The Biomechanics Of Hiv/Aids And The Prediction Of Lambda (λ)

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---ABSTRACT---
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) parameters. It is established in this study that when the HIV enters the human blood circulating system, it takes about 60 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 is $12803 \le \lambda \le 13070$. The time it takes the HIV infection to degenerate into AIDS if uncontrolled is about 7years (84months). The displacement of the carrier wave that describes the biological system of Man finally goes to zero - a phenomenon called death, when the multiplier approaches the critical value of 13070 and the time it takes to attain this value is about 11 years (132 months).

KEY WORDS: *latent vibration, carrier wave, 'host wave', 'parasitic wave', multiplicative factor, wave function.*

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1. INTRODUCTION

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 basicintrinsicparameters 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 theHIV.

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 5×10^{-6} m and 10×10^{-6} m, so that the red blood cells whose diameter is about 8×10^{-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 (600×10^{-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 notinclude 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 $0.003 kgm^{-1}s^{-1}$ and $0.004 kgm^{-1}s^{-1}$, the arterial blood perfusion rate (w_b) is $0.5 kgm^{-3}s^{-1}$ [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 waveform. 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 normalperson 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, ε 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 wavefunction 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 wavefunction 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 wavefunction 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 wavefunction 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 biomechanical behavior of blood in the blood vessels and to report the methodology developed in our labouratory to characterize the dynamics of the 'host wave' and those of 'HIV wave' in the constitutive carrier wave equation 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.

III. MATHEMATICAL THEORY OF SUPERPOSITION OF WAVES

Let us consider two incoherent waves defined by the non - stationary displacement vectors

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

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

where all the symbols retain their usual meanings. In this study, (2.1) is regarded as the 'host wave' whose propagation depends on the inbuilt raising multiplier $\beta\big(= 0, \ldots, 1\big)$. While (2.2) represents a 'parasitic wave' with a multiplicative factor or an inbuilt raising multiplier $\lambda\big(= 0, 1, 2, \cdots, \lambda_{\max}\big)$. The inbuilt multipliers are both dimensionless and as the name implies, they are capable of gradually raising the basic intrinsic parameters of both waves respectively with time. It is clear that the two equations are incoherent with different source functions.

Let us superpose (2.2) on (2.1), with the hope to realize a common wave function.

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

Suppose, we assume that for a very small parameter ζ, the below equation holds,

$$n'\lambda = \zeta + n\beta \tag{2.4}$$

$$y = a\beta\cos(\vec{k}\,\beta.\vec{r} - n\,\beta t - \varepsilon\beta) + b\lambda\cos(\vec{k}'\lambda.\vec{r} - n\beta t - \zeta t - \varepsilon'\lambda) \tag{2.5}$$

Again in (2.5), we let

$$\varepsilon_1' = \zeta\,t + \varepsilon'\lambda \tag{2.6}$$

$$y = a\beta\cos(\vec{k}\,\beta.\vec{r} - n\,\beta t - \varepsilon\beta) + b\lambda\cos(k'\lambda.\vec{r} - n\beta t - \varepsilon_1') \tag{2.7}$$

For the purpose of proper grouping we again make the following assumption:

$$\vec{k}\,\beta.\vec{r} = \vec{k}'\lambda.\vec{r} = \xi \tag{2.8}$$

$$\big(\vec{k}\beta - \vec{k}'\lambda\big).\vec{r} = \xi \tag{2.9}$$

$$y = a\beta\cos\big((\xi - n\,\beta t) - \varepsilon\,\beta\big) + b\lambda\cos\big((\xi - n\beta t) - \varepsilon_1'\big) \tag{2.10}$$

We can now apply the cosine rule for addition of angles to reevaluate each term in (2.10), that is,

$$\cos(A \pm B) = \cos A\cos B \mp \sin A\sin B \tag{2.11}$$

$$\begin{aligned} y = a\beta\big\{\cos(\xi - n\beta t)\cos\beta\,\varepsilon + \sin(\xi - n\beta t)\sin\beta\,\varepsilon\big\} + \\ b\lambda\big\{\cos(\xi - n\,\beta t)\cos\varepsilon_1' + \sin(\xi - n\,\beta t)\sin\varepsilon_1'\big\} \end{aligned} \tag{2.12}$$

$$\begin{aligned} y = a\beta\cos(\xi - n\beta t)\cos\beta\,\varepsilon + a\beta\sin(\xi - n\beta t)\sin\beta\,\varepsilon + \\ b\lambda\cos(\xi - n\,\beta t)\cos\varepsilon_1' + b\lambda\sin(\xi - n\,\beta t)\sin\varepsilon_1' \end{aligned} \tag{2.13}$$

$$y = \cos(\xi - n\beta t)\big\{a\beta\cos\beta\,\varepsilon + b\lambda\cos\varepsilon_1'\big\} + \sin(\xi - n\beta t)\big\{a\beta\sin\beta\varepsilon + b\beta\sin\varepsilon_1'\big\} \tag{2.14}$$

For technicality, let us make the following substitutions so that we can further simplify (2.14).

$$A\cos E = a\beta\cos\beta\varepsilon + b\lambda\cos\varepsilon_1' \tag{2.15}$$

$$A\sin E = a\beta\sin\beta\varepsilon + b\lambda\sin\varepsilon_1' \tag{2.16}$$

$$y = A\big\{\cos(\xi - n\beta t)\cos E + \sin(\xi - n\beta t)\sin E\big\} \tag{2.17}$$

$$y = A\cos\big\{\xi - n\beta t - E\big\} \tag{2.18}$$

$$y = A\cos\big\{(\vec{k}\beta - \vec{k}'\lambda).\vec{r} - n\beta t - E\big\} \tag{2.19}$$

