Quantitative Treatment of Hiv/Aids Inthe Human Micro-Vascular Circulating Blood System

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ABSTRACT

Vibration is the cause ofall that exists.Generally, any form of matterincluding biological systemscould be described by vibration and hence represented by a purely defined wave form. This study presents a model for determining the dynamic mechanical characteristics of HIV/AIDS in the human blood circulating system. Our work assumes that the physical dynamic components of the HIV responsible for their destructive tendency are $b\lambda$, $n'\lambda$, $\varepsilon'\lambda$ *and* $k'\lambda$ *been influenced by the multiplicative factor* λ *whose physical range of interest is* $0 \le \lambda \le 13070$. *We constructed the constitutive carrier wave equation on the basis of the vibratory dynamic components of the human (host) parameters and those of the HIV (parasite). It is established in this study that when the HIV enters the human blood circulating system, it takes between 60 to 240 days before its absolute effects would begin to manifest. This in the literature of clinical disease is referred to as the window period. The negative influence of the HIV in the human system becomes intense and more pronounced when the HIV is about 5 years (60 months) counting from the day it is contacted. This study revealed that AIDS actually results when these destructive dynamic components of the HIV gradually become equal to their corresponding active dynamic components in the human blood circulating system. This is when the range of the multiplicative factor* $12803 \le \lambda \le 13070$. *The time it takes the HIV infection to degenerate into AIDS if uncontrolled is about 8 years (96months). The displacement of the carrier wave that describes the biological system of Man finally goes to zero - a phenomenon called death, when the multiplicative factor approaches the critical value of* 13070 *and the time it takes to attain this value is about 11 years (132 months).*

KEYWORDS: *latent vibration, 'host wave', 'parasitic wave', carrier wave, HIV/AIDS, amplitude, oscillating phase.*

I. INTRODUCTION

Some waves in nature behave parasitically when they interfere with another one. Such waves as the name implies has the ability of transforming the initial characteristics and behaviour of the interfered wave to its own form and quality after a given period of time. Under this circumstance, all the active constituents of the interfered wave would have been completely eroded and the resulting wave which is now parasitically monochromatic, will eventually attenuateto zero, since the 'parasitic wave' does not have its own independent parameters for sustaining a continuous existence.

The role of Human-Immunodeficiency Virus (HIV) in the normal blood circulating system of man (host) has in general been poorly understood. However, its role in clinical disease has attracted increasing interest. Human immunodeficiency virus (HIV) infection / acquired immunodeficiency syndrome (AIDS) is a disease of the human immune system caused by HIV [1].During the initial infection a person may experience a brief period of influenza-like illness. This is typically followed by a prolonged period without symptoms. As the illness progresses it interferes more and more with the immune system, making people much more likely to get infections, including opportunistic infections, which do not usually affect people with immune systems. In the absence of specific treatment, around half of the people infected with HIV develop AIDS within ten years [2] and average survival time after infection with HIV is estimated to be 9 to 11 years [3].

According to the literature of clinical diseases, the HIV feeds on and in the process kills the active cells that make up the immune 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 a living organism and with its own peculiar characteristics even before it entered the system of Man.

It is the vibration of the 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. Fortunately the blood stands as a means of conveying this vibration to all units of the human system.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. 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 wave function have been altered.

The activity of the HIV is everywhere the same within the human blood circulating system, mutation if at all does not affect its activity. 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 amplitude, angular frequency, wave number and phase angle of the host which are the basic parameters of vibration were initially greater than those of the HIV. The human aorta is the main truck of a series of vessels which convey the oxygenated blood from the heart to the tissues [4]. It is described in several portions, viz, the ascending aorta the arch of the aorta, and the descending aorta. The ascending aorta is about 7cm (0.07m) in length and it has a radius of 1.5cm (0.015m). Arch of the Aorta is about 1.8cm (0.018m) in length and its radius is 1cm (0.01m) while the descending aorta has a length of 1.14cm (0.0114m) and a radius of 1.11cm (0.0111m). The human artery is an extension of the aorta and there are various forms with approximate radius of about 0.4cm (0.004m) [5]. The smallest vessels, the capillaries, have a diameter of about $5x10^{-6}$ m and $10x10^{-6}$ m, so that the red blood cells whose diameter is about $8x10^{-6}$mcan pass through it [6]. There are about 250 capillaries/$mm^3$of body tissues and average length of a capillary is about 600 microns ($600x10^{-6}$m). However, we are going to utilize only the ascending parameters of the aorta in our calculation and assume a uniform geometry and structure for all the vascular blood vessels. This assumption is reasonable since the latent

vibration takes its first unique course through the ascending aorta. The human veins are not taken into consideration in our work, because it only conveys denatured blood (deficient in oxygen and food nutrients) to the human heart for reactivation. Also because of the limited length of the human capillaries the exchange process of active blood in this region of space does not take time as a result our computation will not include the capillaries as well. Human blood is a liquid tissue composed of roughly 55% fluid plasma and 45% cells. The three main types of cells in blood are red blood cells, white blood cells and platelets. 92% of blood plasma is composed of water and the other 8% is composed of proteins, metabolites and ions [7]. The density of blood plasma is approximately 1025 kg/m^3 and the density of blood cells circulating in the blood is approximately 1125 kg/m^3. Blood plasma and its contents are known as whole blood [8]. The average density of whole blood for a human is about 1050 kg/m^3. Blood viscosity is a measure of the resistance of blood to flow, which is being deformed by either shear or extensional strain [9]. The dynamic viscosity (μ) of the human blood at 37^0C is usually between $0.003kgm^{-1}s^{-1}$and$0.004kgm^{-1}s^{-1}$, the arterial blood perfusion rate (w_b) is $0.5kgm^{-3}s^{-1}$[10]. The viscosity of blood thus depends on the viscosity of the plasma, in combination with the particles. However, plasma can be considered as a Newtonian fluid, but blood cannot due to the particles which add non-idealities to the fluid.If a wave is to travel through a medium such as water, air, steel, or a stretched string, it must cause the particles of that medium to oscillate as it passes [11]. For that to happen, the medium must possess both mass (so that there can be kinetic energy) and elasticity (so that there can be potential energy). Thus, the medium's mass and elasticity property determines how fast the wave can travel in the medium.The initial characteristics of a given wave with a definite origin or source can best be determined by the use of a sine wave function. However, for the deductive determination of the initial behaviour of a wave whose origin is not certain, the cosine wave function can best be effectively utilized.

The principle of superposition of wave states that if any medium is disturbed simultaneously by a number of disturbances, then the instantaneous displacement will be given by the vector sum of the disturbance which would have been produced by the individual waves separately. Superposition helps in the handling of complicated wave motions. It is applicable to electromagnetic waves and elastic waves in a deformed medium provided Hooke's law is obeyed Generally, it is the human blood that responds to the latent vibration from the heart with a specified wave form. The blood then propagates away from the region of the disturbance and in the process circulates oxygen and food nutrients to nourish the biological cells of the human system. Any alteration to this process results to starvation, a gradual weakening of the fundamental cells and a subsequent breakdown of the entire human system if uncontrolled.

II. RESEARCH METHODOLOGY

[1] The wave characteristics of blood in the circulating system of a normal person free from HIV/AIDS is assumed to be measured and the following observations about the wave function were recorded: (i) the amplitude, a (ii) the phase angle, ε (iii) the angular frequency, n and(iv) the wave number, k. Note that $a, \varepsilon \text{ and } n$ are assumed to be constant with time in the human system except for some fluctuating factors, e.g. illness, which of course can only alter them slightly and temporarily.

