International Journal of Scientific and Innovative Mathematical Research (IJSIMR)
Volume 4, Issue 7, July 2016, PP 12-23
ISSN 2347-307X (Print) & ISSN 2347-3142 (Online)
DOI: http://dx.doi.org/10.20431/2347-3142.0407003
www.arcjournals.org

Perturbation-Iteration Method for Solving Mathematical Model of Hypoxemic Hypoxia Tissue-Blood Carbon Dioxide Exchange during Physical Activity Activity

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Abstract*: This paper aims at solving of a mathematical model of hypoxemic hypoxia tissue-blood carbon dioxide exchange using a new recent method: Perturbation-iteration method. The description of this method for different order derivatives in the Taylor Series expansion is discussed. This method provides the solution in the form of an infinite series for ordinary differential equation. The efficiency of the method used is investigated by a comparison of Euler method and Runge Kutta. Numerical simulations of all these three methods are implemented in Matlab. The validation has been carried out by taking the values of determinant parameters for a 30 years old woman who is supposed to make practice of three regular physical activities: Walking, Jogging and Running fast. The results are in good agreement with experimental data.*

Keywords: *Hypoxic hypoxia, Perturbation iteration method, tissue, oxygen, Physical activity, Numerical simulation*

1. INTRODUCTION

Hypoxia, or hypoxiation, is defined as a pathological condition related to adequate oxygen supply in human body. It is in two main types: the generalized hypoxia that is characterized by the deprived adequate oxygen supply in whole body and tissue hypoxia which happens in its region. It differs from hypoxemia called also hypoxaemia in that within the arterial blood the oxygen concentration is abnormally low. Hypoxemia was originally defined as a deficiency of oxygen in arterial blood but standard manuals take this to mean an abnormally low partial pressure of oxygen, content of oxygen or percent saturation of hemoglobin with oxygen, either found singly or in combination. The serious cases of the hypoxemia happen when the decreased partial pressure of oxygen in blood is less than $60 mmHg$. The reason of this is this point constitutes the beginning of the steep portion of the hemoglobin dissociation curve, where a small decrease in the partial pressure of oxygen results in a large decrease in the oxygen content of the blood or when hemoglobin oxygen saturation is less than 90%.

In addition, the generalized hypoxia occurs in healthy people when they ascend to high altitude, where it causes altitude sickness leading to potentially fatal complications including high altitude pulmonary edema (HAPE) and high altitude cerebral edema (HACE) [1]. It also occurs in healthy individuals when breathing mixtures of gases with a low oxygen content.

Hypoxic hypoxia is a result of insufficient oxygen available to the lungs. The examples of how lungs

can be deprived of oxygen are a blocked airway, a drowning or a reduction in partial pressure (high altitude above 10,000 feet). Hypoxia is also a serious consequence of pre-term birth in the neonate. The main cause for this is that the lungs of the human fetus are among the last organs to develop during pregnancy. To assist the lungs to distribute oxygenated blood throughout the body, infants at risk of hypoxia are often placed inside an incubator capable of providing continuous positive airway pressure (also known as a humidicrib). The insufficient delivery of oxygen (low Pa_{O_2}) or inability to utilize oxygen (normal Pa_{O_2}) causes also the hypoxia where we assist to oxygen deficiency at the mitochondrial sites. This phenomenon occurs when Pa_{O_2} less than $7.3kPa$ $(55mmHg)$. Below this threshold the ventilation starts to stimulate carotid body activity. The hyperventilation reduces Pa_{CO_2} and $[H^+]$, which limits the initial rise in ventilation, because it decreases the carotid body and central chemoreceptor stimuli. In fact, in humans, hypoxia is detected by chemoreceptors in the carotid body. This response does not control ventilation rate at normal Pa_{O_2}, but below normal the activity of neurons innervating these receptors increases dramatically, so much so to override the signals from central chemoreceptors in the hypothalamus, increasing Pa_{O_2} despite a falling Pa_{CO_2}.

Any physical activity obviously causes the body to demand more oxygen for normal functioning. The muscles rob the brain of the marginal amounts of oxygen available in the blood and the time of onset of hypoxic symptoms is shortened. However, the improvement of performance of athlete in high altitude results in a mild and non-damaging intermittent hypoxia used intentionally during training to develop an athletic performance adaptation at both the systemic and cellular level. Mathematical models quantifying hypoxic hypoxia have been proposed [2], [3], [4] and [5]. All these mathematical models are governed by ordinary differential equations. The numerical resolution of mathematical models of governing ordinary differential equations can be done using different numerical methods. This paper focuses on a mathematical model for hypoxemic hypoxia where the role of physical activity is taken into account [6] where the resolution is done using Perturbation Iteration method which is a recent method that can be used has been derived in [7].