The simultaneous nature of (2.15) and (2.16) would enable us to square though them and add the resulting equations term by term, so that

$$A^2\cos^2 E = a^2\beta^2\cos^2\beta\varepsilon + b^2\lambda^2\cos^2\varepsilon_1' + 2a\beta\lambda\cos\beta\varepsilon\cos\varepsilon_1' \tag{2.20}$$

$$A^2\sin^2 E = a^2\beta^2\sin^2\beta\varepsilon + b^2\lambda^2\sin^2\varepsilon_1' + 2ab\beta\lambda\sin\beta\varepsilon\sin\varepsilon_1' \tag{2.21}$$

$$A^2 = a^2\beta^2 + b^2\lambda^2 + 2ab\beta\lambda\big\{\cos\beta\varepsilon\cos\varepsilon_1' + \sin\beta\varepsilon\sin\varepsilon_1'\big\} \tag{2.22}$$

$$A^2 = a^2\beta^2 + b^2\lambda^2 + 2a\,b\beta\lambda\cos\big(\beta\varepsilon - \varepsilon_1'\big) \tag{2.23}$$

$$A^2 = a^2\beta^2 + b^2\lambda^2 + 2ab\beta\lambda\cos\big(\beta\varepsilon - (n'\lambda - n\beta)t - \varepsilon'\lambda\big) \tag{2.24}$$

$$A = \sqrt{a^2\beta^2 + b^2\lambda^2 + 2a\,b\,\beta\,\lambda\cos\big((\beta\varepsilon - \varepsilon'\lambda) + (n\beta - n'\lambda)t\big)} \tag{2.25}$$

$$y = \sqrt{a^2\beta^2 + b^2\lambda^2 + 2a\beta b\lambda \cos\left((\beta\varepsilon - \varepsilon'\lambda) + (n\beta - n'\lambda)t\right)} \times \cos\left((\vec{k}\beta - \vec{k}'\lambda).\vec{r} - n\beta t - E\right) \quad (2.26)$$

Upon dividing (2.16) by (2.15), we get that

$$\tan E = \frac{a\beta \sin \beta\varepsilon + b\lambda \sin \varepsilon_1'}{a\beta \cos \beta\varepsilon + b\lambda \cos \varepsilon_1'} \quad (2.27)$$

$$E = \tan^{-1}\left(\frac{a\beta \sin \beta\varepsilon + b\lambda \sin (\varepsilon'\lambda - (n\beta - n'\lambda)t)}{a\beta \cos \beta\varepsilon + b\lambda \cos (\varepsilon'\lambda - (n\beta - n'\lambda)t)}\right) \quad (2.28)$$

Hence (2.26) is the resultant wave function which describes the superposition of the 'parasitic wave' on the 'host wave'. Equation (2.26) representsa resultant wave equation in which the effects of the constitutive waves are additive in nature. However, suppose the effects of the constitutive 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 without loss of dimensionality we can recast (2.26) and (2.28) as

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

where we have redefined the amplitude and the total phase angle as

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

$$E = \tan^{-1}\left(\frac{a \sin \varepsilon - b\lambda \sin ((n - n'\lambda)t - \varepsilon'\lambda)}{a \cos \varepsilon - b\lambda \cos((n - n'\lambda)t - \varepsilon'\lambda)}\right) \quad (2.31)$$

The spatial oscillating phase is $\cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E\right)$. Equation (2.29) is 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.

3.1 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 if the spatial oscillating phase is equal to 1. Hence

$$y_m = \sqrt{\left(a^2 - b^2\lambda^2\right) - 2\left(a - b\lambda\right)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)} \quad (2.32)$$

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

$$(2.33)$$

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

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

$$(2.34)$$

The equation of motion obeyed by the constitutive carrier wave as it propagates along the human blood vessels experiences two resistive factors. Firstly, the resistance posed by the elasticity of the walls of the blood vessels, secondly, the elasticresistance of the blood medium where the carrier wave is propagating. The medium's mass and elasticity property determines how fast the 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 non idealities. We can therefore write the equation of motion as

$$F = -\mu\left(\frac{dy^2}{dt}\right) - \sigma\, y^2 \tag{2.35}$$

$$\rho V \frac{d^2 y}{dt^2} + 2\mu\, y\left(\frac{dy}{dt}\right) + \sigma\, y^2 = 0 \tag{2.36}$$

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 (m^3), μ is the dynamic viscosity of blood (kgm^{-1}s^{-1}), σ is the derived elasticity of the blood medium suitable for this study. 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 (2.36) as

$$\rho V \frac{d^2 y_m}{dt^2} + 2\mu\, y_m\left(\frac{dy_m}{dt}\right) + \sigma\, y_m^2 = 0 \tag{2.37}$$

Now with the following boundary conditions that at time $t = 0$, $\lambda = 0$ in (2.32) – (2.34) we obtain

$$y_m = \left(a^2 - 2a^2\cos(-\varepsilon)\right)^{\frac{1}{2}} \tag{2.38}$$

$$y_m^2 = \left(a^2 - 2a^2\cos(-\varepsilon)\right) \tag{2.39}$$

$$\frac{dy_m}{dt} = n\,a^2\sin(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{1}{2}} \tag{2.40}$$

$$\frac{d^2 y_m}{dt^2} = n^2\,a^2\cos(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{1}{2}} - n^2\,a^4\sin^2(-\varepsilon)\left(a^2 - 2a^2\cos(-\varepsilon)\right)^{-\frac{3}{2}} \tag{2.41}$$

