STABILITY AND BIFURCATION ANALYSIS OF A HINDMARSH-ROSE

NEURONAL   MODEL WITH TIME DELAY

A THESIS

PRESENTED TO THE DEPARTMENT OF THEORETICAL & APPLIED PHYSICS

AT THE AFRICAN UNIVERSITY OF SCIENCE AND TECHNOLOGY – AUST

ABUJA, NIGERIA.

IN (PARTIAL) FUFILLMENT OF THE REQUIREMENT FOR THE AWARD OF

MASTER DEGREE IN THEORETICAL & APPLIED PHYSICS

BY

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JULY, 2019.

CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the African University of Science & Technology a dissertation titled: "STABILITY AND BIFURCATION ANALYSIS OF A HINDMARSH-ROSE MODEL WITH TIME DELAY'' in partial fulfilment of the requirements for the award of Master of Science degree (Theoretical & Applied Physics) at the African University of Science and Technology.

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DECLARATION AND COPYRIGHT

I, Ahmad Salihu Jibril, declare that this dissertation is my own original work and it has not been presented and will not be presented in any other University for a similar or any other degree award.

DEDICATION

I dedicated this project work to my parents Mr and Mrs Ahmad.

ACKNOWLEDGEMENT

ABSTRACT

In this research work, we study and analyse Hindmarsh-Rose neuronal system with time delay. Considering the fast sub-system of the model, all the possible non-negative equilibria are obtained and their local as well as global behaviour are studied. Choosing delay as a bifurcation parameter, the existence of the Hopf bifurcation of the system has been investigated. Moreover, we use the Descartes' sign rule, a powerful tool for real polynomials with constant coefficients to determine the number of real zeroes of the polynomial function. Classifications of the imaginary roots of the characteristic equation were presented. Some numerical simulations are given to support the analytical results. Some interesting conclusions are obtained from the results obtained at the end of this work.

Keywords: Hindmarsh-Rose model, neuron, time delay, bifurcation, Descartes's sign rule.

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1. INTRODUCTION

1.1 OVERVIEW OF BIOLOGICAL NEURON

1.1.1 NEURON

Neuron is an electrically excitable cell that receives, processes, and transmits information through electrical and chemical signals[1]. All living animals obtain information from their environment through sensory receptors, and this information is transformed to their brain, where it is processed into perceptions and commands. All these tasks are performed by a system of nerve cells, or neurons. Neurons have four morphologically defined regions: the cell body, dendrites, axon, and presynaptic terminals. A bipolar neuron receives signals from the dendritic system; these signals are integrated at a specific location in the cell body and then sent out by means of the axon to the presynaptic terminals. There are neurons which have more than one set of dendritic systems, or more than one axon, thus enabling them to perform simultaneously multiple tasks; they are called multipolar neurons[2]. The fundamental task of neurons is to receive, conduct, and transmit signals. Neurons carry signals from the sense organs inward to the central nervous system (CNS), which consists of the brain and spinal cord. In the CNS, the signals are analysed and interpreted by a system of neurons, which then produce a response. The response is sent, again by neurons, outward for action to muscle cells and glands. Neurons come in many shapes and sizes, but they all have some common features as shown schematically in Figure 1.1[3].
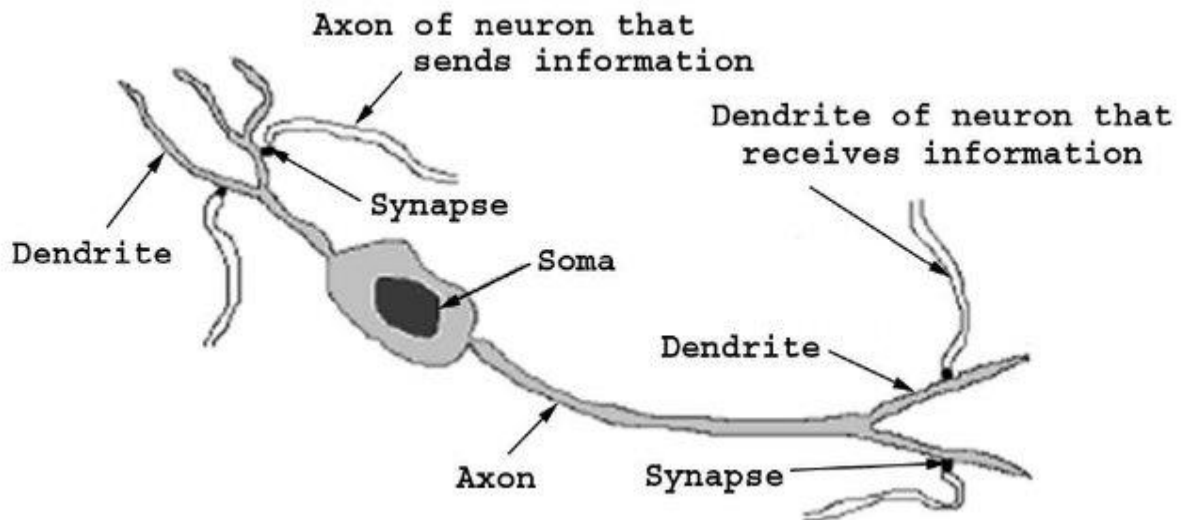
Fig1.1: Schematic diagram of biological neuron [3].