The remainder of this paper is structured as follows. In section 2, we present the mathematical model of ordinary differential equations. The section 3 deals with the the basic idea of the Perturbation Iteration method. The numerical simulation is presented in section 4 where the comparison is done using Euler method and Runge Kutta method to test efficiency of perturbation iteration method. The section 5 focuses on discussion while Section 6 rounds up and deals with the concluding remarks.

2 THE MATHEMATICAL MODEL EQUATIONS

The model we present in this paper involves a modification of model equations as developed by Guillermo Gutierrez [2] in order to include the role of physical activity. The new model equations have been given in [6] where the diagram for a two compartmental model is illustrated in the figure 1.

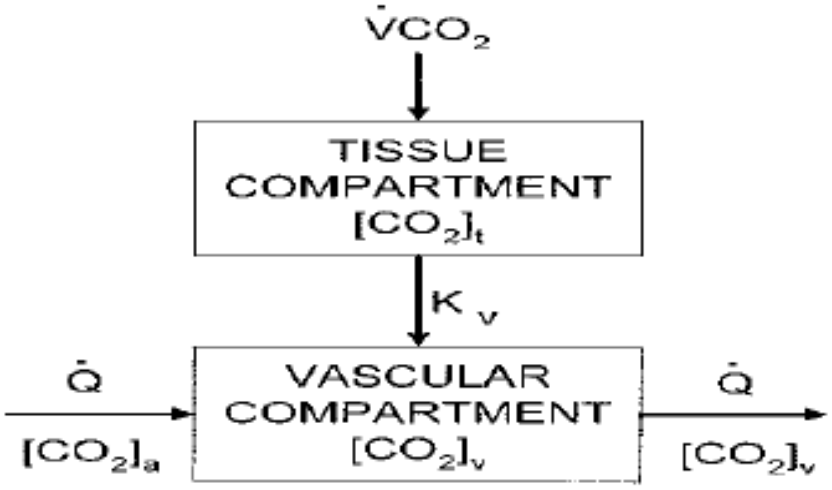

Figure 1. *Diagram for the tissue carbon dioxide (CO_2) exchange model where $[CO_2]$ represents the total CO_2 concentration (dissolved and bound) and the subscripts and denote the tissue and vascular compartments respectively. K_v is the mass transfer coefficient for CO_2.*

For the vascular compartment, the rate of change of $[CO_2]v$ depends on blood flow per unit volume of tissue $(\dot{Q})$. $\dot{V}CO_2$ denotes carbon dioxide production**.**

The mass transport model of tissue CO_2 exchange is developed to examine the relative contributions of blood flow and cellular hypoxia (dysoxia) to increases in tissue and venous blood CO_2 concentration. The model equations we are interested in are (See [6] for details)

$$\frac{d[CO_2]_t}{dt} = RQ \times H \times SV \times ([O_2]_a - [O_2]_v) - K_v([CO_2]_t - [CO_2]_v) \tag{1}$$

$$\frac{d[CO_2]_v}{dt} = \dot{Q}\left[\left(K_{CO_2} \times \frac{\dot{V}CO_2}{\dot{V}_A} K + k_{CO_2}\right) - [CO2]_v\right] + K_v([CO2]_t - [CO2]_v), \tag{2}$$

where CO_2 is carbon dioxide with $[CO_2]$ represents the total CO_2 concentration (dissolved and bound) and the subscripts t and v denote the tissue and vascular compartments respectively. K_v is the mass transfer coefficient for CO_2. For the vascular compartment, the rate of change of $[CO_2]$ depends on blood flow per unit volume of tissue $(\dot{Q})$. $\dot{V}CO_2$ denotes carbon dioxide production. The integral role of physical activity results in the influence of the demand of the tissues for oxygen during hypoxia in altitude. Just as resting ventilation increases dramatically at high altitude, so does ventilation during physical activity. Since carbon dioxide production for a given work level is essentially independent of altitude, this means that measured ventilation is independent of altitude at a given work level. At work levels approaching maximal values at any altitude, alveolar falls compared with the resting level and physical activity ventilation measured at correspondingly rises. Furthermore, during exercise, increases in alveolar ventilation must parallel the increased tissue oxygen consumption and carbon dioxide production by the exercising muscles, both of which rise in direct proportion to the increase in power output. In addition, it is known that the human respiratory control system varies the ventilation rate $\dot{V}_A$ in response to the levels of CO_2 in the body and the control mechanisms of cardiovascular system influences global control in the blood vessels as well as well as heart rate H for impacting blood flow $\dot{Q}$ [8] and [9]. Generally, during physical activity in altitude and particular in the hypoxia case, the control mechanism of these two systems plays a crucial role.