[2] The wave characteristics of blood in the circulating system of a HIV/AIDS infected candidate, whose immune count rate is already zero is assumed to be measured. The following observations about the wave properties were recorded: (i) the amplitude, b (ii) the phase angle, ε' (iii) the angular frequency, n' (iv) the wave number, k'.

[3] Since the immune system of the HIV/AIDS individual is exactly zero, the measured wave function shall depend entirely on the vibrating property of the HIV only as every other active constituents of the blood system have been completely eroded. That is, the measured wave characteristic of the two candidates cannot be the same and whatever that makes the difference are the attributes of the HIV.

[4] The measured wave properties of the HIV infected candidate is independent of intrinsic variables such as the number, size, mass and of course the mutation property (if at all) of the invading HIV.

[5] The measured wave characteristic of the HIV infected candidate is the same everywhere within the host (Man). That is, irrespective of the occupation of the HIV in the host system, be it in the capillary, vein, liver, bone marrow, or in the brain, the activity of the HIV is the same everywhere and hence the wave function must be the same. Of course the veins only carry denatured blood back to the heart for activation and the nutrient blood takes negligible time in the capillary during the energy exchange process.

[6] The wave properties of the HIV cannot be directly measured since it does not have independent existence outside the host system. As a result, the wave function of the HIV can only be deductively measured.

[7] If the HIV exists it must have a peculiar vibration of its own which must be independent of the vibration of the host. If the attributes of this vibration are known, then, it can be selectively destroyed from the body of the host.

[8] That the HIV kills slowly with time shows that the wave function of the HIV and that of the human (host) system are incoherent. As a result, the amplitude, frequency and the phase angle of vibration of the host were initially greater than those of the vibration of the HIV (parasite).

The aim of this work is to describe the biomechanics of HIV/AIDS and to report the methodology developed in our laboratory to characterize the dynamics of the 'host wave' and those of 'HIV wave' in the constitutive carrier wave equation propagating in the human blood circulating system. Understanding wave propagation in arterial walls, local hemodynamics, and wall shear stress gradient is important in understanding the mechanisms of cardiovascular function. Arterial walls are anisotropic and heterogeneous, composed of layers with different bio-mechanical characteristics which make the understanding of the mechanical influences that arteries contribute to blood flow very difficult [12].

This paper is outlined as follows. Section 1, illustrates the basic concept of the work under study. The mathematical theory is presented in section 2. We present the results obtained in section 3. While in section 4, we present the analytical discussion of the results obtained. The conclusion of this work is shown in section 5, and this is immediately followed by an appendix and a list of references.

$$y = \sqrt{\left(a^2 - b^2\lambda^2\right) - 2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)} \times \cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - \left|\partial E\right|\right) \quad (2.29)$$

where we have redefined the resultant amplitude A as

$$A = \sqrt{\left(a^2 - b^2\lambda^2\right) - 2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)} \quad (2.30)$$

The spatial oscillating phase and the total phase angle E are respectively given by

$$\phi = \cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - \left|\partial E\right|\right) \quad (2.31)$$

$$E = \tan^{-1}\left(\frac{a\sin\varepsilon - b\lambda\sin\left((n - n'\lambda)t - \varepsilon'\lambda\right)}{a\cos\varepsilon - b\lambda\cos((n - n'\lambda)t - \varepsilon'\lambda)}\right) \quad (2.32)$$

By definition: the modulation angular frequency is given by $(n - n'\lambda)$, the modulation propagation constant is $(\vec{k} - \vec{k}'\lambda)$, the phase difference δ between the two interfering waves is $(\varepsilon - \varepsilon'\lambda)$, the interference term is given by ; $2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)$, while waves out of phase interfere destructively according to $\left(a - b\lambda\right)^2$ and waves in-phase interfere constructively according to $\left(a + b\lambda\right)^2$ .In the regions where the amplitude of the carrier wave is greater than either of the amplitude of the individual wave, we have constructive interference that means the path difference is $(\varepsilon + \varepsilon'\lambda)$, otherwise, it is destructive in which case the path difference is $(\varepsilon - \varepsilon'\lambda)$. If $n \approx n'$, then the average angular frequency say $(n + n')/2$ will be much more greater than the modulation angular frequency say $(n - n')/2$ and once this is achieved, then we will have a slowly varying carrier wave with a rapidly oscillating phase.

The total differentiation of the total phase angle E gives the exact total phase angle. That is

$$\partial E = \frac{\partial E}{\partial \varepsilon} d\varepsilon + \frac{\partial E}{\partial \varepsilon'} d\varepsilon' \quad (2.33)$$

$$\left|\partial E\right| = \sqrt{\left(\frac{\partial E}{\partial \varepsilon}\right)^2 + \left(\frac{\partial E}{\partial \varepsilon'}\right)^2} \quad (2.34)$$

There is need for us to make the total phase angle of the oscillating phaseof the carrier wave not to depend explicitly on time, since this would enhance quality results so that the subsequent wave form does not produce irregular complex behaviour.

$$\frac{\partial E}{\partial \varepsilon} = \frac{a^2 - ab\lambda\cos\left((\varepsilon + \varepsilon'\lambda) - (n - n'\lambda)t\right)}{a^2 + b^2\lambda^2 - 2ab\lambda\cos\left((\varepsilon + \varepsilon'\lambda) - (n - n'\lambda)t\right)} \quad (2.35)$$

$$\frac{\partial E}{\partial \varepsilon'} = \frac{-b^2\lambda^3 + ab\lambda^2\cos\left((\varepsilon + \varepsilon'\lambda) - (n - n'\lambda)t\right)}{a^2 + b^2\lambda^2 - 2ab\lambda\cos\left((\varepsilon + \varepsilon'\lambda) - (n - n'\lambda)t\right)} \quad (2.36)$$

Equation (2.29) is now the constitutive carrier wave equation necessary for our study. It describes the activity and performance of most physically active systems. As the equation stands, it is a carrier wave, in which it is only the variation in the intrinsic parameters of the 'parasitic wave' that determines the life span of the active biological system which it describes.

2.1 Equation of motion of the carrier wave in the blood vessels of the host

The carrier wave given by (2.29) can only have a maximum value if the spatial oscillating phase is equal to 1. Hence

$$y_m = \sqrt{\left(a^2 - b^2\lambda^2\right) - 2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)} \quad (2.37)$$

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

(2.38)

$$\frac{d^{\,2}y_m}{dt^2} = (n - n'\lambda)^2 (a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right) \times \left((a^2 - b^2\lambda^2) - 2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)\right)^{-\frac{1}{2}}$$

$$- (n - n'\lambda)^2 (a - b\lambda)^4 \sin^2\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right) \times \left((a^2 - b^2\lambda^2) - 2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)\right)^{-\frac{3}{2}}$$

(2.39)

The equation of motion obeyed by the constitutive carrier wave as it propagates along the human blood vessels experiences two major resistive factors. Firstly, the resistance posed by the elasticity of the walls of the blood vessels and secondly, the elastic resistance of the blood medium. The medium's mass and elasticity property determines how fast the carrier wave can travel in the medium. Consequently, the equation of motion would be partly Newtonian due to the fluidize nature of the blood medium and non-Newtonian due to the particle constituent of the blood which creates nonidealities. We can therefore write the equation of motion as

$$F = -\mu\left(\frac{dy^2}{dt}\right) - \sigma y^2 \quad (2.40)$$

$$\rho V \frac{d^{\,2}y}{dt^2} + 2\mu y\left(\frac{dy}{dt}\right) + \sigma y^2 = 0 \quad (2.41)$$

