U_r}{\partial \varphi^2} - \frac{U_r}{r^2} - \frac{2}{r^2} \frac{\partial U_\varphi}{\partial \varphi} + r \frac{\partial^2 U_\varphi}{\partial r^2} + \frac{\partial U_\varphi}{\partial r} - \frac{U_\varphi}{r} + \right. \\ &\left. \frac{1}{r} \frac{\partial^2 U_\varphi}{\partial \varphi^2} + \frac{2}{r} \frac{\partial U_r}{\partial \varphi} \right) - \rho \left(\frac{\partial U_r}{\partial t} + r \frac{\partial U_\varphi}{\partial t} \right) \end{aligned} \quad (2.25)$$

The reader should note that we have ignored the terms in the brackets of the left hand side of (2.20) and (2.21) since they are equal to zero base on the continuity equation of (2.16) as compare with (2.14). Hence we have two independent pressure gradients, the radial pressure gradient and the angular pressure gradient associated by the propagation of the CCW in the Human micro-vascular blood circulating system. However, if the pressure gradients are zero then $\nabla P = 0$ and (2.25) becomes

$$\begin{aligned} \eta \left(\frac{1}{r} \frac{\partial U_r}{\partial r} + \frac{\partial^2 U_r}{\partial r^2} + \frac{1}{r^2} \frac{\partial^2 U_r}{\partial \varphi^2} - \frac{U_r}{r^2} - \frac{2}{r^2} \frac{\partial U_\varphi}{\partial \varphi} + r \frac{\partial^2 U_\varphi}{\partial r^2} + \frac{\partial U_\varphi}{\partial r} - \frac{U_\varphi}{r} + \frac{1}{r} \frac{\partial^2 U_\varphi}{\partial \varphi^2} + \frac{2}{r} \frac{\partial U_r}{\partial \varphi} \right) - \\ \rho \left(\frac{\partial U_r}{\partial t} + r \frac{\partial U_\varphi}{\partial t} \right) = 0 \end{aligned} \quad (2.26)$$

2.2 Determination of the bulk velocity U , radial velocity U_r and the angular velocity U_φ of the CCW.

The bulk velocity is related to the constitutive carrier wave according to the equation below.

$$\begin{aligned} U = \frac{\partial y}{\partial t} = \frac{(n-n'\lambda)(a-b\lambda)^2 \sin((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda)) \cos((k-k'\lambda)r(\cos\varphi + \sin\varphi) - (n-n'\lambda)t - E(t))}{\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}} + \\ \sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \times \\ ((n-n'\lambda) - Z(t)) \sin((k-k'\lambda)r(\cos\varphi + \sin\varphi) - (n-n'\lambda)t - E(t)) \end{aligned} \quad (2.27)$$



The radial velocity and the angular velocity of the CCW as it propagates in the micro-vascular blood circulating system are given by the equation below.

$$U = U(r, \varphi): dU = \frac{\partial U}{\partial r} dr + \frac{\partial U}{\partial \varphi} d\varphi : dU = U_r dr + U_\varphi d\varphi \quad (2.28)$$

$$U_r = \frac{\partial U}{\partial r} = - \frac{(n-n'\lambda)(a-b\lambda)^2(k-k'\lambda) \sin((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}{\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}} \times$$

$$(\cos \varphi + \sin \varphi) \sin((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) +$$

$$\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \times ((n-n'\lambda) - Z(t)) (k-k'\lambda) (\cos \varphi + \sin \varphi) \times$$

$$\cos((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) \quad (2.29)$$

$$U_\varphi = \frac{\partial U}{\partial \varphi} = - \frac{(n-n'\lambda)(a-b\lambda)^2(k-k'\lambda) \sin((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}{\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}} \times$$

$$r(\cos \varphi - \sin \varphi) \sin((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) +$$

$$\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \times ((n-n'\lambda) - Z(t)) (k-k'\lambda)r(\cos \varphi - \sin \varphi) \times$$

$$\cos((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) \quad (2.30)$$

The radial velocity and the angular velocity of the CCW as it propagates in the micro-vascular blood system are related to each other according to the equation below. That is the radial and angular velocities components of the CCW are related to each other by the stream-function given below.

$$U_r = \frac{1}{r} \frac{\partial U}{\partial \varphi} ; U_\varphi = - \frac{\partial U}{\partial r} \quad (2.31)$$

$$U_r = - \frac{(n-n'\lambda)(a-b\lambda)^2(k-k'\lambda) \sin((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}{\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}} \times$$

$$(\cos \varphi - \sin \varphi) \sin((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) +$$

$$\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \times ((n-n'\lambda) - Z(t)) (k-k'\lambda) \times$$

$$(\cos \varphi - \sin \varphi) \cos((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) \quad (2.32)$$

Thus radial velocity of the constitutive carrier wave U_r has a unit of rad/s . Also

$$U_\varphi = \frac{(n-n'\lambda)(a-b\lambda)^2(k-k'\lambda) \sin((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}{\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))}} \times$$

$$(\cos \varphi + \sin \varphi) \sin((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) -$$

$$\sqrt{(a^2 - b^2\lambda^2) - 2(a-b\lambda)^2 \cos((n-n'\lambda)t - (\varepsilon - \varepsilon'\lambda))} \times ((n-n'\lambda) - Z(t)) (k-k'\lambda) \times$$

$$(\cos \varphi + \sin \varphi) \times \cos((k-k'\lambda)r(\cos \varphi + \sin \varphi) - (n-n'\lambda)t - E(t)) \quad (2.33)$$



Thus angular velocity of the constitutive carrier wave U_φ has a unit of rad/s .

2.3. Determination of the latent wave characteristics Man (Host) (a, n, ε, k) contained in the CCW.

Now let us subject the constitutive carrier wave given by equation (2.7) to the 2D constant velocity-pressure gradient of Navier-Stokes equation given by (2.26). The differential equations we have derived for the conservation laws are subject to boundary conditions in order to properly formulate any problem. After a careful isolation and substitution of the various derivatives of U with respect to r and φ into the combined equation of the radial and the angular pressure given by (2.26), the resulting equation can be further simplified and rearranged by eliminating equal and oppositely related terms. However, we have also imposed boundary conditions to reduce the complexity of the resulting equation.

The boundary conditions require that at, $t = 0, \lambda = 0$, for even and symmetric function $\cos(-\varepsilon) = \cos(\varepsilon)$ while for odd and anti-symmetric function $\sin(-\varepsilon) = -\sin(\varepsilon), \varphi = 0, E(t) = \varepsilon, Z(t) = Q(t) = 0$.

$$\begin{aligned}
 & \eta \left\{ -\frac{2na k^3 \sin(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} - 2[1 - 2\cos(\varepsilon)]^{1/2} n a k^3 \cos(kr - \varepsilon) - \frac{4[1 - 2\cos(\varepsilon)]^{1/2} n a k \cos(kr - \varepsilon)}{r^2} + \right. \\
 & \left. \frac{n a k^2 \sin(\varepsilon) \cos(kr - \varepsilon)}{r |1 - 2\cos(\varepsilon)|^{1/2}} + \frac{4n a k^2 [1 - 2\cos(\varepsilon)]^{1/2} \sin(kr - \varepsilon)}{r} + \frac{2n a k^3 r \sin(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} - \right. \\
 & \left. 3n a k^2 [1 - 2\cos(\varepsilon)]^{1/2} \sin(kr - \varepsilon) - \frac{n a k [1 - 2\cos(\varepsilon)]^{1/2} \cos(kr - \varepsilon)}{r} \right\} - \\
 & \rho \left\{ \frac{n^2 a k \sin^2(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{3/2}} - \frac{n^2 a k \cos(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} - \frac{2n^2 a k \sin(\varepsilon) \cos(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} + \right. \\
 & \left. n^2 a k [1 - 2\cos(\varepsilon)]^{1/2} \sin(kr - \varepsilon) + \frac{n^2 a k r \cos(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} - \frac{n^2 a k r \sin^2(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{3/2}} + \right. \\
 & \left. \frac{2n^2 a k r \sin(\varepsilon) \cos(kr - \varepsilon)}{|1 - 2\cos(\varepsilon)|^{1/2}} + n^2 a k r [1 - 2\cos(\varepsilon)]^{1/2} \sin(kr - \varepsilon) \right\} = 0 \tag{2.34}
 \end{aligned}$$