When we substitute (2.38) - (2.41) into (2.37) we get

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

$$+ \sigma\left(a^2 - 2a^2\cos(-\varepsilon)\right) = 0 \tag{2.42}$$

$$\rho V\left(\frac{n^2 a\cos(-\varepsilon)}{(1 - 2\cos(-\varepsilon))^{1/2}} - \frac{n^2 a\sin^2(-\varepsilon)}{(1 - 2\cos(-\varepsilon))^{3/2}}\right) + 2\mu\left(na^2\sin(-\varepsilon)\right) + \sigma\left(a^2(1 - 2\cos(-\varepsilon))\right) = 0 \tag{2.43}$$

To linearize (2.43) we multiply through it by $\left(1 - 2\cos(-\varepsilon)\right)^{\frac{3}{2}}$ such that

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

$$\tag{2.44}$$

Note that $\cos(-\varepsilon) = \cos\varepsilon$ (even and symmetric function) and $\sin(-\varepsilon) = -\sin\varepsilon$ (odd and screw symmetric function),as a result (2.44) yields the following result.

$$\rho V\left(n^2 a\cos\varepsilon(1 - 2\cos\varepsilon) - n^2 a\sin^2\varepsilon\right) + 2\mu\left(-na^2\sin\varepsilon(1 - 2\cos\varepsilon)^{3/2}\right) + \sigma\left(a^2(1 - 2\cos\varepsilon)^{5/2}\right) = 0 \tag{2.45}$$

Later we are going to utilize two types of approximation to reduce (2.45).

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

We know that the elasticity of the human aorta is about $\mu = 10 \times 10^5\, dyne/cm^2 = 1 \times 10^5\, N/m^2$ or more explicitly written as $= 1 \times 10^5\, kgm^{-1}s^{-2}$ since (1N=10^{5}dyne, $10^4 cm^2 = 1m^2$). Given that the dynamic viscosity of the human blood is about $\eta = 0.004 kgm^{-1}s^{-1}$ and also the approximate angular frequency of the human heart is $f = 1.2\,s^{-1}$. With the provision of these parameterswe can calculate the elasticity of the blood medium σ from the equation

$$\sigma = \frac{\eta^2 f^2}{\mu} = \frac{(0.004 kg\ m^{-1} s^{-1})^2 \times (1.2 s^{-1})^2}{1 \times 10^5 kgm^{-1} s^{-2}} = 2.304 \times 10^{-10} kgm^{-1} s^{-2} \quad (2.46)$$

3.3   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 ordertolinearize (2.45). These are the 'third and the fourth world approximation'. Theseapproximations 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

$$(1 + \xi f(\phi))^{\pm n} = \frac{d}{d\phi}\left(1 + n\xi f(\phi) + \frac{n(n-1)}{2!}(\xi f(\phi))^2 + \frac{n(n-1)(n-2)}{3!}(\xi f(\phi))^3 + ...\right) - n\frac{d}{d\phi}(\xi f(\phi)) \quad (2.47)$$

While the 'fourth world approximation'states that

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

$$- n\frac{d}{d\phi}(\xi f(\phi)) - \frac{n(n-1)}{2!}\frac{d}{d\phi}(\xi f(\phi))^2 - \cdots \quad (2.48)$$

Here ϕ is any variable function and ξ is a scalar number. The 'fourth world approximation' enhances minimum functional value since the amplitude of the carrier wave would have to go through the smallest blood vessel – the capillaries. Hence using the 'third world approximation'in (2.45) we get

$$(1 - 2\cos\varepsilon)^{3/2} = (1 + (-2\cos\varepsilon))^{3/2} = (0 + 3\sin\varepsilon - 3\cos\varepsilon\sin\varepsilon + ...) - 3\sin\varepsilon = -3\cos\varepsilon\sin\varepsilon \quad (2.49)$$

$$(1 - 2\cos\varepsilon)^{5/2} = (1 + (-2\cos\varepsilon))^{5/2} = (0 + 5\sin\varepsilon - 15\cos\varepsilon\sin\varepsilon + ...) - 5\sin\varepsilon = -15\cos\varepsilon\sin\varepsilon \quad (2.50)$$

Also with asimilar implementation of the 'fourth world approximation', in (2.45) we get

$$(1 - 2\cos\varepsilon)^{3/2} = (1 + (-2\cos\varepsilon))^{3/2} = -\frac{3}{2}\cos^2\varepsilon\sin\varepsilon \quad (2.51)$$

$$(1 - 2\cos\varepsilon)^{5/2} = (1 + (-2\cos\varepsilon))^{5/2} = \frac{15}{2}\cos^2\varepsilon\sin\varepsilon \quad (2.52)$$

When we substitute (2.49) and (2.50) into (2.45) we realize that

$$\rho V n^2 a\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right) + a^2\left(6\mu n\cos\varepsilon\sin^2\varepsilon - 15\sigma\cos\varepsilon\sin\varepsilon\right) = 0 \quad (2.53)$$

We can now equate the coefficient of the terms in the parentheses to zeroso that we get separately

$$\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right) = 0 \quad (2.54)$$

$$n = \frac{15\ \sigma\cos\varepsilon\sin\varepsilon}{6\mu\cos\varepsilon\sin^2\varepsilon} = \frac{15\ \sigma}{6\mu\sin\varepsilon} \quad (2.55)$$