Where ρ is the density of the human blood (kgm-3), V is the volume of the blood vessel which is considered to be cylindrical vascular geometry ($\pi r^2 l$) and the unit is (m3), μ is the dynamic viscosity of blood (kgm-1s-1) and σ is the elasticity of the blood medium. The influence of gravity on the flow of blood is assumed to be negligible. Hence for maximum value of the carrier wave we then rewrite (2.41) as

$$\rho V \frac{d^{\,2}y_m}{dt^2} + 2\mu y_m\left(\frac{dy_m}{dt}\right) + \sigma y_m^2 = 0 \quad (2.42)$$

Now with the following boundary conditions that at time $t = 0$, $\lambda = 0$ in (2.37) – (2.39) we obtain

$$y_m = \left(a^2 - 2a^2\cos(-\varepsilon)\right)^{\frac{1}{2}} \quad (2.43)$$

$$y_m^2 = \left(a^2 - 2a^2\cos(-\varepsilon)\right) \quad (2.44)$$

$$\frac{dy_m}{dt} = n\,a^2 \sin(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{1}{2}} \quad (2.45)$$

$$\frac{d^{\,2}y_m}{dt^2} = n^2 a^2 \cos(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{1}{2}} - n^2 a^4 \sin^2(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{3}{2}} \quad (2.46)$$

When we substitute (2.43) - (2.46) into (2.42) we get

$$\rho V\left(\frac{n^2a^2\cos(-\varepsilon)}{(a^2-2a^2\cos(-\varepsilon))^{1/2}}-\frac{n^2a^4\sin^2(-\varepsilon)}{(a^2-2a^2\cos(-\varepsilon))^{3/2}}\right)+2\mu\left((a^2-2a^2\cos(-\varepsilon))^{1/2}\times\frac{na^2\sin(-\varepsilon)}{(a^2-2a^2\cos(-\varepsilon))^{1/2}}\right)$$

$$+\sigma\left(a^2-2a^2\cos(-\varepsilon)\right)=0 \qquad (2.47)$$

$$\rho V\left(\frac{n^2a\cos(-\varepsilon)}{(1-2\cos(-\varepsilon))^{1/2}}-\frac{n^2a\sin^2(-\varepsilon)}{(1-2\cos(-\varepsilon))^{3/2}}\right)+2\mu\left(na^2\sin(-\varepsilon)\right)+\sigma\left(a^2(1-2\cos(-\varepsilon))\right)=0 \qquad (2.48)$$

To linearize (2.48) we multiply through it by $(1-2\cos(-\varepsilon))^{\frac{3}{2}}$ such that

$$\rho V\left(n^2a\cos(-\varepsilon)(1-2\cos(-\varepsilon))-n^2a\sin^2(-\varepsilon)\right)+2\mu\left(na^2\sin(-\varepsilon)(1-2\cos(-\varepsilon))^{3/2}\right)+\sigma\left(a^2(1-2\cos(-\varepsilon))^{5/2}\right)=0$$

(2.49)

Note that $\cos(-\varepsilon)=\cos\varepsilon$ (even and symmetric function) and $\sin(-\varepsilon)=-\sin\varepsilon$ (odd and screw symmetric function), as a result (2.49) yields the following result.

$$\rho V\left(n^2a\cos\varepsilon(1-2\cos\varepsilon)-n^2a\sin^2\varepsilon\right)+2\mu\left(-na^2\sin\varepsilon(1-2\cos\varepsilon)^{3/2}\right)+\sigma\left(a^2(1-2\cos\varepsilon)^{5/2}\right)=0$$

(2.50)

Later we are going to utilize two types of approximation to reduce the fractional index in (2.50) since we are dealing with micro-vascular blood vessels.

2.2 Calculation of the elasticity of the blood medium (σ)

We know that the elasticity of the human aorta is about $\mu=10\times10^5\,dyne/cm^2=1\times10^5\,N/m^2$ or more explicitly written as $=1\times10^5\,kgm^{-1}s^{-2}$ since (1N=105dyne, 104 cm2 = 1m2). Given that the dynamic viscosity of the human blood is about $\eta=0.004kgm^{-1}s^{-1}$ and also the approximate angular frequency of the human heart is $f=1.2\,s^{-1}$. With the provision of these parameters we can calculate the elasticity of the blood medium σ from the equation

$$\sigma=\frac{\eta^2f^2}{\mu}=\frac{(0.004kg\,m^{-1}s^{-1})^2\times(1.2s^{-1})^2}{1\times10^5\,kgm^{-1}s^{-2}}=2.304\times10^{-10}\,kgm^{-1}s^{-2} \qquad (2.51)$$

2.3 Calculation of the phase angle (ε), angular frequency (n) and the amplitude (a) of the 'host wave'.

We have said that two types of approximation shall be utilized in order to linearize (2.50). These are the 'third and the fourth world approximation'. These approximations are the differential minimization of the resulting binomial expansion of a given variable function. These approximations have the advantage of converging results easily and also producing expected minimum value of results. Now the 'third world approximation' states that

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

(2.52)

While the 'fourth world approximation' states that

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

$$-n\frac{d}{d\phi}\left(\xi\,f(\phi)\right)-\frac{n(n-1)}{2!}\frac{d}{d\phi}\left(\xi\,f(\phi)\right)^2-\cdots \qquad (2.53)$$

Here ϕ is any variable function and ξ is some scalar number. The 'fourth world approximation' enhances minimum functional value since the amplitude of the constituted carrier wave would have to go through the smallest blood vessel – the capillaries. Hence by using the 'third world approximation' in (2.50) we get

$$\left(1-2\cos\varepsilon\right)^{3/2}=\left(1+(-2\cos\varepsilon)\right)^{3/2}=\left(0+3\sin\varepsilon-3\cos\varepsilon\sin\varepsilon+\ldots\right)-3\sin\varepsilon=-3\cos\varepsilon\sin\varepsilon \quad (2.54)$$

$$\left(1-2\cos\varepsilon\right)^{5/2}=\left(1+(-2\cos\varepsilon)\right)^{5/2}=\left(0+5\sin\varepsilon-15\cos\varepsilon\sin\varepsilon+\ldots\right)-5\sin\varepsilon=-15\cos\varepsilon\sin\varepsilon \quad (2.55)$$

Also by following the same algebraic subroutine in (2.50), the 'fourth world approximation' yields the below result.

$$\left(1-2\cos\varepsilon\right)^{3/2}=\left(1+(-2\cos\varepsilon)\right)^{3/2}=-\frac{3}{2}\cos^2\varepsilon\sin\varepsilon \quad (2.56)$$

$$\left(1-2\cos\varepsilon\right)^{5/2}=\left(1+(-2\cos\varepsilon)\right)^{5/2}=\frac{15}{2}\cos^2\varepsilon\sin\varepsilon \quad (2.57)$$

When we substitute (2.54) and (2.55) into (2.50) and after possible simplifications we realize that

$$\rho Vn^2a\left(\cos\varepsilon-2\cos^2\varepsilon-\sin^2\varepsilon\right)+a^2\left(6\mu n\cos\varepsilon\sin^2\varepsilon-15\sigma\cos\varepsilon\sin\varepsilon\right)=0 \quad (2.58)$$