1.1.2 MEMBRANE

Neurons are surrounded by a membrane that distinguishes the cell's interior from the extracellular space. The quantity of ions on the inside of the membrane is distinct from that in the surrounding fluid. Concentration distinction produces an electrical potential that performs a significant role in neuronal dynamics and this is called the membrane potential.

A neuron is at rest when no signal is sent. On the outside of the neuron, there are relatively more sodium ions (Na+) and more potassium salts (K+) on the inside. Compared to the outside, the neuron inside is negative. The difference in voltage between the neuron's inside and outside gives the resting potential, generally about-70 mV of which is the value [1].

1.1.3 MEMBRANE POTENTIAL

An action (membrane) potential, also called spike or impulse occurs when a neuron sends information along an axon away from the cell body. The action potential is an explosion of electrical activity that is created by a depolarizing current. A stimulus makes the resting potential move up towards positive values. If the potential reaches a certain threshold (about -

55 mV), the neuron fires an action potential, the amplitude of which is always the same. If the potential does not reach this threshold value, no action potential fires (see Fig.1.3).
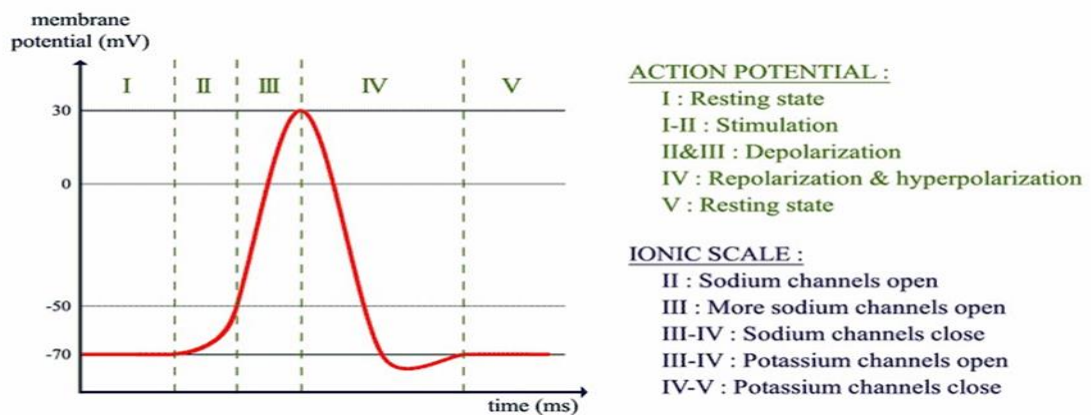

Fig.1.2: Action Potential[1]

1.1.4 IONIC CHANNEL

Ionic channels can be seen in the neuron membrane as micro cell pores. They allow the passage of mo lecules through the membrane.They are the link between intra-cellular and extra-cellular space. There are many types of ionic channels. For example, we may cite those which are always open, those which are voltage-dependent or those which select the molecule allowed to cross the membrane. Sodium channels are so called because they are specific to sodium ions. They can be in an active state or in an inactive one. Potassium channels open and close with delay. Leak channel are always open.

Action potentials are caused by an exchange of ions across the neuron membrane when a stimulus is applied, sodium channels open. Since there is much more sodium ions on the outside of the neuron and since the inside of the neuron is negative compared to the outside, sodium ions rush into the neuron. As sodium has a positive charge, the neuron becomes more positive, that is depolarized. Besides, potassium channels open with delay. When they do open, potassium goes out of the cell, reversing the depolarization. At that time, sodium channels start to close. This induces the repolarization. Indeed, it makes the membrane potential go back

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towards the resting potential. Then, there is the hyperpolarization of the neuron, since the potential goes past the resting potential because the potassium channels close with delay. Gradually, the ion concentrations go back to resting levels and the cell returns to -70 mV (see Fig. 1.2). For more details, see [4].