3 PERTURBATION ITERATION ALGORITHM (PIA)

Perturbation iteration method has been developed recently by Aksoy and al. [7]. This new method of solving a system of first order of nonlinear ordinary differential equations uses a combination of perturbation expansions and Taylor series expansions to give rise to an iteration scheme where Aksoy and al. [7] and Pakdemirli [10] introduced expansion and correction terms of only first derivatives in the Taylor series expansion and one correction term in the perturbation. Therefore, the perturbation iteration algorithm is named by $PIA(1,1)$. Let us be interested in the description of $PIA(1,1)$.

First of all, we discuss the PIA(1,m) which is constructed by taking one correction term in the perturbation expansion and correction terms of m'th order derivatives in the Taylor Series expansion. We consider a system of first order of K nonlinear ordinary differential equations. We note

$$x = (x_1, x_2, ..., x_K)^T$$

a vector state. The system first order of K nonlinear ordinary differential equations can be written as follows

$$E_k \equiv E_k(\dot{x}_k, x_j, \varepsilon, t) = 0, \quad k = 1,2,..., K, \quad j = 1,2,..., K$$

where ε is the perturbation parameter and t denotes the independent variable. That is the system

$$\begin{cases} E_1 \equiv (\dot{x}_1, x_1, x_2, \ldots, x_K, \varepsilon, t) = 0 \\ E_2 \equiv (\dot{x}_2, x_1, x_2, \ldots, x_K, \varepsilon, t) = 0 \\ \qquad\qquad \vdots \\ E_K \equiv (\dot{x}_k, x_1, x_2, \ldots, x_K, \varepsilon, t) = 0. \end{cases} \tag{3}$$

Taking an approximate solution of the system (3) as

$$x_{k,n+1} = x_{k,n} + \varepsilon x^c_{k,n} \tag{4}$$

where subscript n represents the n'th iteration over this approximate solution, we have a solution with one correction term in the perturbation expansion. The system can be approximated with a Taylor series expansion in the neighborhood of $\varepsilon = 0$

$$E_k = \sum_{i=0}^{m} \frac{1}{i!} \left[\left(\frac{d}{d\varepsilon} \right)^i E_k \right]_{\varepsilon=0} \varepsilon^i, \qquad k = 1, 2, ..., K \tag{5}$$

where

$$\frac{d}{d\varepsilon} = \frac{\partial \dot{x}_{k,n+1}}{\partial \varepsilon} \frac{\partial}{\partial \dot{x}_{k,n+1}} + \left(\sum_{j=0}^{K} \frac{\partial x_{j,n+1}}{\partial \varepsilon} \frac{\partial}{\partial x_{j,n+1}} \right) + \frac{\partial}{\partial \varepsilon} \tag{6}$$

is defined for the $(n+1)$'th iterative equation

$E_k(\dot{x}_{k,n+1}, x_{j,n+1}, \varepsilon, t)$.

Substituting (5) into (6), we obtain an iteration equation

$$E_k = \sum_{i=0}^{m} \frac{1}{i!} \left[\left(\dot{x}^c_{k,n} \frac{\partial}{\partial \dot{x}_{k,n+1}} + \left(\sum_{j=0}^{K} x^c_{j,n} \frac{\partial}{\partial x_{j,n+1}} \right) + \frac{\partial}{\partial \varepsilon} \right)^i E_k \right]_{\varepsilon=0} \varepsilon^i, \qquad k = 1, 2, ..., K \tag{7}$$

which is a first order differential equation and can be solved for the correction terms $x^c_{k,n}$. Then using (4), the (n+1)'th iteration solution can be found. Iterations are terminated after a successful approximation is obtained.

Note that for a more general algorithm, n correction terms instead of one can be taken in expansion (4) which would then be a $PIA(n,m)$ algorithm. The algorithm can also be generalized to a differential equation system having arbitrary order of derivatives (See [11] for more details).

After the discussion of $PIA(1,m)$, now we focus on $PIA(1,1)$ which is its simple case of perturbation-iteration method $PIA(n,m)$.

We consider the general cause of first order of differential equation as

$$E(\dot{x}, x, \varepsilon) = 0 \tag{8}$$

where $x = x(t)$. Taking one correction term in perturbation expansion, we have

$$x_{n+1} = x_n + \varepsilon x^c \tag{9}$$

where n denotes the n'th iteration over this approximate solution such that for the perturbation parameter ε the expression εx^c represents the correction term. Substitution of (9) into (8) we obtain

$$E(\dot{x}_n, x_n, 0) + \frac{\partial E(\dot{x}, x, 0)}{\partial x} \varepsilon x^c + \frac{\partial E(\dot{x}_n, x_n, 0)}{\partial \dot{x}} \varepsilon \dot{x}^c + \frac{\partial E(\dot{x}_n, x_n, 0)}{\partial \varepsilon} \varepsilon = 0. \tag{10}$$