To make our work easy we may need to adopt the "third world approximation" to linearize (2.34) by removing the term $[1 - 2\cos(\varepsilon)]^{\pm n}$. The "third world approximations are the differential minimization of the resulting binomial expansion of a given variable function. The approximations have the advantage of converging results easily and also producing expected minimum value of results. Now the 'third world approximation' states that

$$(1 + \xi f(\phi))^{\pm n} = \frac{d}{d\phi} \left(1 + n\xi f(\phi) + \frac{n(n-1)}{2!} (\xi f(\phi))^2 + \frac{n(n-1)(n-2)}{3!} (\xi f(\phi))^3 + \dots \right) - n \frac{d}{d\phi} (\xi f(\phi)) \tag{2.35}$$

$$[1 - 2\cos(\varepsilon)]^{-3/2} = -15 \sin \varepsilon \cos \varepsilon \tag{2.36}$$

$$[1 - 2\cos(\varepsilon)]^{-1/2} = -3 \sin \varepsilon \cos \varepsilon \tag{2.37}$$

$$[1 - 2\cos(\varepsilon)]^{1/2} = \sin \varepsilon \cos \varepsilon \tag{2.38}$$

When equation (2.36), (2.37) and (2.38) are now substituted into (2.34) and after rearrangement we get



$$\begin{aligned}
& \eta \left\{ 6 n a k^3 r^2 \sin^2 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - 2 n a k^3 r^2 \sin \varepsilon \cos \varepsilon \cos(kr - \varepsilon) - \right. \\
& 4 n a k \sin \varepsilon \cos \varepsilon \cos(kr - \varepsilon) - 3 n a k^2 r \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + \\
& 4 n a k^2 r \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - 6 n a k^3 r^3 \sin^2 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - \\
& \left. 3 n a k^2 r^2 \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - n a k r \sin \varepsilon \cos \varepsilon \cos(kr - \varepsilon) \right\} - \\
& \rho \left\{ -15 n^2 a k r^2 \sin^3 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) + 3 n^2 a k r^2 \sin \varepsilon \cos^2 \varepsilon \sin(kr - \varepsilon) + \right. \\
& 6 n^2 a k r^2 \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + n^2 a k r^2 \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) + \\
& 3 n^2 a k r^3 \sin(\varepsilon) \cos^2(\varepsilon) \sin(kr - \varepsilon) + 15 n^2 a k r^3 \sin^3 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - \\
& \left. 6 n^2 a k r^3 \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + n^2 a k r^3 \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) \right\} = 0 \tag{2.39}
\end{aligned}$$

❖

Calculation of the phase angle (ε) of the latent

Human vibration (host wave)

From the clinical literature, blood leaves the Human heart at a rate of about 5 litres per minute (0.08333 litres per second or 0.00008333 cubic meters per second) since $1 m^3 = 1000 \text{ litres}$. Also, from clinical literature, it is given that the cross-sectional area A ($A = \pi r^2$) of the Human Aorta is about $3 - 5 \text{ cm}^2$. In this work, we used the maximum value of the radius from the given range which is 5 cm^2 (0.0005 m^2). Now from these data $r = 0.01262 \text{ m}$ where r is the radius of the Human Aorta assumed to be a circular cylinder. Now, we know from the clinical literature that the elasticity σ of the Human blood is domicile in the red blood cells. Therefore, the quantity or volume of blood that leaves the Human heart can be found from the equation below.

$$Quantity \text{ (volume)} = rate \text{ (m}^3/s) \times time \text{ (s)} \tag{2.40}$$

$$Quantity \text{ (volume)} = 0.000083333 \text{ m}^3/s \times 1 \text{ s} = 8.333 \times 10^{-5} \text{ m}^3 \tag{2.41}$$

Thus, the Human heart pumps a volume of $8.333 \times 10^{-5} \text{ m}^3$ or 8.333×10^{-5} cubic meter of blood per second. Now there are several possible ways of determining the wave characteristics of the host vibration, although, the results obtained may also be different. However, the careful choice we make must be relevant and applicable to the problem under study. Suppose we select our choice from the first, second and the forth terms of the equation with coefficient ρ then we get

$$\left(-15 n^2 a k r^2 \sin^3 \varepsilon \cos \varepsilon + 3 n^2 a k r^2 \sin \varepsilon \cos^2 \varepsilon + n^2 a k r^2 \sin \varepsilon \cos \varepsilon \right) \tan(kr - \varepsilon) = 0 \tag{2.42}$$

$$\left(15 \sin^2 \varepsilon - 3 \cos \varepsilon - 1 \right) \tan(kr - \varepsilon) = 0 \tag{2.43}$$

$$\left(15 \varepsilon^2 - 3 \left(1 - \frac{\varepsilon^2}{2} \right) \right) = 1 \tag{2.44}$$

$$\varepsilon = 0.4924 \text{ rad.} \tag{2.45}$$

Where we have used the fact that at the critical point, which is at time $t = 0$ the critical value of any time dependent variable at the origin is given by



$$\sin \theta \cong \theta ; \cos \theta \cong \left(1 - \frac{\theta^2}{2} \right); \tan \theta \cong \theta \quad (2.46)$$

❖ **latent Human vibration (host wave).**

Calculation of the spatial frequency (k) of the

Now to determine the spatial frequency or the wave number of the host vibration we can also combine the first four terms of the coefficient of ρ in equation (2.39).

$$\begin{aligned} -15n^2 a k r^2 \sin^3 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) + 3n^2 a k r^2 \sin \varepsilon \cos^2 \varepsilon \sin(kr - \varepsilon) + \\ 6n^2 a k r^2 \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + n^2 a k r^2 \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) = 0 \end{aligned} \quad (2.47)$$

$$-15 \sin^3 \varepsilon \cos \varepsilon \tan(kr - \varepsilon) + 3 \sin \varepsilon \cos^2 \varepsilon \tan(kr - \varepsilon) + 6 \sin^2 \varepsilon \cos \varepsilon + \sin \varepsilon \cos \varepsilon \tan(kr - \varepsilon) = 0 \quad (2.48)$$

$$\left(15 \sin^2 \varepsilon - 3 \cos \varepsilon - 1 \right) \tan(kr - \varepsilon) = 6 \sin \varepsilon \quad (2.49)$$

$$\left(15 \varepsilon^2 - 3 \left(1 - \frac{\varepsilon^2}{2} \right) - 1 \right) \tan(kr - \varepsilon) = 6 \sin \varepsilon \quad (2.50)$$

$$\left(30 \varepsilon^2 - 6 + 3\varepsilon^2 - 2 \right) \tan(kr - \varepsilon) = 12 \sin \varepsilon \quad (2.51)$$

$$\tan(kr - \varepsilon) = \frac{12(0.4924)}{33(0.4924)^2 - 8} = \frac{5.9088}{0.00110608} = 5342.1091 \quad (2.52)$$

$$(kr - \varepsilon) = \tan^{-1}(5342.1091) = 1.570609 \quad (2.53)$$

$$k = 163.4714 \text{ rad./m} \quad (2.54)$$

❖ **latent Human vibration (host wave)**

Calculation of the angular frequency (n) of the

In other to calculate the angular frequency or the angular velocity of the host wave we select the first two terms from coefficient of η (viscosity of blood) and the first four terms from the coefficient of ρ (density of blood).