We can now equate the coefficient of the terms in the parentheses to zero so that we get two separate results as

$$\left(\cos\varepsilon-2\cos^2\varepsilon-\sin^2\varepsilon\right)=0 \quad (2.59)$$

$$n=\frac{15\ \sigma\cos\varepsilon\sin\varepsilon}{6\mu\cos\varepsilon\sin^2\varepsilon}=\frac{15\ \sigma}{6\mu\sin\varepsilon} \quad (2.60)$$

Also by a similar substitution of (2.56) and (2.57) into (2.50) we get after a careful simplification

$$2\rho Vn^2a\left(\cos\varepsilon-2\cos^2\varepsilon-\sin^2\varepsilon\right)+a^2\left(6\mu n\cos^2\varepsilon\sin^2\varepsilon+15\sigma\cos^2\varepsilon\sin\varepsilon\right)=0 \quad (2.61)$$

$$a=\frac{-\left(\cos\varepsilon-2\cos^2\varepsilon-\sin^2\varepsilon\right)2\rho V\,n^2}{6\,\mu n\sin^2\varepsilon\cos^2\varepsilon+15\sigma\cos^2\varepsilon\sin\varepsilon} \quad (2.62)$$

Let us now solve for the critical value of the phase angle of the 'host wave' by using the relation $\cos\varepsilon=1-\frac{\varepsilon^2}{2}$ and $\sin\varepsilon=\varepsilon$ in (2.58), so that

$$\varepsilon^4-\varepsilon^2+2=0 \quad (2.63)$$

Upon solving for ε in (2.63) we get four roots as its possible solutions given by

$$\varepsilon_1=-0.9783+0.6761i\,;\ \varepsilon_2=-0.9783-0.6761i\,;\ \varepsilon_3=0.9783+0.6761i\,;\ \text{and}\ \varepsilon_4=0.9783-0.6761i \quad (2.64)$$

Thus a more realistic complex value of ε is $\varepsilon_3=0.9783+0.6761i$ and by converting the result from the complex value to a value in degree we get

$$\tan\varepsilon=\frac{0.6761}{0.9783}=0.6911\ \Rightarrow\ \varepsilon=\tan^{-1}(0.6911)=0.6109\ rad.\ (35^0) \quad (2.65)$$

$$n=\frac{15\times2.304\times10^{-10}kgm^{-1}s^{-2}}{6\times0.004\times\sin(0.6109)\,kgm^{-1}s^{-1}}=2.51\times10^{-7}\ rad./s \quad (2.66)$$

$$a=\frac{-\left(\cos(0.6109)-2\cos^2(0.6109)-\sin^2(0.6109)\right)2\rho V\,n^2}{6(2.51\times10^{-7})(0.004)\cos^2(0.6109)\sin^2(0.6109)+15(2.304\times10^{-10})\cos^2(0.6109)\sin(0.6109)} \quad (2.67)$$

$$a=\left(3202389831kg^{-1}ms^2\right)2\rho V\,n^2 \quad (2.68)$$

Now for the human ascending aorta whose radius $r = 0.015m$ and length $l = 0.07m$, then the volume V is

$$V = \pi r^2 l = 3.142 \times (0.015)^2 \times 0.07 = 4.94865 \times 10^{-5} m^3 \quad (2.69)$$

$$a = \left(3202389831 kg^{-1} m s^2\right) \times 2 \times 1050 kgm^{-3} \times 4.94865 \times 10^{-5} m^3 \times (2.51 \times 10^{-7} rad.s^{-1})^2 = 2.1 \times 10^{-6} m \quad (2.70)$$

Also the amplitude of the carrier wave in the human capillary can be calculated from (2.68). The length of the human capillary is about 600 X10-6m and the diameter is about 10X10-6 m (radius r = 5X10-6 m), the approximate value of the amplitude is $a = 4.99 \times 10^{-12} m$. However, we are not going to use this value in our computation since we have assumed that the blood spends a negligible time in the capillary during the energy exchange process

2.4 Calculation of the wave number or spatial frequency (k)of the 'host wave'.

We have made the assumption that for the carrier wave to have a maximum value then the spatial oscillating phase must be equal to 1, as a result

$$\phi = \cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - |\partial E|\right) = 1 \quad (2.71)$$

$$\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - |\partial E|\right) = 0 \quad (2.72)$$

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j + (k - k'\lambda)_z k \quad (2.73)$$

$$\vec{r} = xi + yj + zk \quad (2.74)$$

If we assume that the motion is constant in the z-direction and the wave vector mode is also the same for both x and y plane in the cylindrical system then with the usual transformation from Cartesian to polar coordinate system ($x = r\cos\theta \; and \; y = r\sin\theta$), we get

$$\vec{r} = r\cos\theta i + r\sin\theta j \quad (2.75)$$

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j \quad (2.76)$$

where $\theta = \pi - (\varepsilon - \varepsilon'\lambda)$ is the variable angle between y_1 and y_2 , please see appendix for details.

$$(\vec{k} - \vec{k}'\lambda).\vec{r} = (k - k'\lambda) r(\cos\theta + \sin\theta) \quad (2.77)$$

The constituted carrier wave can only have a maximum value provided the oscillating phase is equal to one, that is

$$\phi = \cos\left((k - k'\lambda)\; r(\cos\theta + \sin\theta) - (n - n'\lambda)t - |\partial E|\right) = 1 \quad (2.78)$$

$$\left((k - k'\lambda)r(\cos\theta + \sin\theta) - (n - n'\lambda)t - |\partial E|\right) = 0 \quad (2.79)$$

Using the boundary conditions that at time $t = 0$, $\lambda = 0$, $|\partial E| = \varepsilon = 0.6109 rad.$

$$\theta = \pi - (\varepsilon - \varepsilon'\lambda) = \pi - \varepsilon = 3.142 - 0.6109 = 2.5311 rad. \quad (2.80)$$

$$k\, r\left(\cos(2.5311) + \sin(2.5311)\right) - 0.6109 rad. = 0 \quad (2.81)$$

$$k = \frac{0.6109 rad.}{r\,(-0.2460945)} = \frac{0.6109 rad.}{0.015m \times (-0.2460945)} = 166 rad./m \quad (2.82)$$

Note that the radius of the human aorta r = 0.015m and we are working with the absolute value of k .

2.5 Calculation of the phase angle (ε'), angular frequency (n'), wave number (k') and amplitude (b)of the HIV/AIDS parameters

The gradual deterioration in the intrinsic parameters of the biological system of a HIV/ AIDS infected candidate would make us believe that after a sufficiently long period of time all the active constituents of the biological system of the host (Man) would have been completely eroded by the influence of the HIV, on the basis of this argument the below relation holds

$$\left.\begin{aligned} a-b\lambda=0 &\Rightarrow 2.1\times10^{-6}=b\lambda \\ n-n'\lambda=0 &\Rightarrow 2.51\times10^{-7}=n'\lambda \\ \varepsilon-\varepsilon'\lambda=0 &\Rightarrow 0.6109=\varepsilon'\lambda \\ k-k'\lambda=0 &\Rightarrow 166=k'\lambda \end{aligned}\right\} \quad (2.83)$$

Upon dividing the above relations in (2.83) with one another to eliminate λ, we get the following six relations.

$$\left.\begin{aligned} 8.3665n' &= b \\ 3.43755\times10^{-6}\varepsilon' &= b \\ 1.26506\times10^{-8}k' &= b \\ 4.10869\times10^{-7}\varepsilon' &= n' \\ 1.51204\times10^{-9}k' &= n' \\ 3.68012\times10^{-3}k' &= \varepsilon' \end{aligned}\right\} \quad (2.84)$$