1.1.5 BURSTING AND SPIKING

A burst is a group of at least two action potentials that occur close together in time, separated from other action potentials by large time intervals which are called quiescent or silent phases. Spiking occurs when the membrane potential of a specific axon location rapidly rises and fall. Action potential in neurons are also known as 'spike' and the temporal sequence of action potentials generated by a neuron is called its 'spike train'. (See Fig1.3) show the spiking and bursting behaviour of a neuron in a HR model.
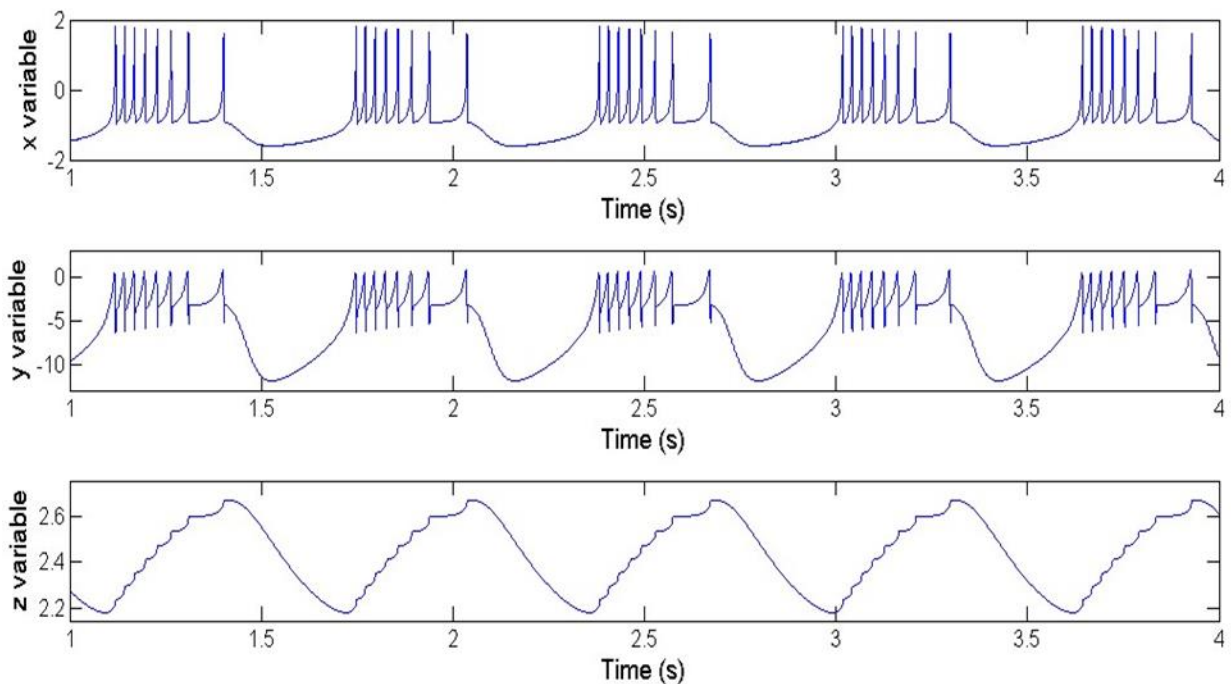

Fig 1.3: Hindmarsh-Rose neuron output for applied current, I = 2.5[1].

1.2 HISTORICAL BACKGROUND OF NEURONAL MODEL

1.2.1 Hodgkin-Huxley (HH) Model

Hodgkin and Huxley in (1952) were the first neurophysiologist to develop an empirical kinetic description of ionic mechanism in a neuron. This model is based on sodium, potassium and leakage ion flow[5].

The model reads as follows

$$
\begin{cases}
-C \dfrac{dV}{dt} = m^3 h g`_{Na}\big(V - E(N_a)\big) + n^4 g`_K\big(V - E(K)\big) + g`_L\big(V - E(L)\big) - I, \\[2mm]
\qquad\qquad \dfrac{dn}{dt} = \alpha_n(1 - n) - \beta_n n, \\[2mm]
\qquad\qquad \dfrac{dm}{dt} = \alpha_m(1 - m) - \beta_m m, \\[2mm]
\qquad\qquad \dfrac{dh}{dt} = \alpha_h(1 - h) - \beta_h h.
\end{cases}
\tag{1}
$$

In Eq. (1),

C is the membrane capacity, V is the total membrane potential, m is the N_a activation variable,

h is the N_a inactivation variable, $E(N_a)$ is the Na equilibrium potential.

$g`_{Na}$ is the maximum sodium conductance, n is the K activation variable.

$E(K)$ is the K equilibrium potential, $g`_K$ is the maximum potassium conductance.

$E(L)$ is the Leakage equilibrium potential, $g`_L$ is the maximum leakage conductance.

I is the applied current (applied during experiment), α_i is the gate inactivation rate.

β_i is the gate activation rate.

This model is not convenient for dynamical analysis of a neuron due it complexity[6].