Also by a similar substitution of (2.51) and (2.52) into (2.45) we get after simplification

$$2\rho V n^2 a\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right) + a^2\left(6\mu n\cos^2\varepsilon\sin^2\varepsilon + 15\sigma\cos^2\varepsilon\sin\varepsilon\right) = 0 \quad (2.56)$$

$$a = \frac{-\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right)2\rho V n^2}{6\mu n\sin^2\varepsilon\cos^2\varepsilon + 15\sigma\cos^2\varepsilon\sin\varepsilon} \quad (2.57)$$

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 (2.54), so that

$$\varepsilon^4 - \varepsilon^2 + 2 = 0 \tag{2.58}$$

Upon solving for ε in (2.58) we get four roots as possible solutions to (2.58) given by

$\varepsilon_1 = -0.9783 + 0.6761i$; $\varepsilon_2 = -0.9783 - 0.6761i$; $\varepsilon_3 = 0.9783 + 0.6761i$; and $\varepsilon_4 = 0.9783 - 0.6761i$ (2.59)

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

$$\tan \varepsilon = \frac{0.6761}{0.9783} = 0.6911 \implies \varepsilon = \tan^{-1}(0.6911) = 0.6109 \, rad. \, (35^0) \tag{2.60}$$

$$n = \frac{15 \times 2.304 \times 10^{-10} kgm^{-1}s^{-2}}{6 \times 0.004 \times \sin(0.6109) kgm^{-1}s^{-1}} = 2.51 \times 10^{-7} \, rad./s \tag{2.61}$$

$$a = \frac{-\left(\cos(0.6109) - 2\cos^2(0.6109) - \sin^2(0.6109)\right) 2\rho V n^2}{6(2.51 \times 10^{-7})(0.004)\cos^2(0.6109)\sin^2(0.6109) + 15(2.304 \times 10^{-10})\cos^2(0.6109)\sin(0.6109)} \tag{2.62}$$

$$a = \left(3202389831 kg^{-1}m s^2\right) 2\rho V n^2 \tag{2.63}$$

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

$$V = \pi r^2 l = 3.142 \times (0.015)^2 \times 0.07 = 4.94865 \times 10^{-5} \, m^3 \tag{2.64}$$

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

$$\tag{2.65}$$

Also the amplitude of the carrier wave in the human capillary can be calculated from (2.63). Thelength of the human capillary is about 600 X10⁻⁶m and the diameter is about 10X10⁻⁶m (radius r = 5X10⁻⁶m),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.

3.4. 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

$$\cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E\right) = 1 \tag{2.66}$$

$$\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E\right) = 0 \tag{2.67}$$

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j + (k - k'\lambda)_z k \tag{2.68}$$

$$\vec{r} = xi + yj + zk \tag{2.69}$$

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

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j \tag{2.70}$$

$$\vec{r} = r\cos\theta \, i + r\sin\theta \, j \tag{2.71}$$

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

$$(\vec{k} - \vec{k}'\lambda).\vec{r} = (k - k'\lambda) r (\cos\theta + \sin\theta) \tag{2.72}$$

$$\left((k - k'\lambda)r(\cos\theta + \sin\theta) - (n - n'\lambda)t - E\right) = 0 \tag{2.73}$$

Using the boundary conditions that at time $t = 0$, $\lambda = 0$, $E = \tan^{-1}(\tan \varepsilon) = \varepsilon = 0.6109 \, rad.$

$$\theta = \pi - (\varepsilon - \varepsilon'\lambda) = \pi - \varepsilon = 3.142 - 0.6109 = 2.5311 rad. \tag{2.74}$$

$$k \, r \left(\cos(2.5311) + \sin(2.5311)\right) - 0.6109 rad. = 0 \tag{2.75}$$

$$k = \frac{0.6109 \, rad.}{r \, (-0.2460945)} = \frac{0.6109 \, rad.}{0.015m \times (-0.2460945)} = 166 \, rad./m \tag{2.76}$$

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

3.5 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

$$a - b\lambda = 0 \implies 2.1\times10^{-6} = b\lambda$$

$$n - n'\lambda = 0 \implies 2.51\times10^{-7} = n'\lambda$$

$$\varepsilon - \varepsilon'\lambda = 0 \implies 0.6109 = \varepsilon'\lambda \qquad\qquad \}\text{-------------------- (2.77)}$$

$$k - k'\lambda = 0 \implies 166 = k'\lambda$$

Upon dividing the above relations in (2.77) with one another to eliminate λ, we get the following six relations.

$$\implies 8.3665n' = b$$

$$\implies 3.43755\times10^{-6}\,\varepsilon' = b$$

$$\implies 1.26506\times10^{-8}\,k' = b$$

$$\implies 4.10869\times10^{-7}\,\varepsilon' = n' \qquad\qquad \}\text{ --------------------(2.78)}$$

$$\implies 1.51204\times10^{-9}\,k' = n'$$

$$\implies 3.68012\times10^{-3}\,k' = \varepsilon'$$

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 reestablish another relationship between k' and ε' then we can simply multiply through the sets of the relations in (2.78) by 3.68012×10^3. Consequently, once this is done then a more realistic and applicable relation for both quantitiescan be found from the 2nd and 3rd relations in (2.78) after equating them, that is when $0.0126505985\varepsilon' = 0.000046555k'$. Hence from simple ratio we can generally establish that

$$\varepsilon' = 0.0000466\,rad.$$

$$k' = 0.0127\,rad./m \qquad\qquad \}\text{-------------------- (2.79)}$$

$$n' = 1.91\times10^{-11}\,rad./s$$

$$b = 1.60\times10^{-10}\,m$$

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 (2.77). Note that for the interest of uniformity and anticipated complications we are using the minimum value which is 13070. Hence the range of the multiplier is $0 \le \lambda \le 13070$.Thus, so far, we have determined the intrinsic vibratingparameters of both the Man 'host wave' and those of the HIV 'parasitic wave'and both parameters arecontained in the constitutive carrier wave.