Reorganizing the equation (10), we have

$$\dot{x}^c + \frac{E_x}{E_{\dot{x}}} x^c = -\frac{\varepsilon E_\varepsilon + E}{\varepsilon E_{\dot{x}}} \tag{11}$$

where $E_z = \frac{\partial E}{\partial z}$ and all derivatives are evaluated at $\varepsilon = 0.$ Setting

$$\mu(t) = \exp\left(\int \frac{E_x}{E_{\dot{x}}} dt\right)$$

as integrating factor, our equation (11) is now transformed into the form

$$\frac{d}{dt}\left(\mu(t)x^c\right) = \mu(t)\left(-\frac{\varepsilon E_\varepsilon + E}{\varepsilon E_{\dot{x}}}\right)$$

so that

$$\mu(t)x^c = -\int \mu(t)\left(\frac{\varepsilon E_\varepsilon + E}{\varepsilon E_{\dot{x}}}\right) + C$$

that is

$$\begin{aligned} x^c &= \frac{C}{\mu(t)} - \frac{1}{\mu(t)}\int \mu(t)\left(\frac{\varepsilon E_\varepsilon + E}{\varepsilon E_{\dot{x}}}\right) \\ &= C\exp\left(-\int \frac{E_x}{E_{\dot{x}}} dt\right) - \left[\int\left(\frac{\varepsilon E_\varepsilon + E}{\varepsilon E_{\dot{x}}}\right)\exp\left(\int \frac{E_x}{E_{\dot{x}}} dt\right)\right]\exp\left(-\int \frac{E_x}{E_{\dot{x}}} dt\right) \end{aligned} \tag{12}$$

Substitution of (12) into (9) and constructing the iteration scheme yields

$$\begin{aligned} x_{n+1} &= x_n + \varepsilon C_n C\exp\left(-\int \frac{E_x(\dot{x}_n, x_n, 0)}{E_{\dot{x}}(\dot{x}_n, x_n, 0)} dt\right) - \left[\int\left(\frac{\varepsilon E_\varepsilon(\dot{x}_n, x_n, 0) + E(\dot{x}_n, x_n, 0)}{\varepsilon E_{\dot{x}}(\dot{x}_n, x_n, 0)}\right)\right. \\ &\left.\exp\left(\int \frac{E_x(\dot{x}_n, x_n, 0)}{E_{\dot{x}}(\dot{x}_n, x_n, 0)} dt\right)\right]\exp\left(-\int \frac{E_x(\dot{x}_n, x_n, 0)}{E_{\dot{x}}(\dot{x}_n, x_n, 0)} dt\right). \end{aligned}$$

4 NUMERICAL SIMULATION

In numerical simulation we consider the parameters presented in the table 1.

Table1. *Values of parameters used in numerical simulation.*

Parameter	Value	Parameter	Value
Q	6	RQ	0.8
Kv	0.05	SV	0.7
$\dot{V}CO_2$	0.21	K	863
$[O_2]_a$	0.197	K_{CO_2}	0.0065
$[O_2]_v$	0.147	k_{CO_2}	0.244

The role of physical activity in hypoxemic hypoxia is to maintain the total concentration of carbon dioxide in the tissue ($[CO_2]_t$) and vascular ($[CO_2]_v$) in a narrow range. The numerical simulation is carried on three types of regular physical activity (physical exercise of 30 minutes per day) for a 30 years old woman: Walking, Jogging and Running Fast. To show the effectiveness of $PIA(1,1)$, we focus on the numerical simulation of the system (1)-(2) by taking the observed values presented in the table 1 [12]

Taking H_e and $\dot{V}_{A_e}$ hear rate and alveolar ventilation at the equilibrium states, the system (1)-(2) is written as

$$\dot{x} = f_e - K_v(x - y) \tag{13}$$

$$\dot{y} = \dot{Q}(g_e - y) + K_v(x - y) \tag{14}$$

where we set

$$x = [CO2]_t, \quad y = [CO2]_v, \quad \dot{x} = \frac{d[CO_2]_t}{dt} \text{ and } \dot{y} = \frac{d[CO_2]_v}{dt},$$

and where we take

$$f_e = RQ \times H_e \times SV \times ([O_2]_a - [O_2]_v) \quad \text{and } g_e = \left(K_{CO_2} \times \frac{\dot{V}CO_2}{\dot{V}_{A_e}} K + k_{CO_2} \right).$$

The system (13)-(14) is solved by using $PIA(1,1)$. The perturbation parameter ε is artificially introduced as

$$\begin{cases} E_1 \equiv \dot{x} + K_v x - f_e - \varepsilon K_v y = 0 \\ E_2 \equiv \dot{y} + (K_v + \dot{Q}) y - \dot{Q} g_e - \varepsilon K_v x = 0. \end{cases} \tag{15}$$