1.2.2 Hindmarsh-Rose (HR) Model

The HR model of neural activity is aimed to study the spiking-bursting behaviour of the membrane potential observed in experiments made with a single neuron. The electric activity of neurons is of great concern. A typical example is given by the Rose–Hindmarsh model of action potential which has continuously attracted considerable attention in the past decades[6]. In 1982, Hindmarsh and Rose simplified the rigorous HH system into a system of two differential equations as follows:

$$\begin{cases} \dfrac{dx}{dt} = y + ax^2 - x^3, \\ \dfrac{dy}{dt} = 1 - dx^2 - y. \end{cases} \tag{2}$$

Two years later, Hindmarsh-Rose decided to add a third equation to their model, so that the dynamics of their model could be similar to the dynamics of a real biological neuron[1]. This model described the dynamical behaviour of membrane potential x in an axon of a neuron and has drawn much attention and was studied intensively in literature. Particular attention was devoted to the study of the transitions between different nonlinear dynamical behaviours[7].

The HR model describes one of the most typical configurations of slow manifolds needed for square wave bursting to occur naturally in various neuron models of the Hodgkin–Huxley type[8].

The model is given by three nonlinear differential equations as follows[6].

$$\begin{cases} \dfrac{dx}{dt} = y - ax^3 + bx^2 - cz + I, \\ \dfrac{dy}{dt} = c - dx^2 - y, \\ \dfrac{dz}{dt} = \varepsilon(S(x - \chi) - z). \end{cases} \tag{3}$$

where

x is the membrane potential, y is the recovery /spiking variable.

z is the bursting variable (adaption current), I is the applied current, ε is the parameter of control. All other parameter a, b, c, d, S and χ are real constant.

Hence, the HR model can be called a slow-fast system that has the (x, y) fast sub-system and the single slow dynamical equation (z) with a small control parameter$(0 < \varepsilon < 1)$.

1.2.3 HR MODEL WITH TIME DELAY

Mathematical models with time delays have been broadly applied in many scientific fields such as neurosciences, biology and mechanics. The effects of delay are popular in dynamical systems due to the finite propagation speed of signals or the finite time of processing. In the study of delayed systems, many delay factors appear in state variables and some of them appear in parameters. In recent years, delay feedback control is widely applied in mechanical and electronic facilities. Recently, it was found that delay factors are inherent in many biological systems due to finite propagation speed of signals and finite processing time in synapses[6,8]. The H-R model with time delay takes the form:

$$
\begin{cases}
\dfrac{dx}{dt} = y - ax^3 + bx(t - \tau)^2 - cz + I, \\
\qquad \dfrac{dy}{dt} = c - dx^2 - y, \\
\quad \dfrac{dz}{dt} = \varepsilon[S(x - \chi) - z].
\end{cases}
\qquad (4)
$$

Where г is the time delay. When τ is zero and $I \in$ [3.28, 3.405] Hindmarsh and Rose observed system (4) to be chaotic and for > 0 , it delineate so many dynamical behaviour.

2. LITERATURE REVIEW

This chapter introduces the theoretical framework of the Hindmarsh-Rose model and the effect of time delay in the model as a bifurcation parameter. A neuron model being a nonlinear system is expected to demonstrate at least three fundamental cell types of activity such as quiescence, tonic spiking and bursting. The nonlinearity of a neuronal model may often lead to a bi- or multi-stability of co-existing cell's activities, which are selected by initial conditions at the same parameter values[8].

2.1 BIFURCATION AND STABILITY

Studies reveal that the dynamical behaviour of system changes quite dramatically with the variation of a system parameters. It is important to know that these changes are not only qualitative, such as change in the location of a fixed point, but also qualitative, such as change in the systems stability. System can change behaviour from regular (steady/periodic) to irregular (chaotic). It is this qualitative changes in the system dynamics that is the subject of interest in the theory of every dynamical system[7].

Therefore, qualitative changes in a system dynamics are called bifurcations and the parameter values at which the bifurcation occurs are called bifurcation points.

HR Model has been studied in[7] by considering the slow-fast system showing the evolution of the solution trajectory between the stable slow manifold and the unstable slow manifold by applying the single perturbation.

As it is expressed above, in dynamical systems, a bifurcation happens when a little smooth change made to the parameter esteems (the bifurcation parameters) of a system causes an unexpected "qualitative" or topological change in its behaviour. Generally, at a bifurcation point, the neighbourhood stability properties of equilibria, periodic circles or other invariant sets change[10]. It has two types:

Local bifurcations, which can be broken down altogether through changes in the local stability properties of equilibria, periodic circles or other invariant sets as parameters cross through critical thresholds.