$$\begin{aligned} \eta \left(6 n a k^3 r^2 \sin^2 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) - 2 n a k^3 r^2 \sin \varepsilon \cos \varepsilon \cos(kr - \varepsilon) \right) = \\ \rho \left(-15 n^2 a k r^2 \sin^3 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) + 3 n^2 a k r^2 \sin \varepsilon \cos^2 \varepsilon \sin(kr - \varepsilon) + \right. \\ \left. 6 n^2 a k r^2 \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + n^2 a k r^2 \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) \right) \end{aligned} \quad (2.55)$$

$$\begin{aligned} \eta \left(6 k^2 \sin \varepsilon \tan(kr - \varepsilon) - 2 k^2 \right) = \rho \left(-15 n \sin^2 \varepsilon \tan(kr - \varepsilon) + 3 n \cos \varepsilon \tan(kr - \varepsilon) + 6 n \sin \varepsilon + \right. \\ \left. n \tan(kr - \varepsilon) \right) \end{aligned} \quad (2.56)$$



$$n = \frac{\eta(6k^2 \sin \varepsilon \tan(kr - \varepsilon) - 2k^2)}{\rho(-15 \sin^2 \varepsilon \tan(kr - \varepsilon) + 3 \cos \varepsilon \tan(kr - \varepsilon) + 6 \sin \varepsilon + \tan(kr - \varepsilon))} \quad (2.57)$$

$$n = \frac{\eta(6 \sin \varepsilon \tan(kr - \varepsilon) - 2)k^2}{\rho(-15 \sin^2 \varepsilon \tan(kr - \varepsilon) + 3 \cos \varepsilon \tan(kr - \varepsilon) + 6 \sin \varepsilon + \tan(kr - \varepsilon))} \quad (2.58)$$

$$n = \frac{\eta(2.6401806)k^2}{\rho(2.95352)} = \frac{0.004 \text{ kg/ms} \times (2.6401806) \times (163.4714)^2 \text{ rad./m}^2}{1060 \text{ kg/m}^3 (2.95352)} = 0.09014 \text{ rad./s} \quad (2.59)$$

❖

Calculation of the amplitude (a) of the latent

Human vibration (host wave).

It is not very possible to calculate the amplitude or the maximum displacement a of the host wave from the available equation (2.39). As a result, we are going to use a slightly different approach to calculate it. Now, the radial acceleration has a unit of rad./s^2 and the angular acceleration has a unit of m/s^2 . These two concepts can be verified from the density ρ part of (2.39) respectively so that the units are at variant with one another. However, we are going to calculate the amplitude from the radial acceleration because the direction of flow of blood is along the radius of the blood vessels. Now, if we multiply the radial acceleration by mass m then the result is radial force which produces a change in the motion of the CCW along the radius of the cylindrical blood vessels. The radial force will cause a change in the elasticity μ of the blood which is stored in the red blood cell. Accordingly, we shall select the first four terms in the coefficient of ρ (density) in (2.39) that has no radial term so that the equation becomes

$$m \left(\frac{\partial U_r}{\partial t} \right) = \mu \quad (2.60)$$

$$m \left\{ \frac{n^2 a k \sin^2(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2 \cos(\varepsilon)|^{3/2}} - \frac{n^2 a k \cos(\varepsilon) \sin(kr - \varepsilon)}{|1 - 2 \cos(\varepsilon)|^{1/2}} + n^2 a k [1 - 2 \cos(\varepsilon)]^{1/2} \sin(kr - \varepsilon) - \frac{2n^2 a k \sin(\varepsilon) \cos(kr - \varepsilon)}{|1 - 2 \cos(\varepsilon)|^{1/2}} \right\} = \mu \quad (2.61)$$

$$m \left\{ -15n^2 a k \sin^3 \varepsilon \cos \varepsilon \sin(kr - \varepsilon) + 3n^2 a k \sin \varepsilon \cos^2 \varepsilon \sin(kr - \varepsilon) + 6n^2 a k \sin^2 \varepsilon \cos \varepsilon \cos(kr - \varepsilon) + n^2 a k \sin \varepsilon \cos \varepsilon \sin(kr - \varepsilon) \right\} = \mu \quad (2.62)$$

$$m \left\{ -15 \sin^3(0.4924) \cos(0.4924) \sin(1.5706) + 3 \sin(0.4924) \cos^2(0.4924) \sin(1.5706) + 6 \sin^2(0.4924) \cos(0.4924) \cos(1.5706) + \sin(0.4924) \cos(0.4924) \sin(1.5706) \right\} n^2 a k = \mu \quad (2.63)$$

$$m \left\{ -2.47164 + 1.79166 + 1.123408 + 0.679608 \right\} n^2 a k = \mu \quad (2.64)$$

$$a = \frac{\mu}{mn^2 k (1.123036)} = \frac{\mu}{\rho V n^2 k (1.123036)} \quad (2.65)$$

$$a = \frac{6.92 \times 10^{-7} \text{ kg s}^{-2}}{1060 \text{ kg m}^{-3} \times 8.333 \times 10^{-5} \text{ m}^3 \times (0.09014)^2 \text{ s}^{-2} \times 163.4714 \text{ m}^{-1} \times (1.123036)}$$



$$a = 5.252 \times 10^{-6} m \quad (2.66)$$

The reader should note that we have also used the critical value equations as stipulated by (2.46).

2.4. Determination of the latent wave characteristics HIV/AIDS (b, n', ε', k') and the raising multiplier

λ contained in the CCW.

Let us now determine the basic parameters of the 'parasitic wave' which were initially not known before the interference from the calculated values of the resident 'host wave' using the below method. We can do this by understanding that the gradual depletion in the physical vibrating parameters of the Host system would mean that after a sufficiently long period of time all the active constituents of the resident 'host wave' would have been completely attenuated and the residual of the constituents CCW is the predominance of the destructive influence of the 'parasitic wave'. On the basis of these arguments, we can now write as follows.

$$\left. \begin{aligned} a - b\lambda = 0 &\Rightarrow 5.252 \times 10^{-6} = b\lambda \\ n - n'\lambda = 0 &\Rightarrow 0.09014 = n'\lambda \\ \varepsilon - \varepsilon'\lambda = 0 &\Rightarrow 0.4924 = \varepsilon'\lambda \\ k - k'\lambda = 0 &\Rightarrow 163.4714 = k'\lambda \end{aligned} \right\} \quad (2.67)$$

Upon dividing the sets of relations in (2.67) with one another with the view to eliminating λ we get

$$\left. \begin{aligned} 5.826492 \times 10^{-5} n' &= b \\ 1.066612 \times 10^{-5} \varepsilon' &= b \\ 3.21279 \times 10^{-8} k' &= b \\ 0.1830625 \varepsilon' &= n' \\ 0.00055141 k' &= n' \\ 0.00301214 k' &= \varepsilon' \end{aligned} \right\} \quad (2.68)$$

Suppose we equate the forth and the fifth terms of (2.68), then based on simple proportion rule it be shown that

$$k' = 0.1831 \text{ rad./m} : \varepsilon' = 0.0005514 \text{ rad.} : n' = 0.0001009 \text{ rad./s} : b = 5.881 \times 10^{-9} m \quad (2.69)$$

Any of these values of the 'parasitic wave' shall produce a corresponding value of lambda $\lambda_{\max} = 892$ upon substituting it into (2.67). Hence the interval of the multiplier is $0 \leq \lambda \leq 892$ and $\lambda = 0, 1, 2, \dots, 892$. Also, we choose to classify the raising multiplier slowly so that we can study clearly all the parameter space assessable to the CCW as it propagates along the human blood circulating system of HIV/AIDS infected person.

2.5. Determination of the attenuation constant (η).

Attenuation is a decay process. It brings about a gradual reduction and weakening in the initial strength of the basic parameters of a given physical system. In this study, the parameters are the amplitude (a), phase angle (ε), angular frequency (n) and the spatial frequency (k). The dimension of the attenuation constant (η) is determined by the system under study. However, in this work, attenuation constant is the relative rate of fractional change FC in the basic parameters of the carrier wave. There are 4 (four) attenuating parameters present in the carrier wave. Now, if a, n, ε, k represent the initial basic parameters of the 'host wave' that is present in the carrier wave and $a - b\lambda, n - n'\lambda, \varepsilon - \varepsilon'\lambda, k - k'\lambda$ represent the basic parameters of the 'host wave' that survives after a given time. Then, the FC is

$$\sigma = \frac{1}{4} \times \left(\left(\frac{a - b\lambda}{a} \right) + \left(\frac{\varepsilon - \varepsilon'\lambda}{\varepsilon} \right) + \left(\frac{n - n'\lambda}{n} \right) + \left(\frac{k - k'\lambda}{k} \right) \right) \quad (2.70)$$



$$\eta = \frac{FC|_{\lambda=i} - FC|_{\lambda=i+1}}{\text{unit time}(s)} = \frac{\sigma_i - \sigma_{i+1}}{\text{unit time}(s)} \quad (2.71)$$

Thus (2.71) gives $\eta = 0.00112s^{-1}$ for all values of $\lambda (= 0, 1, 2, \dots, 892)$. In another development, we can as well select the first two terms of (2.68) so that $5.8265 \times 10^{-5}n' = 1.0666 \times 10^{-5} \varepsilon'$; then $\varepsilon' = 0.00005827$ radians and $n' = 0.00001067$ rad/s.