Setting $X = (x, y)^T$, we want an approximate solution of the system (15) in the form

$$X_{k,n+1} = X_{k,n} + \varepsilon X^c_{k,n}, \quad k = 1,2 \text{ and } n = 0,1,\ldots \tag{16}$$

with one correction term in the perturbation expansion such that the relation (7) is satisfied for

$$E_k\left(X_{k,n}, X^c_{k,n}, \varepsilon\right), \quad k = 1,2 \text{ and } n = 0,1,\ldots.$$

For (15), equation (7) reduces to

$$\begin{cases} E_1 \equiv \varepsilon \dot{X}^c_{1,n} + \varepsilon K_v X^c_{1,n} - K_v X^c_{2,n} \varepsilon = -\dot{X}_{1,n} - K_v X_{1,n} + f_e \\ E_2 \equiv \varepsilon \dot{X}^c_{2,n} + \varepsilon (K_v + \dot{Q}) X^c_{2,n} - \varepsilon K_v X^c_{1,n} = -\dot{X}_{2,n} - (K_v + \dot{Q}) X^c_{2,n} + \dot{Q} g_e. \end{cases} \tag{17}$$

that is

$$\begin{cases} \dot{X}^c_{1,n} + K_v X^c_{1,n} - K_v X^c_{2,n} = \dfrac{-\dot{X}_{1,n} - K_v X_{1,n} + f_e}{\varepsilon} \\ \dot{X}^c_{2,n} + (K_v + \dot{Q}) X^c_{2,n} - K_v X^c_{1,n} = \dfrac{-\dot{X}_{2,n} - (K_v + \dot{Q}) X^c_{2,n} + \dot{Q} g_e}{\varepsilon}. \end{cases} \tag{18}$$

which can be written in matrix form as follows

$$\dot{X}^c_n = A X^c_n + F_e, \tag{19}$$

where

$$A = \begin{pmatrix} -K_v & K_v \\ K_v & -(K_v + \dot{Q}) \end{pmatrix} \text{ and } F_e = \begin{pmatrix} \dfrac{-\dot{X}_{1,n} - K_v X_{1,n} + f_e}{\varepsilon} \\ \dfrac{-\dot{X}_{2,n} - (K_v + \dot{Q}) X^c_{2,n} + \dot{Q} g_e}{\varepsilon} \end{pmatrix}.$$

If $X^c_{n_h}$ denotes the fundamental solution for homogeneous system of (19), a particular solution $X^c_{n_p}$ for this system is

$$X^c_{n_p} = X^c_{n_h} \int \left(X^c_{n_h}\right)^{-1} F_e dt$$

and we have

$$X^c_n = X^c_{n_h} + X^c_{n_p}.$$

We consider the initial values of carbon dioxide in the tissue ($[CO_2]_t$) and carbon dioxide in the vascular ($[CO_2]_v$) as taken in [6]. Furthermore we have

$$X_{1,0} = 27\,g/mol \text{ and } X_{2,0} = 22\,g/mol. \tag{20}$$

For a 30 years old woman during physical activity, the table 2 shows the values of heart rate and alveolar ventilation at the equilibrium states as in [6].

Table 2. *The mean value of the heart rate and the alveolar ventilation for the rest and three cases of physical activities. A part the rest, other values represent equilibrium values related to 30 years old woman three physical activity.*

Exercise intensity	Rest	Walking	Jogging	Running Fast
Ventilation (L/min)	6	8.5	15	25
Heart rate (Beats /min)	70	85	140	180

Taking $n = 0$ and using the values of parameters given in the table 1, the fundamental matrix for the homogeneous system is

$$X^{c}_{0_h} = \begin{pmatrix} -0.00833\,e^{-6.0504t} & -0.99997\,e^{-0.0496t} \\ 0.99997\,e^{-6.0504t} & -0.00833\,e^{-0.0496t} \end{pmatrix}$$

and

$$\left(X^{c}_{0_h}\right)^{-1} = \begin{pmatrix} -0.00833\,e^{6.0504t} & 0.99997\,e^{6.0504t} \\ -0.99997\,e^{0.0496t} & -0.00833\,e^{0.0496t} \end{pmatrix}$$

Now we are interested in determining the particular solution and the solution of the system (13)-(14) for each physical activity.

1) Walking Case

After calculation we obtain

$$F_e = \begin{pmatrix} 2.3800 \\ 0.3826 \end{pmatrix}$$

so that

$$X^{c}_{0_p} = \begin{pmatrix} 0.09992 \\ 2.19578 \end{pmatrix}.$$

Finally using the relation (7), we have

$$\begin{pmatrix} X_{1,1} \\ X_{2,1} \end{pmatrix} = \begin{pmatrix} 27 \\ 22 \end{pmatrix} - 2.1954 \begin{pmatrix} -0.00833e^{-6.0504t} \\ 0.99997e^{-6.0504t} \end{pmatrix} + 0.11821 \begin{pmatrix} -0.99997e^{-0.0496t} \\ -0.00833e^{-0.0496t} \end{pmatrix} + \begin{pmatrix} 0.09992 \\ 2.19578 \end{pmatrix}.$$