Global bifurcations, which frequently happen when larger invariant a set of the system "crash" with one another, or with equilibria of the system. They can't be distinguished absolutely by a stability analysis of the equilibria (fixed or equilibrium points)[10].

Time delays are inevitable in neural systems due to finite propagation speed of signals and time processing in synapses[7]. Studies portrait that time delay can significantly impact the behaviours of dynamical systems[11]. Signals transmission time delays in a network of non-linear oscillators are known to be responsible for a variety of interesting dynamic behaviours including phase-flip transitions leading to synchrony or out of synchrony[12]. The dynamical transitions resulting from time delay in single HR system are investigated using the method of stability switch and geometric singular perturbation theory, which reveals that as the time delay varies, the structure of the slow manifold changes[7]. In both systems of coupled HR neurons, time-delay induced phase-flip bifurcations to synchrony was observed[11].

2.1.1 Hopf bifurcation

Hopf bifurcation occurs in systems of differential equations consisting of two or more equations. As it is well known, Hopf bifurcations occur when a conjugated complex pair of eigenvalues crosses the boundary of stability. In the time-continuous case, a limit cycle bifurcates. It has an angular frequency which is given by the imaginary part of the crossing pair. In the discrete case, the bifurcating orbit is generally quasi-periodic[10]. The Hopf bifurcation theorem is one of the most important results for delay differential equations because it is essentially the only method for rigorously establishing the existence of periodic solution.

The double Hopf bifurcations are induced if two pairs of imaginary roots appear simultaneously on the margins of the "death island" regions [9].

Analytic method is used to derive the condition under which Hopf bifurcation occurs, for instance by applying the formula of the Sturm sequence to consider the stability, when imaginary roots iω of the corresponding polynomial equation has high multiplicity. Hopf bifurcation curves are plotted in the (I -τ) plane by the numerical software DDE-Biftool. Double Hopf bifurcation points are obtained at the intersection points of Hopf bifurcation curves by changing values of time delay τ, parameters S and I, and fixing values for other parameters[6].

a =1.0, b=3.0, c=1.0, d=5.0, χ = -1.6, ε=0.001.

3. METHODOLOGY

Equation (1) has been studied in[7]&[6], where the Hopf bifurcations and dynamical behaviour near the equilibrium point are focused. In this present research, stability and Hopf bifurcation of the fast subsystem of the HR model with time lag are to be investigated, and as the time delay varies, the mechanism of the transitions between bursting oscillation, relaxation oscillation, chaotic bursting, and other complex oscillations is to be illuminated, by using the methods of stability switch and geometric singular perturbation theory.

3.1 DELAY INDUCED-DYNAMICAL TRANSITION

We take τ as a bifurcation parameter in Eq. (3), and other parameters as:

$a= 1.0, b = 3.0, c = 1.0, d = 5.0, \varepsilon = 0.01, x = -1.6$, and $I = 3.25$

Therefore, Eq. (3) can be rewritten as

$$\begin{cases} \dfrac{dx}{dt} = y - x^3 + 3x(t-\tau)^2 - z + 3.25 = F(x,y,z), \\ \qquad \dfrac{dy}{dt} = 1 - 5x^2 - y = P(x,y), \\ \quad \dfrac{dz}{dt} = 0.01[4(x+1.6)-z] = Q(x,z). \end{cases} \qquad (5)$$

Without the time delay, Eq. (5) undergoes bursting oscillation[12].

According to the geometric singular perturbation theory, the key step in understanding the time-delay effects on the dynamics is to determine how the time delay impacts on the structure of the slow manifold including the shape and stability of the slow manifold and the bifurcation points in the slow manifold[7].

3.2 STABILITY AND HOPF BIIFURCATION ANALYSIS

We now consider the fast subsystem (x, y) of the model and by setting the third equation of the model to zero and take z as system parameter reducing the model to 2D, we have

$$\begin{cases} \dfrac{dx}{dt} = y - x^3 + 3x(t-\tau)^2 - z + 3.25, \\ \qquad \dfrac{dy}{dt} = 1 - 5x^2 - y. \end{cases} \qquad (6)$$

3.2.1 Analyses of the model in the absence of the delay

In this subsection, we find the possible equilibrium points to the system Eq. (6) without delay, and then, we analyse stability criterion of those points.