2.6. Determination of the time (t) that the CCW lasted.

The maximum time the CCW lasted as a function of the raising multiplier λ is also calculated from the attenuation equation shown by (2.71). The reader should note that we have adopted a slowly varying regular interval for the raising multiplier since this would help to delineate clearly the physical parameter space accessible to our model. However, it is clear from the calculation that the different attenuating fractional changes contained in the carrier wave are approximately equal to one another. We can now apply the attenuation time equation given below.

$$\sigma = e^{-2(\eta t)/\lambda} \quad (2.72)$$

$$t = - \left(\frac{\lambda}{2\eta} \right) \ln \sigma \quad (2.73)$$

The equation is statistical and not a deterministic law. It gives the expected basic intrinsic parameters of the 'host wave' that survives after time t . Clearly, we used (2.73) to calculate the exact value of the decay time as a function of the raising multiplier. Thus (2.73) yield $t = t_0 + t_1 + t_2 + \dots + t_{\max} = 8.5$ years for all values of the multiplier $\lambda = \lambda_0 + \lambda_1 + \lambda_2 + \dots + \lambda_{\max} = 398278$.

However, in another development, we can as well select the first two terms of (2.68) so that $5.8265 \times 10^{-5}n' = 1.0666 \times 10^{-5} \varepsilon'$; then $\varepsilon' = 0.00005827$ radians and $n' = 0.00001067$ rad/s. These values will yield a corresponding approximate value of $\lambda = 8426$. Although, we are not using these values of ε' and n' since the corresponding total time these sets of values produced is $t = t_0 + t_1 + t_2 + \dots + t_{\max} = 7100$ years which is not a realistic value.

2.0. Presentation of results.

The results of the calculated values of the wave characteristics of the human vibration (host wave) and those of the HIV vibration (parasitic wave) are shown in Table 3.1 below. The reader should note that the total value of the raising multiplier λ and the corresponding successive time t that the constitutive carrier wave lasted can be found from the addition of successive values. That is, $\lambda = \lambda_0 + \lambda_1 + \lambda_2 + \dots + \lambda_{\max}$; $t = t_0 + t_1 + t_2 + \dots + t_{\max}$. However, in the previous work which we also carried out by using Newtonian mechanics approach we did not add successive terms of the multiplier and time in other to get the actual time taken by the propagating constitutive carrier wave. What we did was to pick a given value of the multiplier and the corresponding value of the time. We may also represent interval or coordinate of the multiplier and time as $[\lambda, t]$.

First of all, we want to explain that fig.2 is merely inferred from fig.1 while fig. 4 is also inferred from fig.3. The inferred figures are necessary so that all the initial behaviour associated with the spectrum of the constitutive carrier wave within the given initial range or interval of the raising multiplier can be clearly and totally accessed. Finally, fig. 5 is a generalization of figs. 6 – 10. The reason is also to understand the physics initially available to the spectrum of the propagating constitutive carrier wave within the stipulated intervals of the multiplier. For instance, fig. 5 does not revealed completely or give detailed information about the radial and angular velocity of the CCW for a whole range of the raising multiplier and time taken the same time.

Table 3.1: shows the summary of the calculated values of the latent Human vibration (host wave) and latent HIV vibration (parasitic wave). The table also show comparison of two models our present work of Navier-Stokes and with a previous one of Newtonian mechanics approach.

S/N	Physical Quantity	Symbol	Navier-Stokes Approach	Newtonian Approach
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	Human wave characteristics		(Present work) Value / unit	(Previous work) Value/units
1	Amplitude	a	$5.252 \times 10^{-6} \text{ m}$	$2.1 \times 10^{-6} \text{ m}$
2	Angular velocity	n	0.09014 rad/s	$2.51 \times 10^{-7} \text{ rad/s}$
3	Phase angle	ε	$0.4924 \text{ rad (radians)}$	$0.6109 \text{ rad (radians)}$
4	Spatial frequency	k	163.4714 rad/m	166 rad/m
S/N	HIV Parasitic wave characteristics	Symbol	Value	Value
1	Amplitude	b	$5.881 \times 10^{-9} \text{ m}$	$1.6 \times 10^{-10} \text{ m}$
2	Angular velocity	n'	0.0001009 rad/s	$1.91 \times 10^{-11} \text{ rad/s}$
3	Phase angle	ε'	$0.0005514 \text{ rad (radians)}$	$0.0000466 \text{ rad (radians)}$
4	Spatial frequency	k'	0.1831 rad/m	0.0127 rad/m
Raising Multiplier		λ	892 ($0 \leq \lambda \leq 892$) Dimensionless constant	13070 ($0 \leq \lambda \leq 13070$) Dimensionless constant

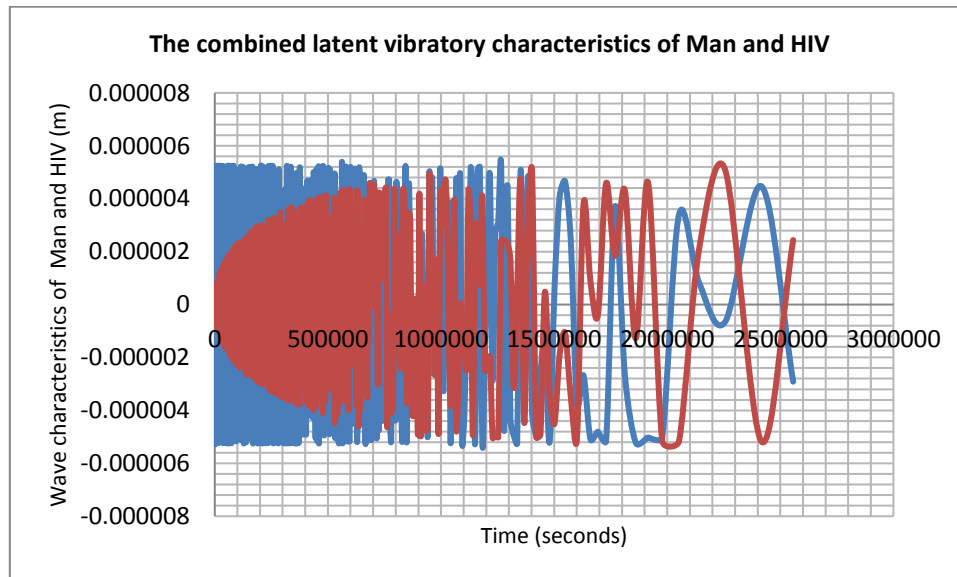


Fig. 1: shows the spectrum of the latent vibration of the resident host (Man – blue colour) and the parasitic wave (HIV/AIDS – brown colour) in the whole interval of the raising multiplier $\lambda[0-892]$ and with a corresponding total time interval of $t[0-255677]$ seconds or 8.5 years. The spectrum of the human vibration shows frequency separation gap beyond $t \geq 1368374s$ while the spectrum of the HIV vibration show frequency separation gap $t \geq 1140825 \text{ s}$. The respective total time are $t = t_0 + t_1 + t_2 + \dots + t_{1368374} = 221281574s$ (85 months or 7.1 years) and $t = t_0 + t_1 + t_2 + \dots + t_{1140825} = 198755026s$ (76 months or 6.3 years). The graph represents equation (2.1) for $\beta=1$ and equation (2.2) for $\lambda(= 0, 1, 2, \dots, 892)$.