2) Jogging Case

We have

$$F_e = \begin{pmatrix} 3.92000 \\ \\ 0.32253 \end{pmatrix}$$

and

$$X_{0_p}^{c} = \begin{pmatrix} 0.179946 \\ \\ 1.755196 \end{pmatrix}.$$

Hence using the relation (7), we get

$$\begin{pmatrix} X_{1,1} \\ \\ X_{2,1} \end{pmatrix} = \begin{pmatrix} 27 \\ \\ 22 \end{pmatrix} - 1.7541 \begin{pmatrix} -0.00833e^{-6.0504t} \\ \\ 0.99997e^{-6.0504t} \end{pmatrix} + 0.19456 \begin{pmatrix} -0.99997e^{-0.0496t} \\ \\ -0.00833e^{-0.0496t} \end{pmatrix} + \begin{pmatrix} 0.179946 \\ \\ 1.755196 \end{pmatrix}.$$

3) Running Fast Case

We obtain

$$F_e = \begin{pmatrix} 5.04000 \\ \\ 0.29112 \end{pmatrix}$$

and

$$X_{0_p}^{c} = \begin{pmatrix} 0.012554 \\ \\ 1.507488 \end{pmatrix}.$$

Thus the relation (7) gives

$$\begin{pmatrix} X_{1,1} \\ \\ X_{2,1} \end{pmatrix} = \begin{pmatrix} 27 \\ \\ 22 \end{pmatrix} - 1.50773 \begin{pmatrix} -0.00833e^{-6.0504t} \\ \\ 0.99997e^{-6.0504t} \end{pmatrix} + 0.02511 \begin{pmatrix} -0.99997e^{-0.0496t} \\ \\ -0.00833e^{-0.0496t} \end{pmatrix} + \begin{pmatrix} 0.012554 \\ \\ 1.507488 \end{pmatrix}.$$

Two others numerical methods have been used to test the efficiency of Perturbation-iteration algorithm $PIA(1,1)$. The preferred numerical methods are Matlab approach. They are often used to solve system of ordinary differential equations (ODEs). The Matlab approach has been implemented using its ODEs solver from Runge-Kutta of order 4 and 5, this is ode45. The numerical results are shown in the figures 2, 3 and 4.