Now, we rewrite Eq. (5) as

$$\begin{cases} \dfrac{dx}{dt} = y - x^3 + 3x^2 - z + 3.25, \\ \qquad \dfrac{dy}{dt} = 1 - 5x^2 - y. \end{cases} \qquad (7)$$

At equilibrium (x_0, y_0), we have

$$\begin{cases} 0 = y_0 - x_0{}^3 + 3x_0{}^2 - z_0 + 3.25 \\ \qquad 0 = 1 - 5x_0{}^2 - y_0 \\ \Longrightarrow z = -x_0{}^3 - 2x_0{}^2 + 4.25. \end{cases} \qquad (8)$$

From Eq. (8), we have

$$z = -x_0{}^3 - 2x_0{}^2 + 4.25. \qquad (9)$$

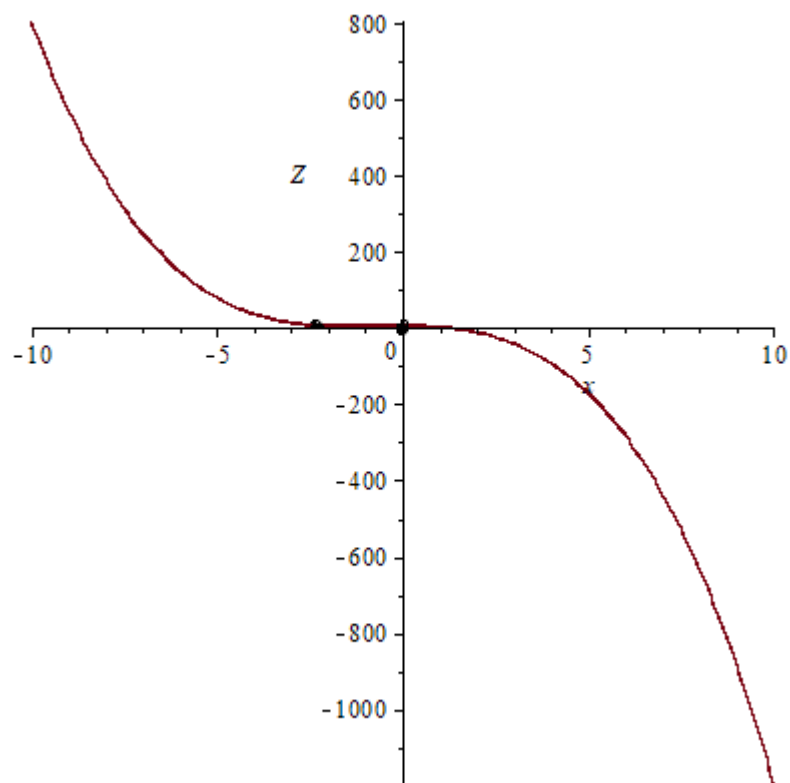Differentiating with respect to time, leads

$$-3x_0{}^2 - 4x_0 = 0.$$

Equivalently, we have $x_0(3x_0 + 4) = 0$.

Finally, we have $x_0 = 0 \ or \ x_0 = -\frac{4}{3}$.

Which has two stationary points $(0 \ and -\frac{4}{3})$, the slow manifold M is an S-shape curve

plotted with Maple as shown below:

To investigate the stability and bifurcation of the slow manifold M, consider local stability of

Eq. (6) about the equilibrium point $(x_0, y_0) \in M$. We have two fixed points

$$P_1 = (0,1) \ \& \ P_2 = \left(-\frac{4}{3}, -\frac{71}{9}\right).$$

We linearize Eq. (6) about the possible equilibrium points $(x_0, y_0) \in M$.

1. **Linearization about point P_1 ($\boldsymbol{trivial\ equilibrium\ point}$):**

   We now obtain the Jacobian J as

   $$J = \begin{vmatrix} F_x & F_y \\ P_x & P_y \end{vmatrix} = \begin{vmatrix} -3x_0{}^2 + 6x_0 & 1 \\ -10x_0 & -1 \end{vmatrix},$$

   where $F_x = \dfrac{\partial F}{\partial x}, F_y = \dfrac{\partial F}{\partial y}, P_x = \dfrac{\partial P}{\partial x}, P_y = \dfrac{\partial P}{\partial y}$.

Therefore, to find the characteristic polynomial, we now equate the Jacobian to zero and compute the determinant at point P_1, we have:

$$\begin{vmatrix} -\lambda & 1 \\ 0 & -1 - \lambda \end{vmatrix} = 0.$$

That is

$$\lambda(1 + \lambda) = 0 \iff \lambda = 0 \ or \ \lambda = -1.$$

Which implies instability of the system. Hence the system is not stable at $\boldsymbol{P_1}$.

2. **Linearization about point P_2:**

   In a similar vein, we have

   $$\begin{vmatrix} -\dfrac{40}{3} - \lambda & 1 \\ 40/3 & -1 - \lambda \end{vmatrix} = 0.$$

That is,

$$(1 + \lambda)\left(\frac{80}{3} + \lambda\right) = 0 \iff \lambda = -1 \ or \ \lambda = -\frac{80}{3}.$$

We see that,

$$\lambda < 0, hence, the\ fast\ sub\ system\ is\ stable\ at\ this\ fixed\ points.$$

Hence, the system is locally stable at $\boldsymbol{P_2}$.