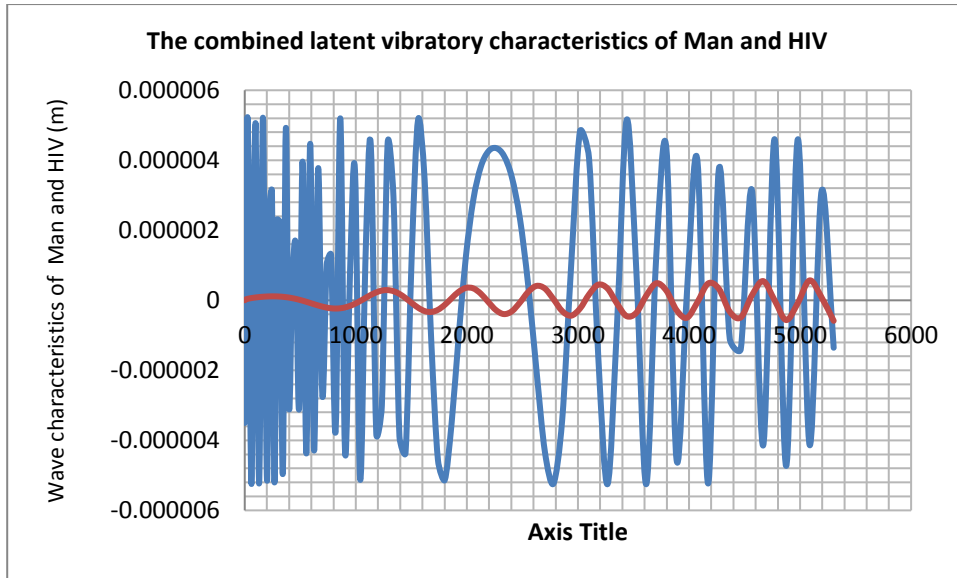


Fig. 2: shows the spectrum of the latent vibration of the resident host (Man – blue colour) and the parasitic wave (HIV/AIDS – brown colour) in the interval of the raising multiplier $\lambda[0-100]$. The host vibration shows anomaly at $t=2193$, while the HIV vibration progresses from the origin with increasing frequency and small amplitude. The total time taken for the host wave to show the anomaly is $t = t_0 + t_1 + t_2 + \dots + t_{2193} = 48173s$ (13 hours). The graph represents equation (2.1) for $\beta=1$ and equation (2.2) for $\lambda(= 0, 1, 2, \dots, 100)$.

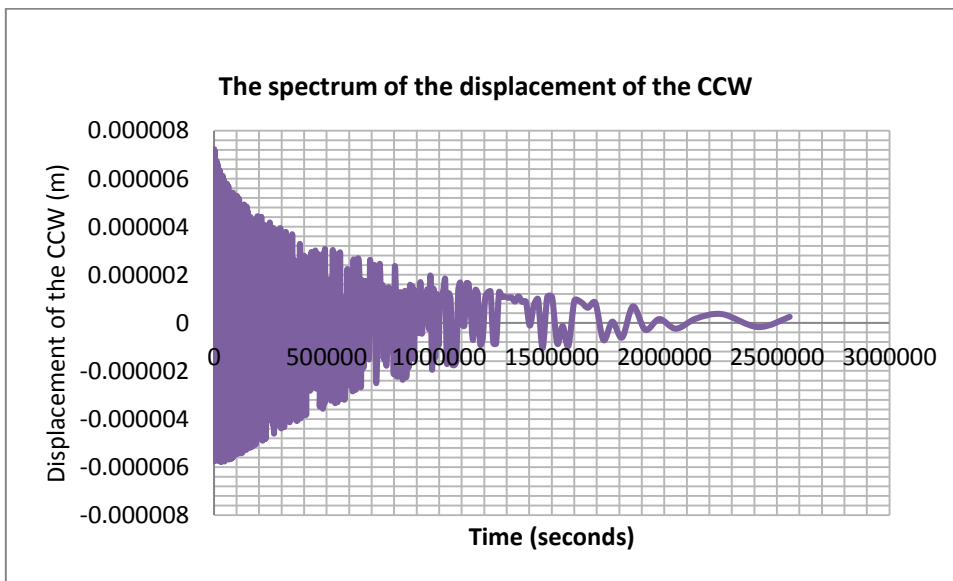


Fig. 3: shows the spectrum of the CCW in the interval $\lambda[0-600]$. The spectrum shows irregular frequency beyond $[492, 1292903s]$ and the time taken is $t = t_0 + t_1 + t_2 + \dots + t_{1292903} = 214595714$ seconds (82 months or 6.8 years). The spectrum of the HIV/AIDS vibration ceases to exist when $[892, 2556771s]$. Hence the total time that the CCW lasted is $t = t_0 + t_1 + t_2 + \dots + t_{2556771} = 2.65 \times 10^8 s$ (3067 days or 102 months or 8.5 years). The spectrum represents equation (2.7) for $\lambda = 0, 1, 2, \dots, 892$.

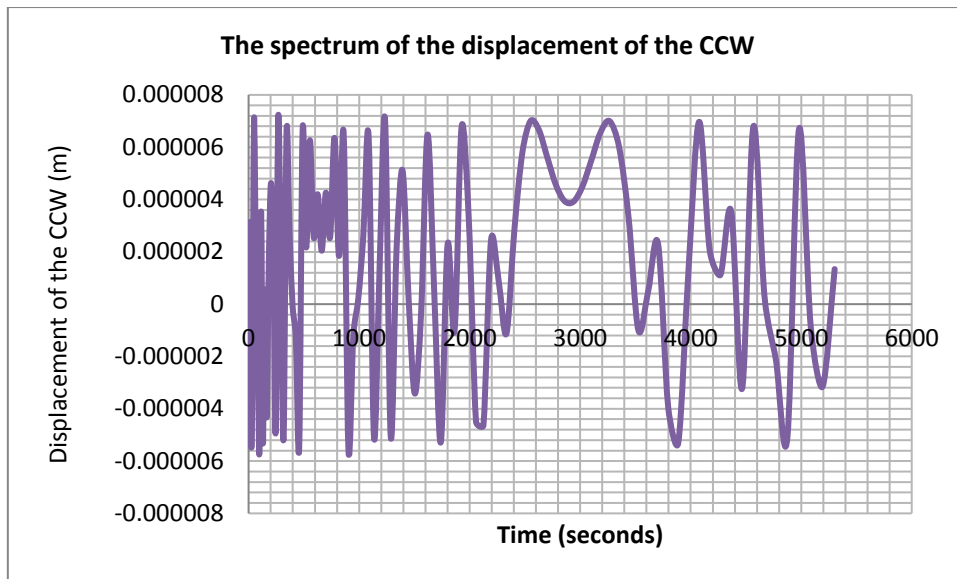


Fig. 4: shows the spectrum of the CCW in the interval $\lambda [0-100]$. The spectrum shows a partial separation when $[33, 554s]$ and $[76, 3000 s]$. The corresponding time taken for the multiplier to reach these values is $t = t_0 + t_1 + t_2 + \dots + t_{554} = 6354$ seconds (1 hour) and $t = t_0 + t_1 + t_2 + \dots + t_{3000} = 77125$ seconds (21 hours). The spectrum represents equation (2.7) for $\lambda = 0, 1, 2, \dots, 892$.