Thus from all indications k' is related to ε' according to; $3.68\times10^{-3}k'=\varepsilon'$ or $3.68k'=10^{3}\varepsilon'$. Suppose we want to re-establish another relationship between k' and ε' then we can simply multiply through the sets of the relations in (2.84) by 3.68012×10^{3}. Consequently, once this is done then a more realistic and applicable relation for both quantities can be found from the 2nd and 3rd relations in (2.84) after equating them, that is, when $0.0126505985\varepsilon'=0.000046555k'$. Hence from simple ratio we can generally establish that

$$\varepsilon'=0.0000466rad.;\ k'=0.0127rad./m;\ n'=1.91\times10^{-11}rad./s;\ b=1.60\times10^{-10}m \quad (2.85)$$

Any of these estimated values of the HIV parameters shall produce a corresponding approximate value of the multiplicative factor $\lambda=13070$ upon substituting them into (2.83). Hence the range of themultiplicative factor is $0\le\lambda\le13070$.

2.6 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 intrinsic 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 σ (FC) in the basic parameters of the carrier wave function. Thereare 4 (four) attenuating parameters present in the carrier wave. Now, suppose a, n, ε, k represent the initial 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 final parameters of the 'host wave' that survives after a given time. Then, the FC is

$$\text{Fractional change, } \sigma=\frac{1}{4}\times\left(\left(\frac{a-b\lambda}{a}\right)+\left(\frac{\varepsilon-\varepsilon'\lambda}{\varepsilon}\right)+\left(\frac{n-n'\lambda}{n}\right)+\left(\frac{k-k'\lambda}{k}\right)\right) \quad (2.86)$$

$$\eta=\frac{FC\big|_{\lambda=i}-FC\big|_{\lambda=i+1}}{unit\,time(s)}=\frac{\sigma_i-\sigma_{i+1}}{unit\,time(s)} \quad (2.87)$$

The dimension is per second (s^{-1}). Thus (2.81) gives $\eta=0.0000763\,s^{-1}$ for all values of $\lambda\,(i=0,1,2,3,\ldots,13070)$.

2.7 Determination of the attenuation time (t)

We used the information provided in section 2.6, to compute the various times taken for the constitutive carrier wave to attenuate to zero. The maximum time the carrier wave lasted as a function of the raising multiplier λ is also calculated from the attenuation equation shown by (2.87). The reader should note that we have adopted a slowly varying regular interval for the multiplicative factor λ for our study. The slow varying interval we adopted will help to delineate clearly the physical parameter space accessible to our model. However, it is clear from the calculation that different attenuating fractional changes contained in the carrier wave function are approximately equal to one another. We can now apply the attenuation time equation given below.

$$\sigma = e^{-(\alpha \eta t)/\lambda} \quad (2.88)$$

$$t = -\left(\frac{\lambda}{\alpha \eta}\right) \ln \sigma \quad (2.89)$$

In this case, α is the HIV index factor andfor a non-local biological diseasesuch as the HIV $\alpha = 3$. However, the factor α is different for any other human biological diseases that are not localized. The equation is statistical and not a deterministic law, it gives the expected intrinsic parameters of the 'host wave' that survives after time t. Generally, we used the table scientific calculator and Microsoft Excel to compute our results. Also the GNUPLOT 4.6 version was used to plot the corresponding graphs.

III. PRESENTATION OF RESULTS

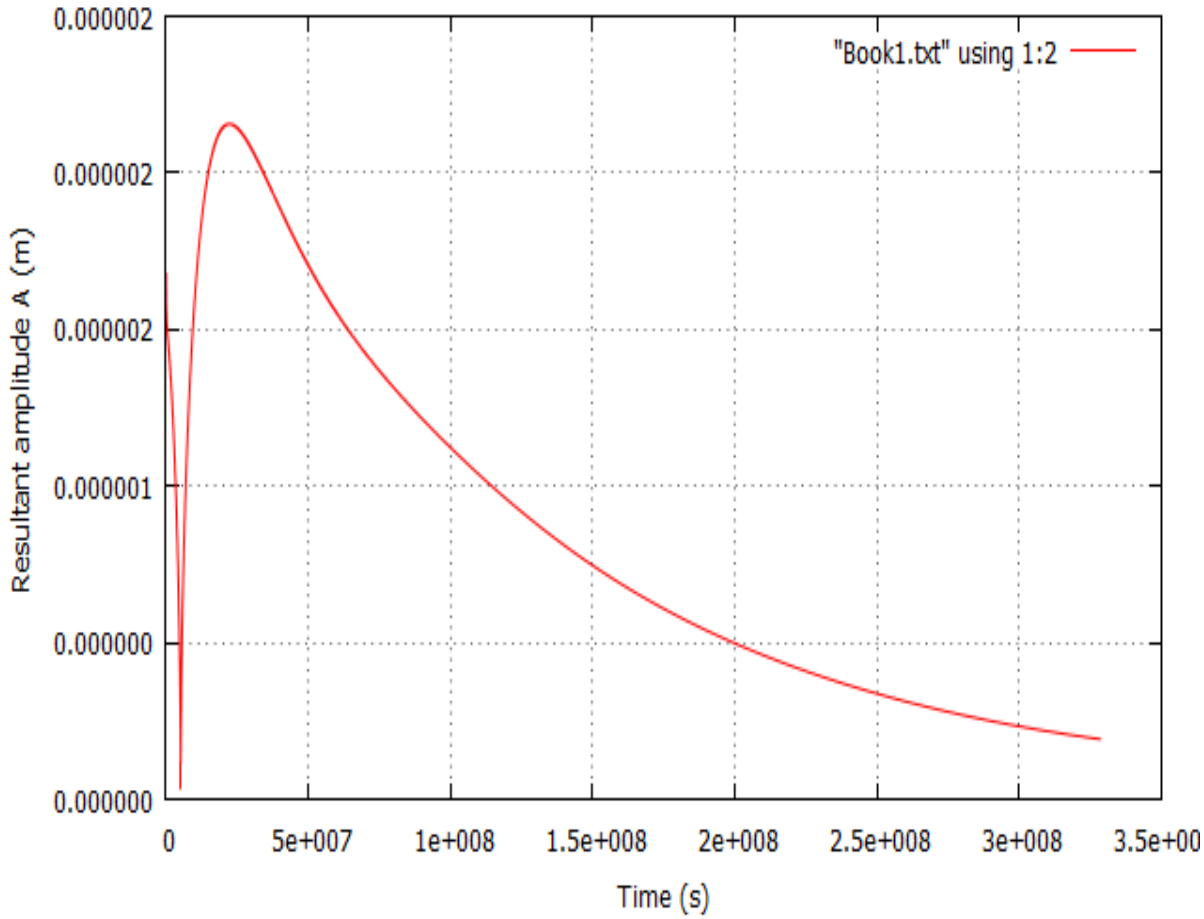

Fig. 1: Represents the graph of the resultant amplitude A of the carrier wave y as a function of time t.