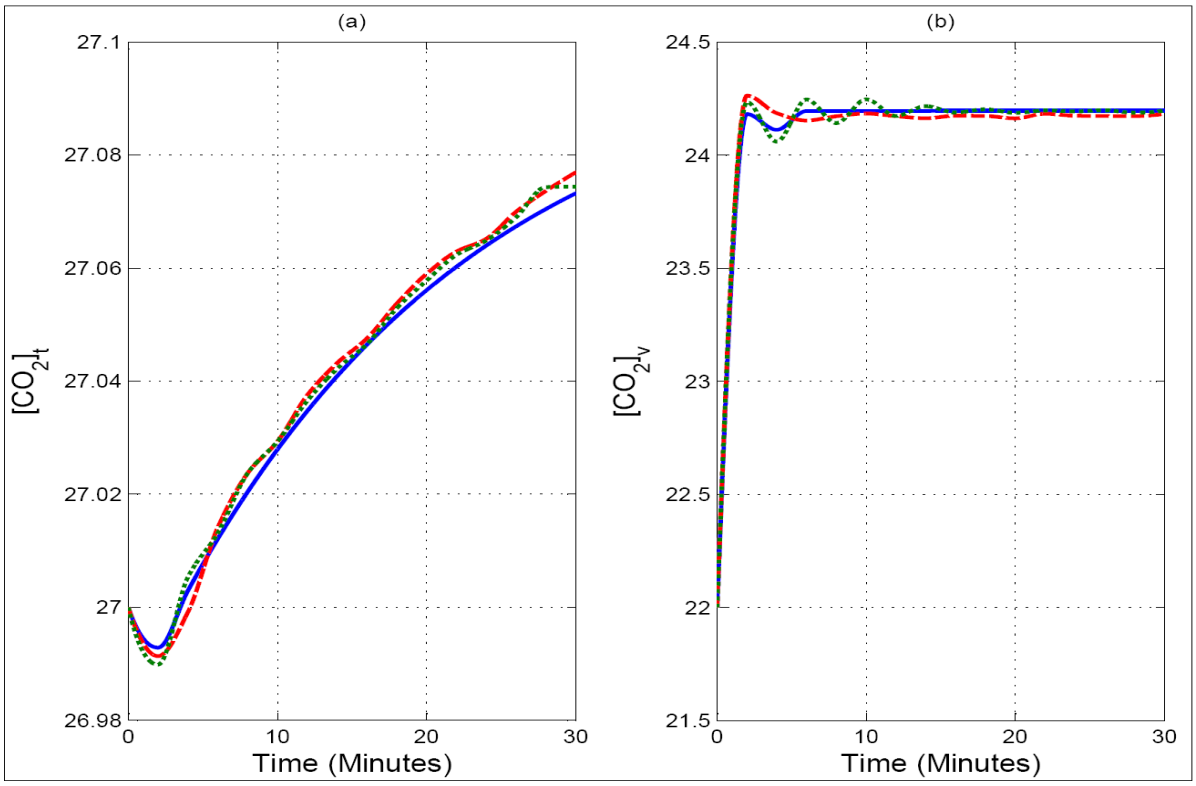

Figure 22. *Variation trajectory of concentration of tissue carbon dioxide $[CO_2]_t$ (a) and vascular carbon dioxide $[CO_2]_v$ (b) for a 30 years old woman during walking physical activity. Three curves are compared using three different methods: Perturbation-iteration algorithm (Solid line), Euler method (dashed line) and Matlab approach (Dot line)*

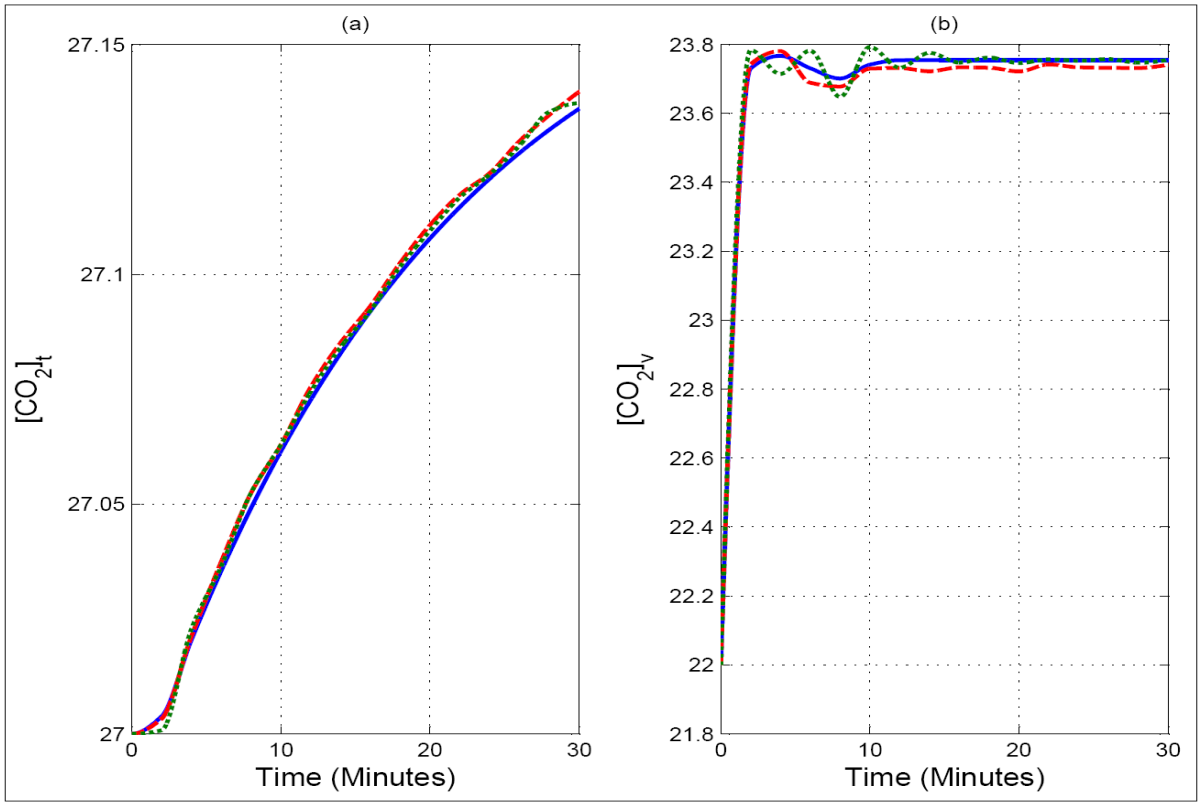

Figure 3. *Variation trajectory of concentration of tissue carbon dioxide $[CO_2]t$ (a) and vascular carbon dioxide $[CO_2]v$ (b) for a 30 years old woman during jogging physical activity. Three curves are compared using three different methods: Perturbation-iteration algorithm (Solid line), Euler method (dashed line) and Matlab approach (Dot line)*