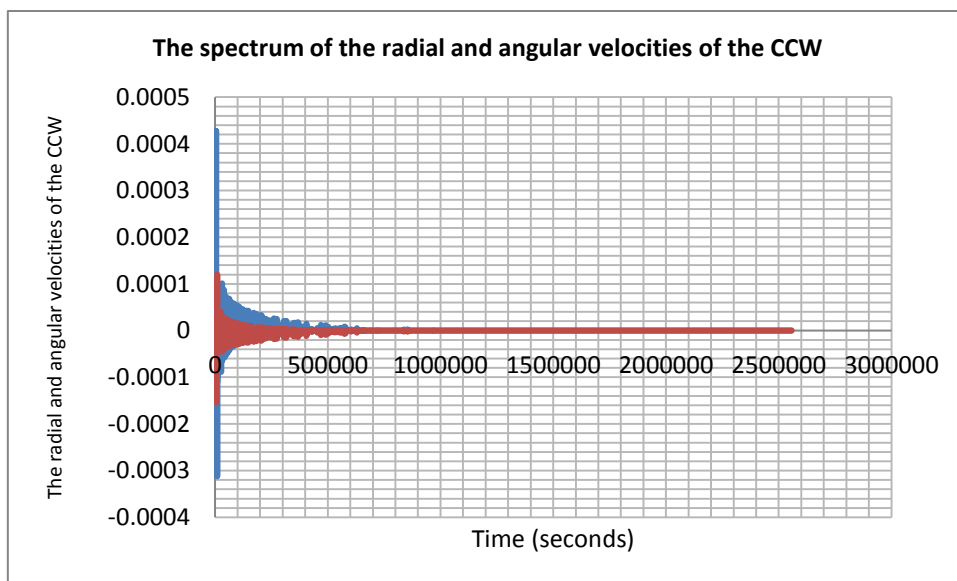


Fig. 5: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the whole interval of the raising multiplier $\lambda [0-892]$ and with a corresponding total time interval of $t [0-255677]$ seconds or 8.5 years. The graph represents equation (2.32) and (2.33) for $\lambda (= 0, 1, 2, \dots, 892)$.

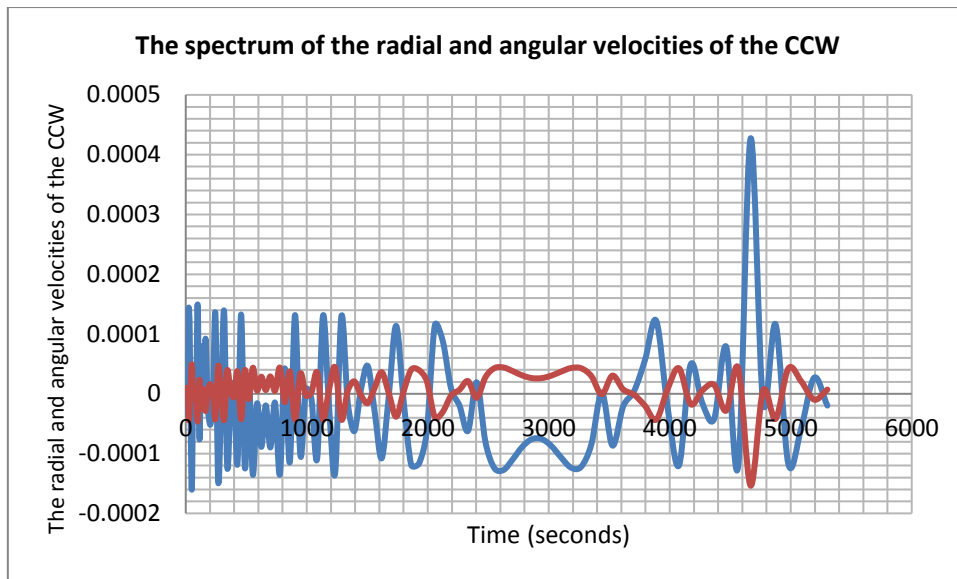


Fig. 6: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the interval of the raising multiplier $\lambda[0-100]$. The spectrum of both velocities shows separation at 554s, 3000s and a significant peak at 4668s. The total time to display these separations and the peak value are respectively: $t = t_0 + t_1 + t_2 + \dots + t_{554} = 6354s$ (1.7 hours), $t = t_0 + t_1 + t_2 + \dots + t_{300} = 77125s$ (21 hours) and $t = t_0 + t_1 + t_2 + \dots + t_{4668} = 146565s$ (1 day). The graph represents equation (2.32) and (2.33) for $\lambda (= 0, 1, 2, \dots, 100)$.

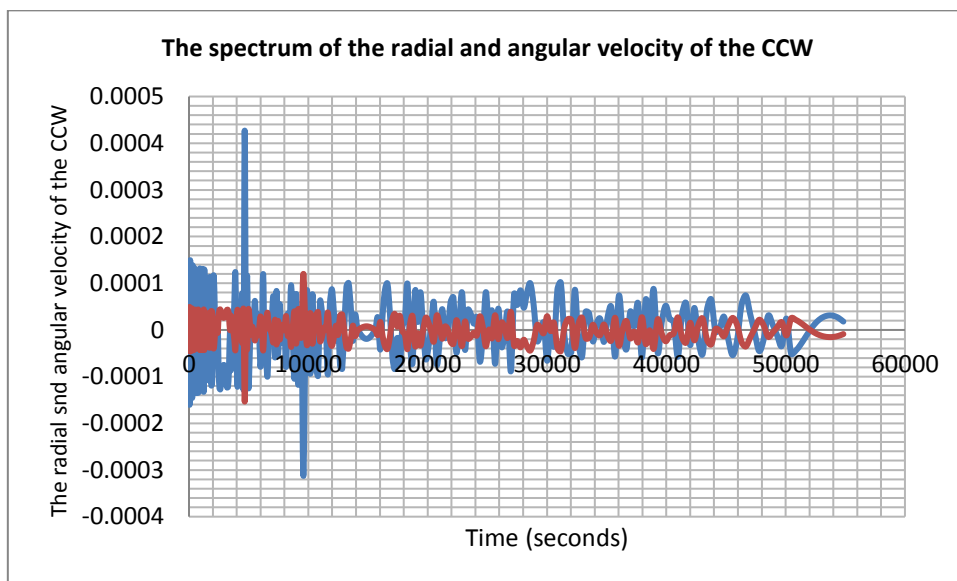


Fig. 7: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the interval of the raising multiplier $\lambda[0-300]$ and with a corresponding total time interval of $t [0-522370]$ seconds or 2 months. The graph represents equation (2.32) and (2.33) for $\lambda (= 100, 101, 102, \dots, 300)$.

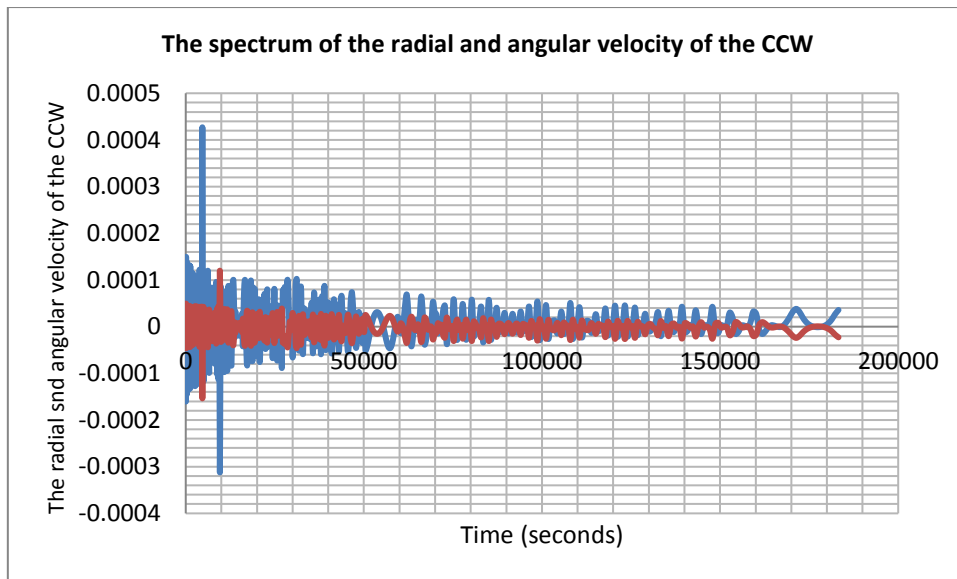


Fig. 8: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the interval of the raising multiplier $\lambda[0-500]$ and with a corresponding total time interval of $t [0-2736480]$. The spectrum shows several constrictions but the significant ones are at time 52022s, 162691s and 173174s. The various total time are: $t = t_0 + t_1 + t_2 + \dots + t_{52022} = 4848385s$ (1.8 months), $t = t_0 + t_1 + t_2 + \dots + t_{162691} = 23379986s$ (8 months) and $t = t_0 + t_1 + t_2 + \dots + t_{173174} = 25399930s$ (9 months). The graph represents equation (2.32) and (2.33) for $\lambda (= 0, 1, 2, \dots, 500)$.