3.6 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

$$\text{Fractional change},\ \sigma = \frac{1}{4}\times\left(\left(\frac{a-b\lambda}{a}\right) + \left(\frac{\varepsilon-\varepsilon'\lambda}{\varepsilon}\right) + \left(\frac{n-n'\lambda}{n}\right) + \left(\frac{k-k'\lambda}{k}\right)\right) \qquad (2.80)$$

$$\eta = \frac{FC\big|_{\lambda=i} - FC\big|_{\lambda=i+1}}{unit\,time\,(s)} = \frac{\sigma_i - \sigma_{i+1}}{unit\,time\,(s)} \qquad (2.81)$$

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

3.7   Determination of the attenuation time (t)

We used the information provided in section 2.6, to compute the various times taken for the carrier wave to decay 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 in (2.81). The reader should note that we have adopted a slowly varying regular interval for the multiplicative factor or raising multiplier 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.

$$\sigma = e^{-(\alpha\eta t)/\lambda} \qquad (2.82)$$

$$t = -\left(\frac{\lambda}{\alpha\eta}\right)\ln\sigma \qquad (2.83)$$

In this case, α is the HIV factor and in this study $\alpha = 3$.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.Clearly, we used (2.83) to calculate the exact value of the decay time with respect to the increasing multiplicative factor. We used table scientific calculator and Microsoft excel to compute our results. Also the GNUPLOT 4.6 version was used to plot the corresponding graphs.
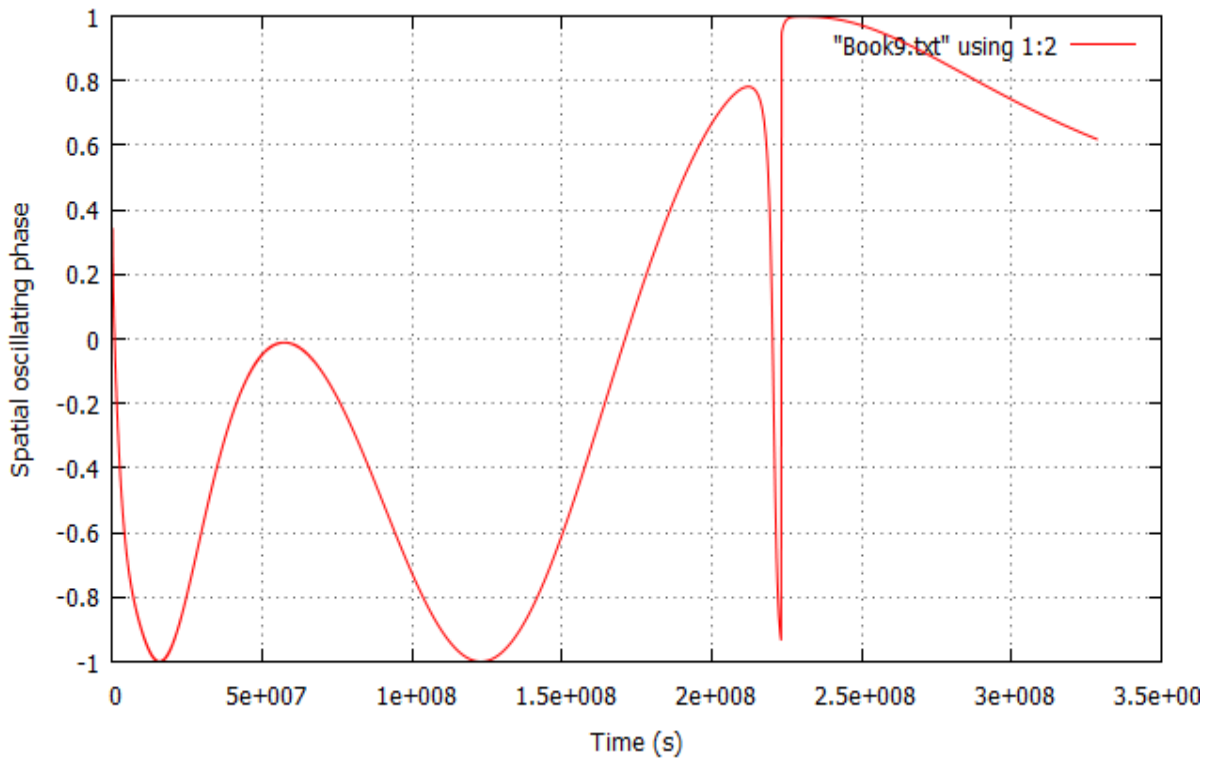
IV. PRESENTATION OF RESULTS