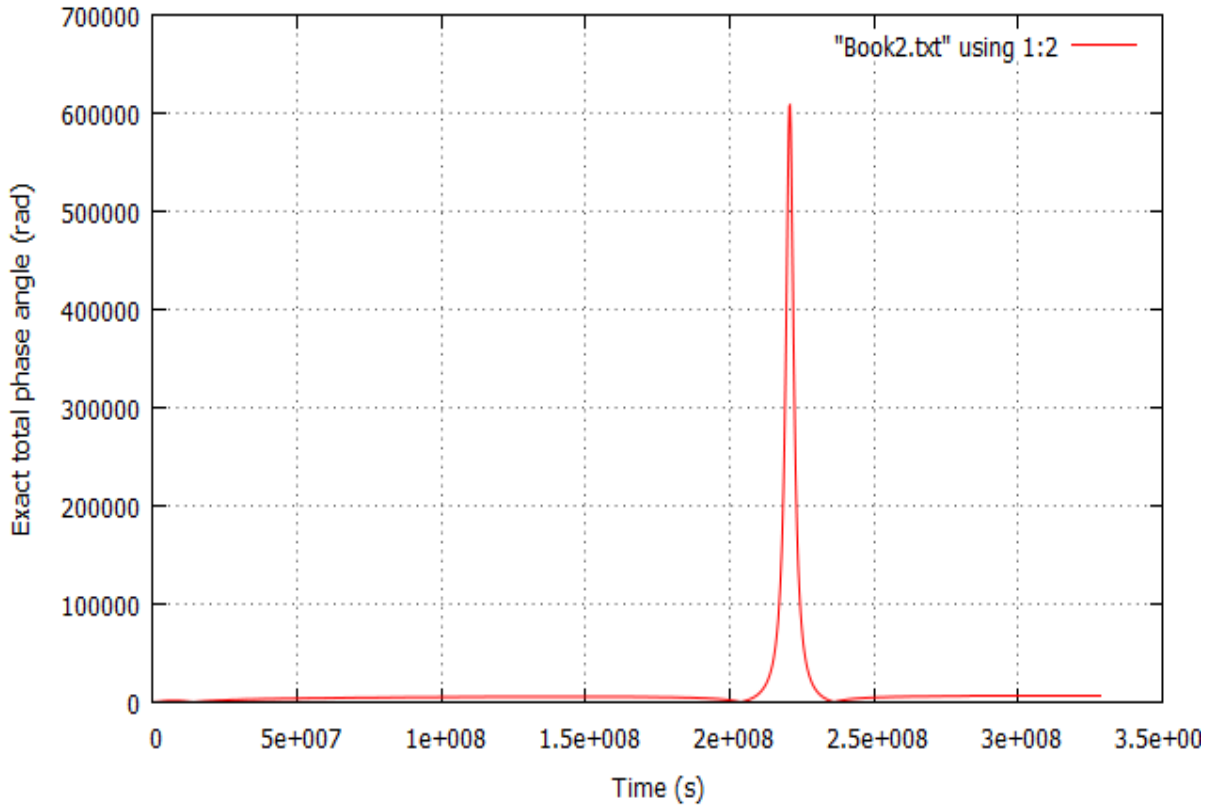

Fig. 2: Represents the graph of the exact total phase angle $|\partial E|$ of the carrier wave y as a function of time t .

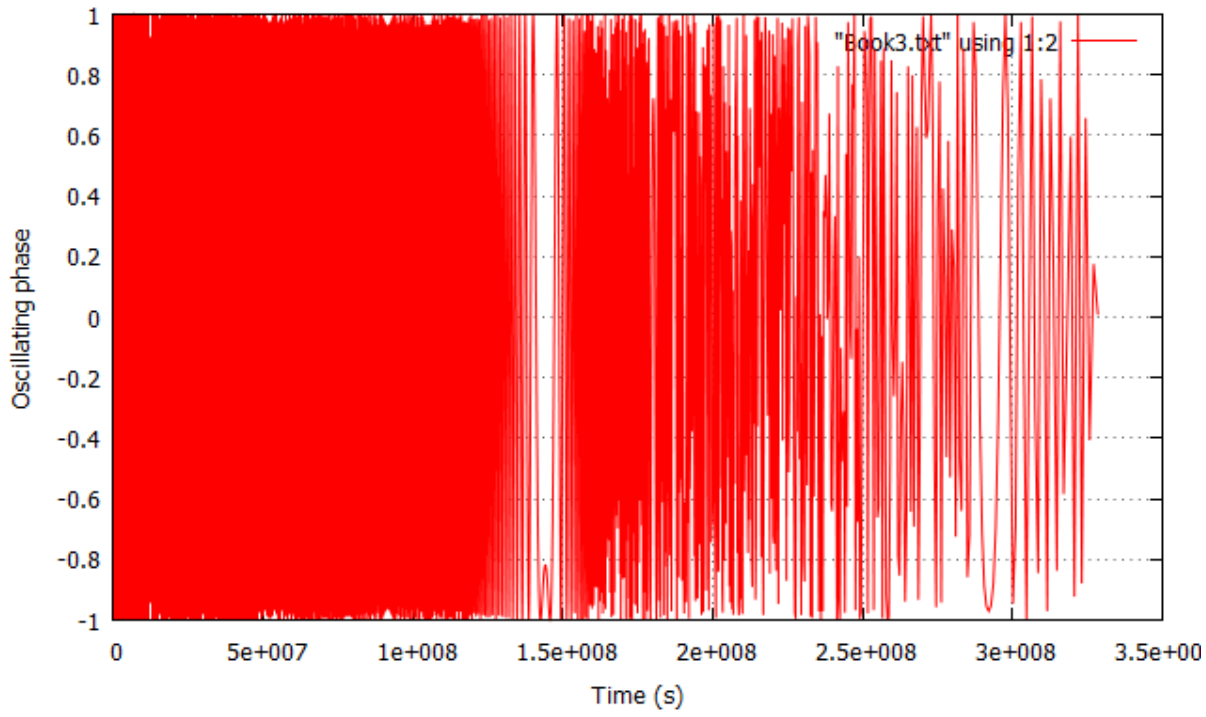

Fig. 3: Represents the graph of the spatial oscillating phase ϕ of the carrier wave y as a function oftime t .