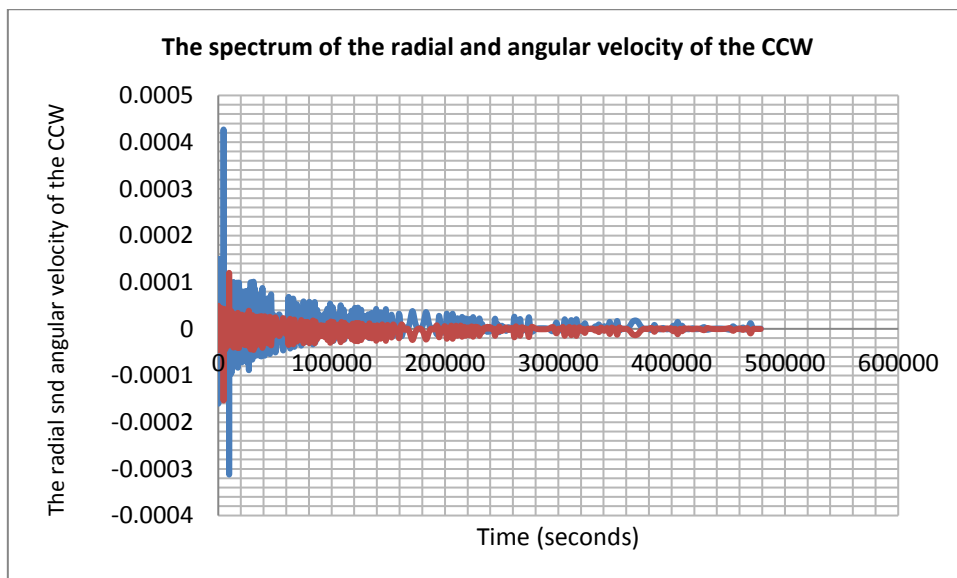


Fig. 9: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the interval of the raising multiplier $\lambda[0-700]$ and with a corresponding total time interval of $t [0-478757]$. Thus, the total time taken in this interval is $t = t_0 + t_1 + t_2 + \dots + t_{478757} = 89339250s$ (34 months or 2.8 years). The spectrum also displays several constrictions beyond $t \geq 100000s$. The graph represents equation (2.32) and (2.33) for $\lambda (= 0, 1, 2, \dots, 700)$.

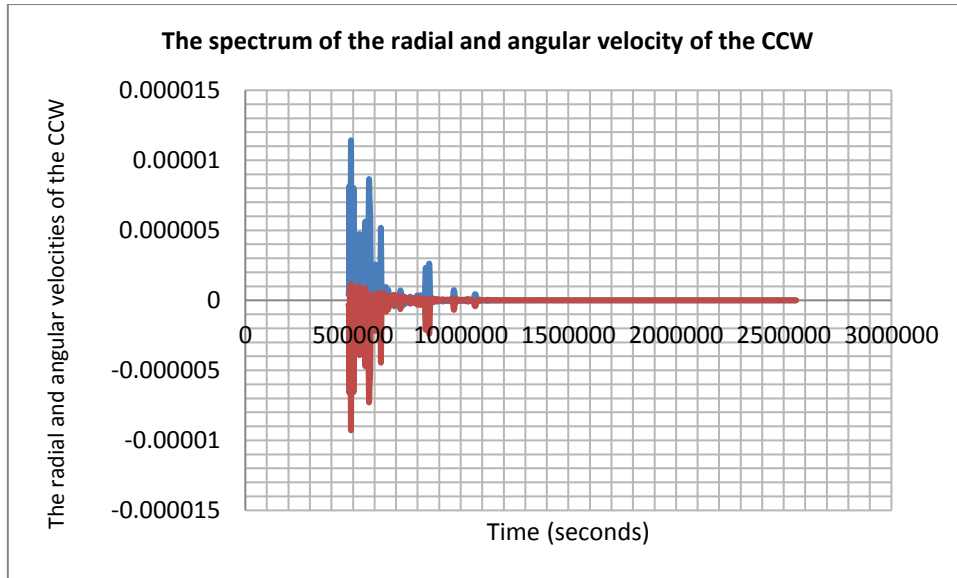


Fig. 10: shows the spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave in the interval of the raising multiplier $\lambda[500-700]$. It is obvious that both spectra go to zero when $t \geq 1011535s$. Hence the total time both velocities go to zero is $t = t_0 + t_1 + t_2 + \dots + t_{1011535} = 182592658s$ (70 months). Both velocities finally cease to exist or come rest at 2556771s. The total time taken before coming to rest is $t = t_0 + t_1 + t_2 + \dots + t_{2556771} = 265092265s$ (102 months or 8.5 years). The graph represents equation (2.32) and (2.33) for $\lambda (= 700, 701, 702, \dots, 892)$.

4.0 Discussion of Results.

Table 3.1 shows the values of the latent human vibration and the HIV latent vibration which we calculated in a previous study using Newtonian mechanics approach. However, the two results are quite at variance with the present results in which case we applied Navier-Stokes equation approach. Although, the amplitude and the spatial frequency of the host wave are slightly in agreement but we feel that the results of the present work are more sensible and reliable since the Navier-Stokes equation are strictly meant for fluid that are incompressible like blood.

The spectrum of the vibration of the Man (host wave) and the HIV (parasitic wave) as shown in fig. 1 are oppositely related. Initially the HIV oscillating phase increases with time as the multiplier is increased from the origin while the host wave almost have a stable oscillating phase with an amplitude of about $\pm 5.2336 \times 10^{-6} m$ starting from the origin. It is clear from the figure that both Man and HIV vibrate at a very high frequency, with that of Man been higher. It is the high frequency of vibration that accounts for the blurred nature of the spectrum. However, the frequency of both wave become low with increasing wavelength beyond $t \geq 1420639s$. The total time when both vibrations display this low frequency and hence increase in wavelength is 225489864 seconds (86 months or 7 years). However, one final remark is that both independent vibrations under the given condition do not finally go to zero. That means within the given interval $\lambda[0-892]$ and $t[0-8.5]$ years there exists a residual vibration (positive or negative) which could still be felt even after the termination of the independent waves.

Fig. 2 is inferred from fig. 1. The spectrum of the independent vibrations of Man and HIV shows that the source function of both vibrations is actually incoherent. While that of the HIV takes off from the origin with zero phase angle the human vibration has a specific phase angle from the origin. This is evident of the fact that it is not possible to determine the origin of the human vibration. The fig. 2 also shows that the frequency and oscillating amplitude of the human vibration are much higher than those of the HIV vibration, although the wavelength of the HIV vibration is longer. The frequency of the HIV vibration increases as the multiplier is increased with time.

It is obvious from fig. 3 that the spectrum of the constitutive carrier wave almost becomes zero with amplitude of $2.5385 \times 10^{-7}m$ when the raising multiplier is 892 and after a total time of 265092265 seconds or 8.5 years. Although, there are regions of anomaly in the frequency of the amplitude at the coordinate $[860, 1265932s]$ and $[878, 1600821s]$. Now by



summation we get that the total time taken for the first anomaly counting from the origin is 212023594 seconds (81 months or 6.8 years) while for the second anomaly is 237617409 seconds (91 months or 7.6 years). These two results agree suitably with the time for the low frequency regime of the independent vibrations of fig. 1. Beyond this regime is characterised by highly reduced amplitude and irregular frequency before the CCW finally goes to zero and ceases to exist when the raising multiplier attains a critical value of 892.

Fig. 4 is deduced from fig. 3. It is evident from the figure that the spectrum of the propagating constitutive carrier wave display anomalous behaviour at the coordinate [76, 3000s]. The total time taken for this anomalous behaviour in the spectrum of the CCW counting from the origin is 77125 seconds (21 hours). This characteristic in the spectrum shows that within 21 hours after infection with HIV the biological system of Man must imaginarily recognize and react to the interference of a strange velocity-like body whose influence may be destructive or constructive as the case may be. However, the recuperative inbuilt factor in the human system tends to annul this effect and renormalizes the frequency of vibration.