3.3 Analysis of the model in the presence of delay

By linearization of Eq. (6), we have

$$\begin{vmatrix} -\lambda - 3x_0{}^2 + 6x_0 e^{-\lambda\tau} & 1 \\ -10x_0 & -\lambda - 1 \end{vmatrix} = 0.$$

Which leads to

$$\lambda^2 + \kappa\lambda - \beta(1-\lambda)e^{-\lambda\tau} + \gamma = 0, \tag{10}$$

where $\quad \kappa = 3x_0{}^2 + 1, \ \beta = 6x_0, \ \gamma = 3x_0{}^2 + 10x_0.$

Let $\lambda(\tau) = \mu + i\omega$, be an eigenvalue of the system at point P_2. But the change of stability of this point will occur when $R_e(\lambda) = 0$. Thus, to find out the position of change of stability, we substitute for $\lambda(\tau) = i\omega \ i.e(\omega > 0)$ in Eq. (7):

$$-\omega^2 + i\omega\kappa - \beta(1-i\omega)e^{-i\omega\tau} + \gamma = 0,$$

Which can be written as

$$-\omega^2 + i\omega\kappa - \beta(1-i\omega)[\cos(\omega\tau) - isin(\omega\tau)] + \gamma = 0 \tag{11}$$

We now separate the real and imaginary part of Eq. (8) above computed using a Maple software as:

Real part

$$-\omega^2 - \beta\cos(\omega\tau) - \beta\sin(\omega\tau)\,\omega + \gamma = 0.$$

$$\cos(\omega\tau) = \frac{-\omega^2 + \beta\sin(\omega\tau)\,\omega + \gamma}{\beta}$$

Imaginary part

$$\kappa\omega + \beta\sin(\omega\tau) + \beta\omega\cos(\omega\tau) = 0.$$

$$\Leftrightarrow \begin{cases} \sin(\omega\tau) = \dfrac{-\omega(\omega^2 + \kappa - \gamma)}{\beta(1 + \omega^2)}, \\ \cos(\omega\tau) = \dfrac{\kappa\omega^2 - \omega^2 + \gamma}{\beta(1 + \omega^2)}. \end{cases} \tag{12}$$

$$-\omega^2 + \gamma = \beta\cos(\omega\tau) - \beta\sin(\omega\tau)\,\omega. \tag{13}$$

$$\kappa\omega = -\beta\omega\cos(\omega\tau) - \beta\sin(\omega\tau). \tag{14}$$

By squaring Eq. (10 and 11) and adding the result, we have:

$$\omega^4 + (\kappa^2 - \beta^2 - 2\gamma)\omega^2 + \gamma^2 - \beta^2 = 0 \tag{15}$$

Which is a quadratic equation in ω^2. Therefore, for simplicity, let $\phi = \omega^2$.

Therefore, Eq.(15) becomes:

$$F(\phi) = \phi^2 + (\kappa^2 - \beta^2 - 2\gamma)\phi + (\gamma^2 - \beta^2) = 0. \tag{16}$$

We now apply the Descartes' sign rule in order to know the number of possible positive, negative and imaginary zeros of Eq. (16):

$With\ \left(\kappa^2 - \beta^2 - 2\gamma\right) \geq 0,\ (\gamma^2 - \beta^2) \geq 0:$

Positive	Negative	Imaginary
0	2	0
0	0	2

$With \left(\kappa^2 - \beta^2 - 2\gamma\right) < 0, \ (\gamma^2 - \beta^2) \geq 0 :$

Positive	Negative	Imaginary
2	0	0
0	0	2

$With \left(\kappa^2 - \beta^2 - 2\gamma\right) \geq 0, \ (\gamma^2 - \beta^2) < 0 :$

Positive	Negative	Imaginary
1	1	0
1	1	0

Lemma 3.1: [13] The equilibrium P_2 is locally asymptotically stable for any $\tau \geq 0$ if and only if P_2 is locally asymptotically stable at $\tau = 0$.

We have already seen that in Eq. (7), P_2 is always asymptotically stable in the absence of the delay i.e. $(\tau = 0)$. So we have the following theorem:

Theorem 3.1: Suppose that $F(\phi)$ has no positive zero, then P_2 is locally asymptotically stable for all $\tau \geq 0$.

Proof: P_2 is locally asymptotically stable at $\tau = 0$ as we have seen earlier. Also the condition $F(\phi)$ has no positive zero shows that no root of $\ \triangle (i\omega, \tau) = 0$ is purely imaginary. Thus, there is no pure imaginary value of λ of the characteristic Eq. (7). Hence, by the above discussion the system is locally asymptotically stable at P_2 for ≥ 0 . Hence the theorem.