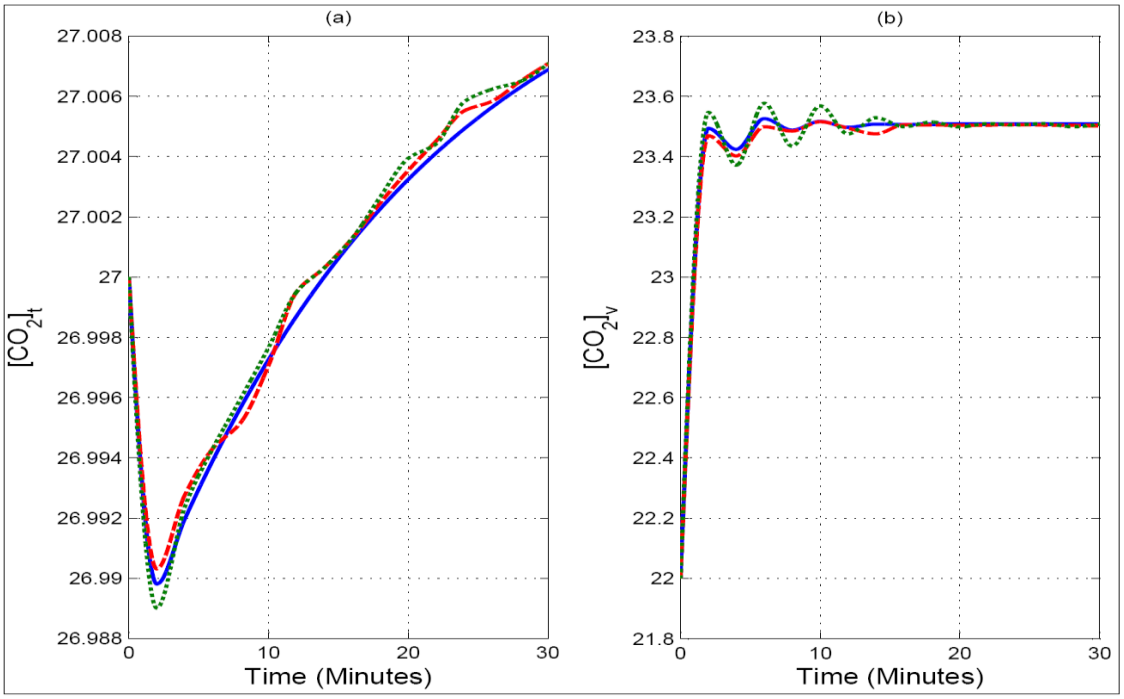

Figure 3. *Variation trajectory of concentration of tissue carbon dioxide [CO₂]t (a) and vascular carbon dioxide [CO₂]v (b) for a 30 years old woman during running fast physical activity. Three curves are compared using three different methods: Perturbation-iteration algorithm (Solid line), Euler method (dashed line) and Matlab approach (Dot line)*

5 DISCUSSION

During three types of physical activities, the response of controls of cardiovascular-respiratory system are illustrated in figure , and where we have a decrease of concentration of tissue carbon dioxide $[CO_2]_t$ at the beginning of physical activity for walking and running fast cases. The decrease is followed by an exponential increase of this parameter (See figures 2(a) and 4(a)). This does not happen in jogging case (See 3(a)). This mechanism results in the effect of heart rate and alveolar ventilation during physical activity where motor center activity and afferent impulses from proprioceptors of the limbs, joints and muscles play a crucial role. Since peripheral chemoreceptors are responsible for increasing ventilation, central chemoreceptors may be readjusted to increase ventilation so that tissue carbon dioxide concentration increases.

The figures 2(b), 3(b) and 4(b) show that vascular carbon dioxide concentration increases at the onset of all concerned physical activities to be stabilized at a value. At the onset of physical activity, the heart rate and alveolar ventilation increase. Generally, heart rate increases to about 90% of its maximum values during strenuous physical activity. Furthermore, the ventilation increases with increases in work rate at sub-maximal physical activity intensities. These physiological effects of physical activity on cardiovascular-respiratory system are justified by the variation of its controls. According to intensity of physical activity, these controls reach a equilibrium value and they are stabilized.

The results obtained in this work are rather satisfactory. In particular, the reaction of the controls of cardiovascular-respiratory system to physical activity can be modeled and a feedback can be approximated by the solution of its mathematical model governed by ordinary differential system for some chronic diseases of this biological system. Physical activity reduces any chronic disease of cardiovascular-respiratory system and it induces important improvement in health of patients.

6 CONCLUDING REMARKS

In this paper, we have investigated Perturbation-iteration method which is a new numerical method for solving a system of ordinary differential equations. To test its efficiency, we have used two other convergent methods: Euler method and Runge-Kutta method. Numerical simulations implemented using Matlab packages illustrate the responses of tissue and vascular carbon dioxide concentrations due to the controls of cardiovascular-respiratory system that is heart rate and alveolar ventilation. The

numerical results confirmed the analytical analysis for a 30 years old woman during three different physical activities: Walking, Jogging and Running fast.

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