We are only going to discuss the physics associated with fig. 6 – 10 since they are inferred from fig. 5 which covers the whole range of the multiplier and time. This full coverage as we have already explained does not provide the full information about the behaviour of the CCW. The spectrum of the radial velocity U_r (blue colour) and the angular velocity U_φ (brown colour) of the constitutive carrier wave have some features in common. It is shown in fig. 6 that the velocities spectrum show anomalous behaviour at the coordinate [33, 554 s] and [76, 3000s]. The total time taken for the first anomalous behaviour of the CCW counting from the origin is 6354 seconds (1.7 hours) while for the second anomalous behaviour of the CCW is 77125 seconds (21 hours). This shows that the velocities of the propagating CCW are altered within the first 21 hours counting from the moment one is infected with HIV.

There are two anomalous broad peaks display by the spectrum of the radial velocity of the propagating CCW. The peaks are at the coordinate [94, 4668 s] and [133, 9576 s] in fig. 6 and 7 respectively. The total time taken for the first anomalous peak behaviour counting from the origin is 146565 seconds (1 day). The peaks represent the constructive (positive value) and destructive (negative value) interference between the human vibration and the HIV vibration respectively with regard to the radial velocity until it displayed the last pulse in fig. 10.

The last pulse has a coordinate of [832, 1011535s] with velocities amplitude of $\pm 3.0147 \times 10^{-8} \text{ rad/s}$. The total time taken for the radial and angular velocity to show the last pulse behaviour is 182592658 s (70 months or 5.8 years). Thereafter, both velocities go to zero and ceases to exist with a final value of about $\pm 2.5477 \times 10^{-12} \text{ rad/s}$ when the multiplier attains a critical value of 892 and with a corresponding total time of 265092265 seconds (8.5 years).

Another obvious significant feature common to the fig. 6 – 10 is the depletion in the spectra of the radial and angular velocity. The interpretation of this depletion is that the HIV is now taking active dominant control of the host biological system. Thus, the constituent parameters of the HIV wave function are gradually becoming equal to those of the host. Under this situation the destructive effect of the HIV in the host system is now becoming very intense and difficult to control.

The spectra of the radial velocity and the angular velocity are characterized by two major phases with respect to the time taken. The first phase is between time $t = 0$ and the time of the last pulse which took about 69 months or 5.7 years, the second phase is between the time of the last pulse to when the radial velocity and the angular velocity of the CCW goes to zero which is about (102 – 69) months and this is equal to 33 months (2.7 years). Now let us classify or relate these phases with special reference to the clinical literature on HIV/AIDS.

The first phase is related to the HIV infection when it is now taking absolute destructive and noticeable effect in the Human biological system. Because of the significant behaviour of the radial velocity and the angular velocity of the constitutive carrier wave in this interval we believe that the HIV infection becomes more intensive and pronounced during the first 5 years. According to the clinical literature this is the region of increase in viral load.

The second phase is related to AIDS condition, that is when all the active constituents of the resident Host wave (Human vibration) contained in the constitutive carrier wave are now becoming completely eroded by the destructive tendency of the interfering parasitic wave (HIV vibration). In the absence of specific treatment, the HIV infection suddenly degenerates to AIDS after about 5.7 years. This period involves a steady decay process which results in a gradual reduction and weakening in the initial strength of the intrinsic parameters of the host biological system.



5.0. Conclusion

The spectrum of the vibration of the Man (host wave) and the HIV (parasitic wave) are oppositely related. Initially, the HIV oscillating phase increases with time as the multiplier is increased from the origin while the host wave almost have a stable oscillating phase starting from the origin. It is clearly established in this study that both Man and HIV vibrate at a very high frequency, with that of Man been higher. The wave characteristics of Man and that of the HIV are actually incoherent and are initially out of phase. The characteristic spectrum of the radial velocity and the angular velocity show that within 21 hours after infection with HIV the biological system of Man must imaginarily recognize and react to the interference of a strange velocity-like body (HIV) whose influence may be destructive or constructive as the case may be. However, the recuperative inbuilt factor in the human system initially tends to annul this effect and renormalizes the frequency of vibration until it finally goes to zero or ceases to exist after a specified time. It is also shown in this work that the radial and angular velocity are oppositely related.

References

- [1]. Sepkowitz, K. A. (2001) 'AIDS-the first 20 years' N. Engl. J. Med. 344 (23) : 1764 – 72.
- [2]. Kallings LO (2008). "[The first postmodern pandemic: 25 years of HIV/AIDS](#)". *J Intern Med* **263** (3): 218–43. [doi:10.1111/j.1365-2796.2007.01910.x](#). [PMID 18205765](#). (subscription required)
- [3]. Gilbert, PB et al. (28 February 2003). "Comparison of HIV-1 and HIV-2 infectivity from a prospective cohort study in Senegal". *Statistics in Medicine* **22** (4): 573–593. [doi:10.1002/sim.1342](#). [PMID 12590415](#).
- [4]. Reeves, J. D. and Doms, R. W (2002). "Human Immunodeficiency Virus Type 2". *J. Gen. Virol.* **83** (Pt 6): 1253–65. [doi:10.1099/vir.0.18253-0](#). [PMID 12029140](#).
- [5]. Hel Z, McGhee JR, Mestecky J (June 2006). "HIV infection: first battle decides the war". *Trends Immunol.* **27** (6): 274–81. [doi:10.1016/j.it.2006.04.007](#). [PMID 16679064](#).
- [6]. Gilbert, PB et al. (28 February 2003). "Comparison of HIV-1 and HIV-2 infectivity from a prospective cohort study in Senegal". *Statistics in Medicine* **22** (4): 573–593. [doi:10.1002/sim.1342](#). [PMID 12590415](#).
- [7]. Reeves, J. D. and Doms, R. W (2002). "Human Immunodeficiency Virus Type 2". *J. Gen. Virol.* **83** (Pt 6): 1253–65. [doi:10.1099/vir.0.18253-0](#). [PMID 12029140](#).
- [8]. Tortora, Gerard J.; Derrickson, Bryan (2012). "The Cardiovascular System: Blood Vessels and Hemodynamics". Principles of Anatomy & Physiology (13th ed.). John Wiley & Sons. "Laminar flow analysis". p. 817. [ISBN 978-0470-56510-0](#).
- [9]. L. Pirkl and T. Bodnar, Numerical Simulation of Blood Flow Using Generalized Oldroyd-B Model, European Conference on Computational Fluid Dynamics, 2010
- [10] G. Thurston, Plasma Rheological Parameters for the Viscosity, Viscoelasticity, and thixotropy of Blood, Biorheology 1979, 16, 149–162
- [11]. Faber, T. E. *Fluid Dynamics for Physicists*. New York: Cambridge University Press, 1995.
- [12]. Tritton, D. J. *Physical Fluid Dynamics, 2nd ed.* Oxford, England: Clarendon Press, pp. 52-53 and 59-60, 1988.
- [13] Clay Mathematics Institute. "Navier-Stokes Equations." http://www.claymath.org/Millennium_Prize_Problems/Navier-Stokes_Equations/.
- [14] Stam, Jos. "Stable Fluids." Alias|wavefront, n.d. Web. 14 May 2012. <<http://www.dgp.toronto.edu/people/stam/reality/Research/pdf/ns.pdf>>.
- [15] T. Hsieh, A.B. Wardlaw Jr., T.J. Bogar, P. Collins, and T. Coakley, "Numerical Investigation of Unsteady Inlet Flowfields," AIAA Journal, vol. 25, no. 1, pp. 75–81, 1987.



[16]. Bakker, Andre. "Computational Fluid Dynamics." *Lecture 4 - Classification of Flows*. N.p., 2006. Web. 14 May 2012. <<http://www.bakker.org/dartmouth06/engs150/04-clsfm.pdf>>.

[17]. Edison A. Enaibe, Akpata Erhieyowwe, Umukoro Judith and E. U. Nwose (2017): 'Quantitative Eradicative Cure Profile of HIV/AIDS Using Electromagnetic (EM) Wave Destruction Technique' *Journal of scientific Research & Reports*. Vol. 13, No. 3, pp 1 – 28. Science Domain international www.sciencedomain.org.