Fig. 3.1: This **r**epresents the graph of the spatial oscillating phase of the constitutive carrier wave against time t .
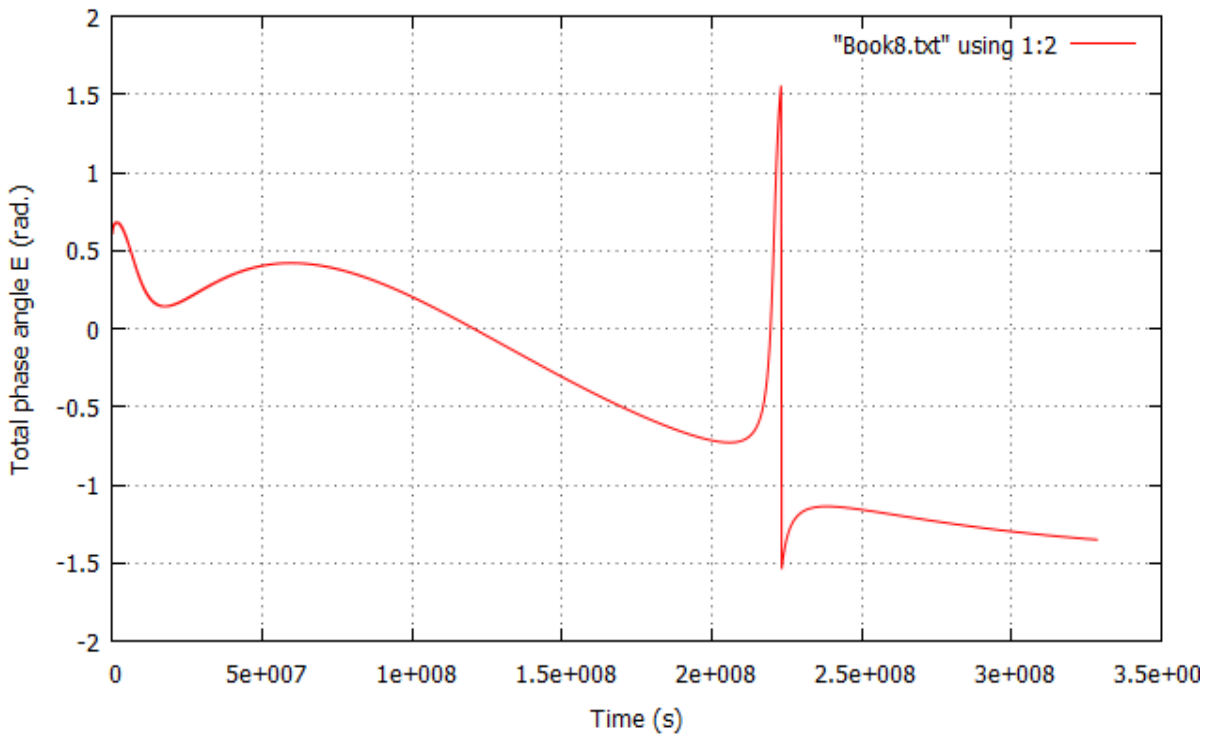

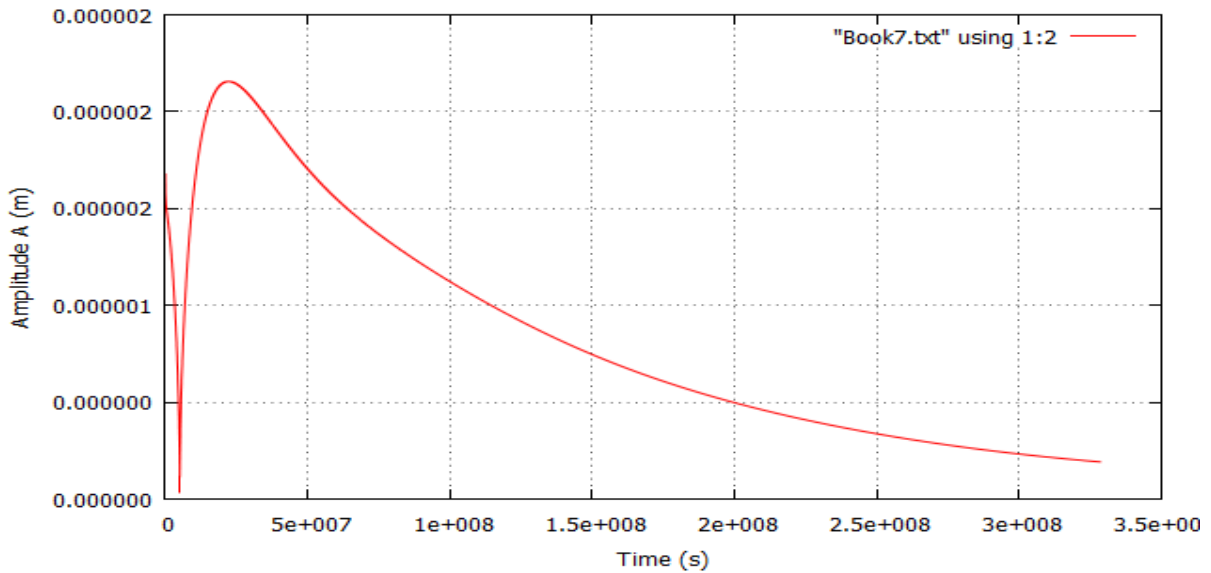**Fig. 3.2:** This **r**epresents the graph of the total phase angle E of the constitutive carrier wave y against time t .