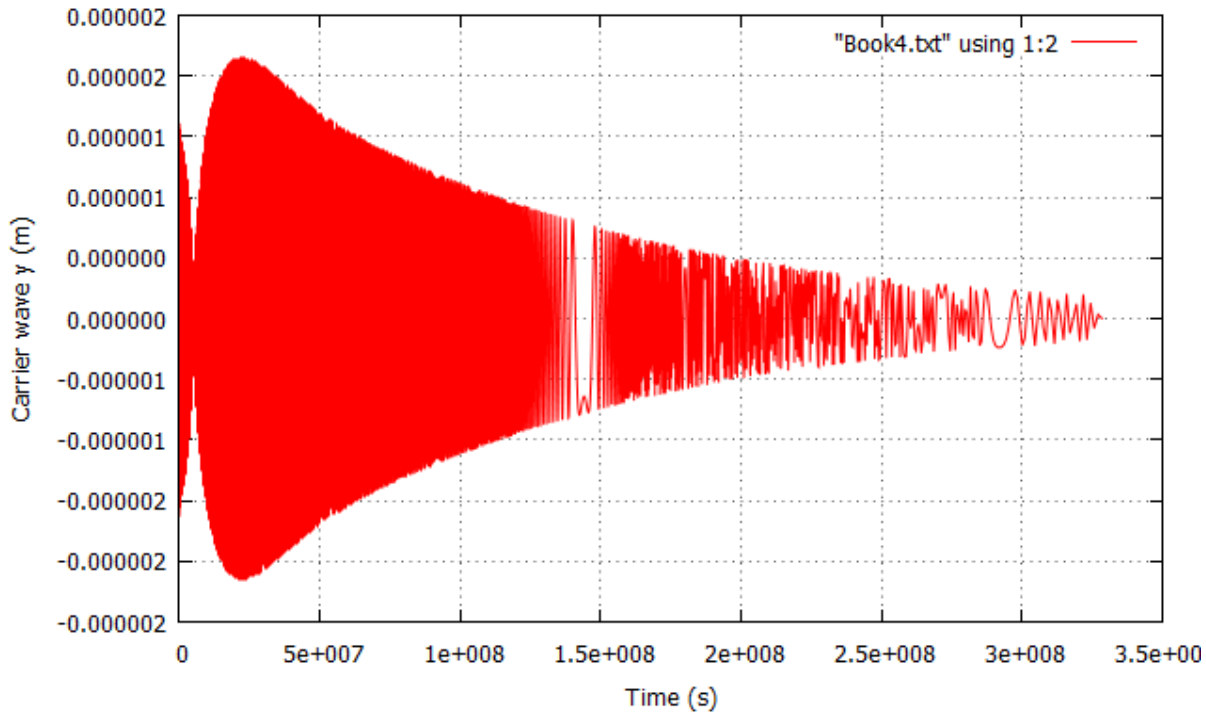

Fig. 4: Represents the graph of the carrier wave y as a function of time t .

IV. DISSCUSION OF RESULTS

It is clear from fig. 1, that within the interval of the multiplicative factor $0 \le \lambda \le 3627$ the absolute value of the resultant amplitude of the carrier wave first decreases consistently to a minimum value of 6.6564x10-9m when the time is 5133294s (2 months).When the multiplicative factor $3627 \le \lambda \le 6874$, the amplitude then rises with real values to a maximum value of 2.15862 10-6m at time t = 22837600s (8.8 months). This characteristic is also reflected in fig.4.Thus within the range of the raising multiplier $0 \le \lambda \le 6874$the HIV would have lasted for about 8 months in the human system. After this time, the amplitude steadily decreases exponentially to zero after a prolonged time of328479340s (10 years and 4 months).There is need for us to emphasize here that the initial value of the amplitude of the 'host wave' which we calculated to be 2.1X10-6 m is the static value. However, in practice due to the non-stationary oscillating behavior of the amplitude it turns out that the absolute value of the 'host wave' amplitude is 1.677X10-6 m.

From our calculation that within the interval of the multiplicative factor $0 \le \lambda \le 3627$and the time $0 \le t \le 5133294s$, the amplitude of the carrier wave initially experiences a steady imaginary decrease in value from $i1.6777 \times 10^{-6} m$ to $i6.6564 \times 10^{-9} m$. This behaviour is also reflected in the Fig.4. However, the reader

should note that it is the absolute values of the amplitude that we used in the graphical presentation. From our calculation therefore, it takes about 2 to 8 months for the HIV parasite to incubate before its absolute manifestation is felt. This according to the literature of clinical disease is the window period. The window period therefore, is the difference in time between the end of the imaginary value of the amplitude and the maximum positive value that the amplitude attained. In other words, the HIV 'parasitic wave' does not take immediate absolute effect on the human system when it is contacted. Within this interval of time, there is constant agitation by the intrinsic parameters of the 'host wave' to resist, thereby suppressing the destructive influence of the interfering HIV 'parasitic wave'. During this period, although unnoticeable as it may, but much imaginary harm would have been done to the constituent parameters of the biological system of the host (Man). Consequently, the amplitude of the carrier wave is actually made up of the imaginary and real part, $A = A_1 + iA_2$. This shows that the motion is actually two-dimensional (2D). Thus A_1 and A_2 are the components of the amplitude in x and y - directions, and A is tangential to the path of the moving amplitude in the carrier wave.

The graph of the exact total phase angle $|\partial E|$ ofthe carrier wave as a function of time as shown in fig. 2, almost show undefined behaviour everywhere except at time t = 220862000 s (7 years) and this occurs when $|\partial E|$ =604442 rad. The predominant behaviour of the carrier wave at this particular time is referred to as the wave packet. The wave packet is a localized pulse that is composed of both the 'host wave' and the 'parasitic wave' that cancel each other everywhere else except at this time during the interference. Since each component of the wave packet has different phase velocity in the medium, the modulation propagation number $(k - k'\lambda)$ of the components of the carrier wave changes in the medium and consequently the group velocity changes. This results to a change in the width of the wave packet.

It is clear from both figures that the events associated with the spatial oscillating phase and the carrier wave is similar. Initially, the spectra of fig. 3 and 4 are both blurred up to 4.5 years(144112000s) followed by a gradual depletion of the wave form. The blurred nature of the spectra is an indication of the resistance of the intrinsic parameters of the 'host wave' to the destructive tendency of the interfering 'parasitic wave'. While the subsequent depleting behaviour involves a steady decay process, resulting to a gradual reduction and weakening in the initial strength of the intrinsic parameters of the host biological system. The spatial oscillating phase oscillates between the maximum and minimum values of +1 and -1. In phasor language, for positive spatial oscillating phase y_1 leads y_2 and ε leads ε' (or ε' lags ε)in the CCW, while for negative value, y_2 leads y_1 and ε' leads ε (or ε lags ε').

As we can observe, the spectrum of fig. 3 and 4 show an exemplary behaviour or a sharp transition from one system phase to another around 1.44112×10^8 s (4.5 years).After a prolonged time, the spectra of figs. 3 and 4, again experiences a more pronounced depletion after 2.8x108s (8.8 years) and the constituents of the carrier wave becomes monochromatic in nature, that means a predominance of the 'host wave' after this time. In this case, the group velocity of the carrier wave becomes equal to the phase velocity.Consequently, it is obvious from both figures that the infected biological system could possibly degenerate from HIV infection to a phenomenon of AIDS between 4.5 years or 8.8 years in the absence of specific treatment.

The attenuating behaviour of the carrier wave in the interval $0 \le t \le 5133294\,s$ is a consequence of the fact that the carrier wave do not steadily go to zero, rather it fluctuates. The fluctuation is due to the constructive and destructive interference of both the 'host wave' and the 'parasitic wave' contained in the carrier wave. In the regions where the amplitude of the carrier wave is greater than either of the amplitude of the individual wave, we have constructive interference that means the path difference is $(\varepsilon + \varepsilon'\lambda)$, otherwise, it is destructive in which case the path difference is $(\varepsilon - \varepsilon'\lambda)$. If $n \approx n'$, then the average angular frequency say $(n + n')/2$ will be much more greater than the modulation angular frequency say $(n - n')/2$ and once this is achieved, then we will have a slowly varying carrier wave with a rapidly oscillating phase.

The spectrum of the displacement vector of the carrier wave as a function of time shown in fig. 4 is similar to that of fig. 1, since the carrier wave is the product of the resultant amplitude A and the oscillating phase ϕ. The exception to this similarity is that the carrier wave oscillates non-consistently with quadratic dispersion before it finally comes to rest.The carrier wave first decreases from the initial absolute value of 1.6777x10-6m to 2.05156x10-9m for a period of time t = 5133294s (2 months). Then the carrier wave oscillates to a maximum and minimum value of 2.14358x10-6m and -2.17386x10-6m respectivelyat a time t=22473700s (8.6 months).The displacement vector of the carrier wave decreases from the initial absolute value of 1.6777x10-6 m to 2.05156x10-9m. Hence the phenomenon called acquired immunodeficiency syndrome AIDS, actually occurs in the interval when the multiplicative factor $\lambda \ge 12803$and the time t $\ge$ 2.8x108s (8.8 years). Thus in this region, the system of the infected candidate can no longer recover from the HIV infection irrespective of any treatment administered. The resistance to treatment which means lack of immunity is as a result of the fact that all the active constituents of the 'host wave' would have been completely attenuated by the influence of the interfering 'parasitic wave'which is now the predominant constituent of the carrier wave.