Fig. 3.3: This **r**epresents the graph of the amplitude A of the constitutive carrier wave y against time t.
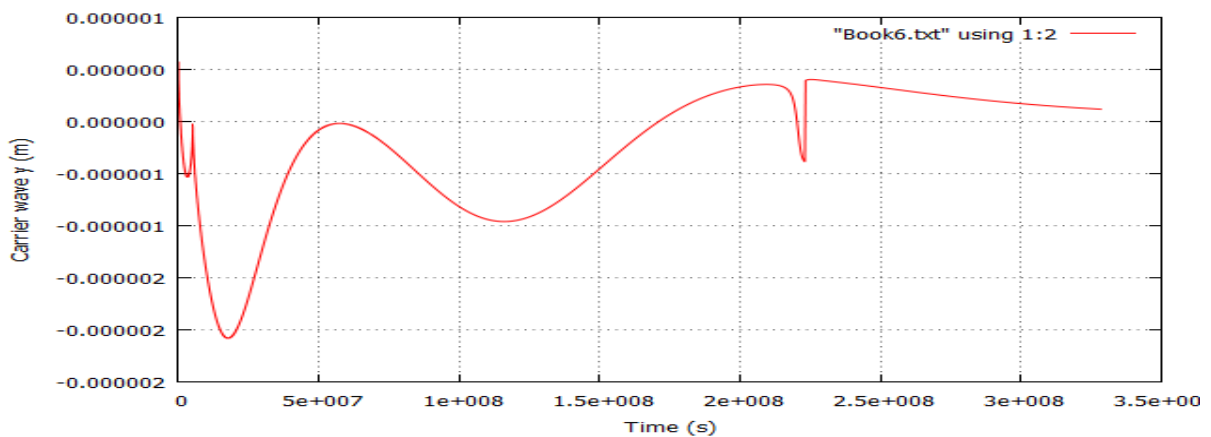

Fig. 3.4: This **r**epresents the graph of the constitutive carrier wave y displacement against time t.

V. DISSCUSION OF RESULTS

The figures show exemplary behavior or a sharp transition from one system phase to another in the interval when the 2×10^8 $s \leq t \leq 2.5 \times 10^8$ s. This behavior is reflected in figs. 3.1, 3.2 and 3.4. The events associated with the spatial oscillating phase and thecarrier waveissimilar. It is clear from the figure that the spatial oscillating phase oscillates between +1 and -1. The phase first increases negatively in values before it begins to fluctuate between positive and negative values. The fluctuation ends around $t = 2.25 \times 10^8 s$ (7 years) and then it increases to a maximum value of +1 and thereafter it decreases exponentially. The Fig. 3.2 shows that within the first 4 years the total phase angle E experiences irregular values, it increases and decreases within the interval of the multiplicative factor $0 \leq \lambda \leq 11837$. The total phase angle increases negatively in value within the value range of $-0.00039664 \, rad. \leq E \leq -0.72952239 \, rad.$, when the multiplicative factor interval is $11838 \leq \lambda \leq 12784$. In this regime the phase angle of the 'host wave' are in constructive interaction with that of the 'parasitic wave'.When the multiplicative factor is within the interval $12785 \leq \lambda \leq 12849$, the total phase angle of the carrier wave decreases negatively from $-0.72948871 \, rad.$ to $-0.04401858 \, rad..$ In this regime the phase angle of the 'host wave' is losing adherence with that of the 'parasitic wave'.When the HIV is about 7 years in the system of the infected candidate, the total phase angle then increases positively with a value which ranges from $0.04401858 \, rad.$ to $1.54715744 \, rad.$ and the multiplier is $12850 \leq \lambda \leq 12864$. It

decreases negatively again with a value $-1.54680027\,rad. \leq E \leq -1.1386545\,rad.$ when the multiplicative factor is $12865 \leq \lambda \leq 12919$. Finally, when the multiplier is $12920 \leq \lambda \leq 13070$, the total phase angle increases negatively with a value range of $-1.1386559\,rad. \leq E \leq -1.3510916\,rad.$. This is when the HIV infection is about 11 years in the biological system of the host. The total phase angle of the constitutive carrier wave has a maximum value of $1.54715744\,rad.$ and a minimum value of $-1.54680027\,rad.$.

It is clear from Fig. 3.3, that within the interval of the multiplicative factor $0 \leq \lambda \leq 3627$ and the time $0 \leq t \leq 5133294s$, 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. 3.4. However, it is the absolute values of the amplitude that we used in the graphical presentation. The time taken for this imaginary decrease in the amplitude is about 60 days. This is the said window period as it is referred to in the literature of clinical disease. In other words, the HIV 'parasitic wave' does not take immediate absolute effect on the human system when it is contacted.

Hence it takes about 60 days for the HIV parasite to incubate before its absolute manifestation is felt. Within this time, there is constant agitation by the intrinsic parameters of the 'host wave' to resist thereby suppressing the destructive influence of the interfering 'parasitic wave'. The effect of the imaginary decay in the amplitude is unnoticeable or inadequately felt by the human system in this interval. Although, unnoticeable as it may, but much imaginary harm would have been done to the constituent parameters of the biological system of the host. The value of the amplitude then experiences absolute fluctuations in the interval $3628 \leq \lambda \leq 6878$, and time $5136381s \leq t \leq 22351292s$. The amplitude of the carrier wave decreases consistently from the value $2.1563 \times 10^{-6}\,m$ to $1.9168 \times 10^{-7}\,m$ in the interval of the multiplicative factor $6879 \leq \lambda \leq 13070$ and time $22359365s \leq t \leq 328616068s$. The non-consistent attenuating behavior in this interval is a consequence of the fact that the amplitude of 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 (the 'host wave' and the 'HIV wave'), we have constructive interference, otherwise, it is destructive. 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. This region of imaginary values of the amplitude is an indication of the human system to guide and renormalizes the system against the effect of the interfering HIV 'parasitic wave'. There is need for us to emphasize here that the initial value of the amplitude of the 'host wave' which we calculated to be 2.1×10^{-6}m is the static value.

However, in practice due to the non-stationary oscillating behavior of the 'host wave', it turns out that the absolute value of the amplitude is 1.677×10^{-6}m with a statistical error of 2.1×10^{-6}m $\pm 4.223 \times 10^{-7}$m. The graph of the carrier wave against time is shown in fig. 3.4. Of course we know that the carrier wave is the product of the amplitude and the spatial oscillating phase. From the figure, the displacement of the carrier wave first experiences a constant imaginary decrease with a value range of $5.70747 \times 10^{-7}\,m \leq y \leq 3.96221 \times 10^{-10}\,m$ in the interval of the multiplier $0 \leq \lambda \leq 1395$, and time $0 \leq t \leq 685843s$. The carrier wave then fluctuates with negative values in the interval $1396 \leq \lambda \leq 12534$, and time $686856\,s \leq t \leq 171043126\,s$. The negative values of the fluctuation experienced by the carrier wave in this region is an indication of attraction and hence constructive interference between the intrinsic parameters of the 'host wave' and those of the HIV 'parasitic wave'. Then again the carrier wave fluctuates with positive values when the multiplier is $12535 \leq \lambda \leq 12802$ and the time $171151701s \leq t \leq 209590526s$ or ($5\ years \leq t \leq 7\ years$). Because of the significant behavior of the carrier wave in this interval we believe that the HIV infection becomes more intensive and pronounced in the biological system of an infected individual during the first five years.

In the absence of specific treatment, the HIV infection suddenly degenerates to AIDS after 7 years. 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 case, the displacement of the carrier wave

consistently decreasesfrom the value of $3.5671 \times 10^{-7}m$ to $1.18698 \times 10^{-7}m$. Hence the phenomenon calledacquired immunodeficiency syndrome AIDS, actually occurs in the interval when the multiplicative factor and the time is about $12803 \le \lambda \le 13070$ and $209787918s \le t \le 328616068s$ or ($7 \ years \le t \le 11 \ years$). 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 totreatment 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'. The displacement of thecarrier wave which describes the coexistence of the biological system of Man and the HIV ceases to exist –the phenomenon called death, around 11 years ($328616068s$) when $\lambda = 13070$.

VI.   CONCLUSION

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 7 years and the average survival time after infection with HIV is found to be 7 to 11 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. Initially the biological system of Man tends to annul the destructive influence of the HIV starting from the moment an individual contacted it. Consequently, the existence or the life span of any physically 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 during experimental measurement. Secondly, the study relied on measurements of sensitive physical parameters such as the radius and length of the human aorta, which may differ slightly between individuals. Which means that the findings presented here may not be generalizable to the greater sense except if one can be provided with uninterrupted physically measured data.

APPENDIX: Vector representation of the superposition of the wave function of Man (host) and that of the HIV.
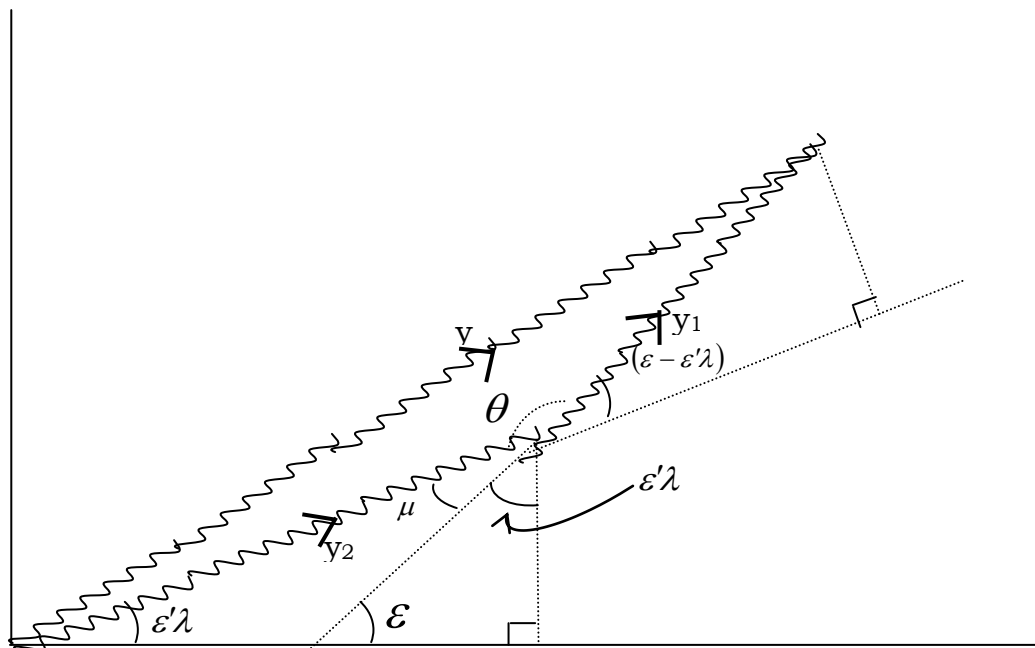
Fig. A 2. This represents the human 'host wave' y_1 and the HIV 'parasitic wave' y_2 after the interference. The superposition of both waves y_1 and y_2 is represented by the carrier wave displacement y. It is clear thatfrom the geometry of the figure: $\mu + \varepsilon'\lambda + 180^0 - \varepsilon = 180^0$; $\mu = \varepsilon - \varepsilon'\lambda$; $\theta = 180^0 - (\varepsilon - \varepsilon'\lambda)$; and $\theta = \pi - (\varepsilon - \varepsilon'\lambda)$. Note that θ is the variable angle between y_1 and y_2.

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