Our calculation shows that in the absence of specific treatment, the HIV infection degenerates to AIDS after either4 years or 8 years and that is when the multiplicative factor $12803 \le \lambda \le 13070$. This period involves a steady decay process which results to a gradual reduction and weakening in the initial strength of the

intrinsic parameters of the host biological system. In this casethe displacement of the carrier wave which describes the coexistence of the biological system of Man and the HIV ceases to exist – the phenomenon called deatharound $328479340s$ (10 years)and the multiplicative factor λ would have attained the critical value of 13070.

V. CONCLUSION

Initially, the carrier wave spectrum shows a blurred characteristics followed by a gradual depletion of the wave form. The blurred nature of the resulting spectra is an indication of the resistance of the intrinsic parameters of the 'host wave' to the destructive tendency of the interfering 'parasitic wave'. While the subsequent depleting behaviour means a predominance of the 'parasitic wave'. After this time, a steady decay process resulting to a gradual reduction and weakening in the initial strength of the intrinsic parameters of the host biological system becomes prominent. The constituents of the carrier wave becomes monochromatic in nature since each component of the wave packet has different phase velocity in the medium, the modulation propagation number $(k - k'\lambda)$ of the components of the carrier wave changes in the medium and consequently the group velocity changes. This results to a change in the width of the wave packet. It seems from the results, that the actual dynamic components of the HIV responsible for their destructive tendency are $b\lambda$, $n'\lambda$, $\varepsilon'\lambda$ and $k'\lambda$. This study revealed that in the absence of specific treatment, people infected with HIV develop AIDS within 8 years and the average survival time after infection with HIV is found to be 8 to 10 years. Also the cessation of the carrier wave which describes the coexistence of the system of Man and the HIV is not instantaneous but gradual. Consequently, the life span of any biologically active system is determined by the resistance of its intrinsic parameters to the destructive influence of any internal or external factor. Finally, the significance of the HIV pandemic has been scientifically exaggerated, since the search strategy for the cure may lie outside the propounded complex scientific ideas and approach. Limitations in this work would include the use of some physical data such as the dynamic viscosity of blood whose values may not be exact based on small variations which differs from one individual to another. Secondly, the study relied on measures of sensitive physical parameters such as the radius and length of the human aorta, which may also differ slightly between individuals. Thus the findings presented here are approximate and may not be generalizable to the greater sense.APPENDIX: Vector representation of the superposition of the 'host wave' and the 'parasitic wave'. The amplitude of the CCW both waves are not constant with time but they oscillate at a given frequency.

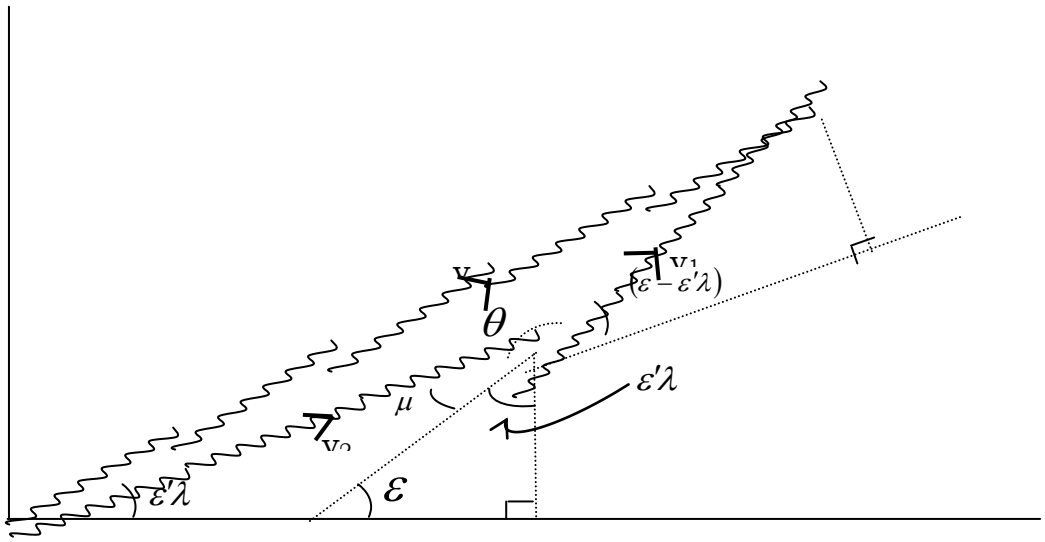

Fig. A 1:Represents the human 'host wave' y_1 and the HIV 'parasitic wave' y_2 after the interference. The superposition of both waves y_1 and y_2 is represented by the carrier wave displacement y. It is clear that from the geometry of the figure: $\mu + \varepsilon'\lambda + 180^0 - \varepsilon = 180^0$; $\mu = \varepsilon - \varepsilon'\lambda$; $\theta = 180^0 - (\varepsilon - \varepsilon'\lambda)$; and $\theta = \pi - (\varepsilon - \varepsilon'\lambda)$.

REFERENCES

[1] Sepkowitz, K. A. (2001) 'AIDS-the first 20 years'
[2] N. Engl. J. Med. 344 (23) : 1764 – 72.
[3] Mandel, Bennet, and Dolan (2010)
[4] HIV/AIDS – Wikipendia, Chapter 118.
[5] UNAIDS, WHO (December 2007) '2007 AIDS Epidemic Update' (PDF)
[6] The Aorta – Grays anatomy of the human body.
[7] http://education.yahoo.com
[8] Mette S. Olufsen, Charles S. Peskin, Won Yong Kim, Erik M. Pedersen, Ali Nadim and Jesper Larsen (2000). Numerical simulation and Experimental validation of Blood flow in Arteries with structured-Tree outflow conditions.
[9] Annals of Biomedical Engineering, Vol. 28, pp. 1281 – 1299.
[10] Stefan Anitei (softpedia) (December 2007). '7 Things about the heart and blood vessels
[11] www.google.com.ng
[12] Cutnel, John and Johnson, Kennet
[13] Physics, 4th Edition, Wiley, 1998, p 308.
[14] Alexander, R. W., Schlant, R. C., and Fuster, V. 'Hurst's the Heart, Arteries and Veins.
[15] 9th Edition McGraw-Hill, New York, NY. 1998.
[16] Glenn Elert (2008). 'Viscosity'
[17] http://hypertextbook.com/physics/matter/viscosity
[18] Tzu-Ching Shih et al. (2012) 'Numerical analysis of Coupled effects of pulsatile blood flow and thermal relaxation time during thermal therapy.
[19] International Journal of Heat and mass transfer, 55: 3763 – 3773
[20] DavidHalliday, Robert Resnick and Jearl Walker
[21] Fundamentals of Physics, 6th Edition, John Weley and Sons, Inc. New york 2001 : p378.
[22] Valenta J.(1993). In Biomechanics, clinical aspects of biomedicine, 2, Elsevier, Amsterdam.