Note: We only have one case for which $F(\phi)$ *have non positive zero that is when*

$(\kappa^2 - \beta^2 - 2\gamma) \geq 0 \ and \ (\gamma^2 - \beta^2) \geq 0$. Shown in the above Table 3.1.

Theorem 3.2: Suppose that $F(\phi)$ has a unique positive zero, then, if the system is unstable around P_2 for $\tau = \tau'$, $i.e \ \tau' \geq 0$, then, it remain unstable for any $\tau > \tau'$.

Proof: If $F(\phi)$ has at least one positive zero, then, at that point $F(\phi)$ is an increasing function. Thus, it has a positive eigenvalue at P_2, and this value is increasing as τ increases. Hence, if the system is unstable around P_2, for $\tau = \tau'$ $i.e \ \tau' \geq 0$, then, it remains unstable for $\tau > \tau'$.

Note, here, we have two cases for which $F(\phi)$ *have at least one positive zero:*

(i) $(\kappa^2 - \beta^2 - 2\gamma) \geq 0 \ and \ (\gamma^2 - \beta^2) < 0.$

(ii) $(\kappa^2 - \beta^2 - 2\gamma) < 0 \ and \ (\gamma^2 - \beta^2) \geq 0.$

Now, from Eq. (15) and (16) we have:

$$\phi = \frac{-(\kappa^2 - \beta^2 - 2\gamma) \pm \sqrt{(\kappa^2 - \beta^2 - 2\gamma)^2 - 4(\gamma^2 - \beta^2)}}{2},$$

$$\begin{cases} \omega_1 = \sqrt{\dfrac{-(\kappa^2 - \beta^2 - 2\gamma) + \sqrt{(\kappa^2 - \beta^2 - 2\gamma)^2 - 4(\gamma^2 - \beta^2)}}{2}}, \\[4mm] \omega_2 = \sqrt{\dfrac{-(\kappa^2 - \beta^2 - 2\gamma) - \sqrt{(\kappa^2 - \beta^2 - 2\gamma)^2 - 4(\gamma^2 - \beta^2)}}{2}}. \end{cases} \tag{17}$$

Theorem 3.3 Suppose that ω^{*2} is the only positive zero of $F(\phi)$, then, P_2 undergoes a Hopf –bifurcation as τ passes through τ_0, where τ_0 is the least positive value of τ_k (with k being an integer) and this can be obtained from Eq.(9) above.

Proof: System Eq. (6) is locally asymptotically stable at P_2, for $\tau = 0$ (absence of delay). But if $\phi = \omega^{*2}$ is a simple positive zero of $F(\phi)$, then, it has an eigenvalue with non-

negative real part at P_2, and so, it cannot be stable for all positive τ. Thus, a change of stability will occur in this case. Now, for $\omega^2 = \omega^{*2}\ and\ \omega = \omega^*$, the system has an eigenvalue of the form $\lambda = i\omega^*$, hence, the condition for the Hopf-bifurcations holds.

Thus, we can conclude that at $\tau = \tau_0$, the change of stability which is a Hopf-bifurcation could occur where τ_0 is the least value of τ_k (k being an integer) and this τ_k can be obtained by solving for τ in Eq. (12):

$$\tan(\omega\tau) = \frac{-\omega(\omega^2 + \kappa - \gamma)}{\kappa\omega^2 - \omega^2 + \gamma},$$

Which can be written as

$$tan(\omega\tau) = \frac{-\omega^3 + (\gamma - \kappa)\omega}{\omega^2(\kappa - 1) + \gamma},$$

Leading to

$$\tau = \frac{1}{\omega}\arctan\left(\frac{-\omega^3+(\gamma-\kappa)\omega}{\omega^2(\kappa-1)+\gamma}\right). \tag{18}$$

In general, we have $\omega = \omega^*$. Which gives

$$\tau_k = \frac{2\pi k}{\omega_i^*} + \frac{1}{\omega_i^*}\arctan\left(\frac{-\omega_i^{*3} + (\gamma - \kappa)\omega_i^*}{\omega_i^{*2}(\kappa - 1) + \gamma}\right) \tag{19}$$

$$k = 0, \pm1, \pm2, \dots\ and\ i = 1,2 \ .$$

Using the above equations, time series, phase portrait and bifurcation diagrams would be plotted in the subsequent Chapter.

4. RESULTS AND DISCUSSION

In this chapter, we try to interpret, compare and discuss the analytical and numerical results obtained.

Case1: The RH fast sub-system $with\ \tau = 0\ evaluated\ around\ P_1$

(a)
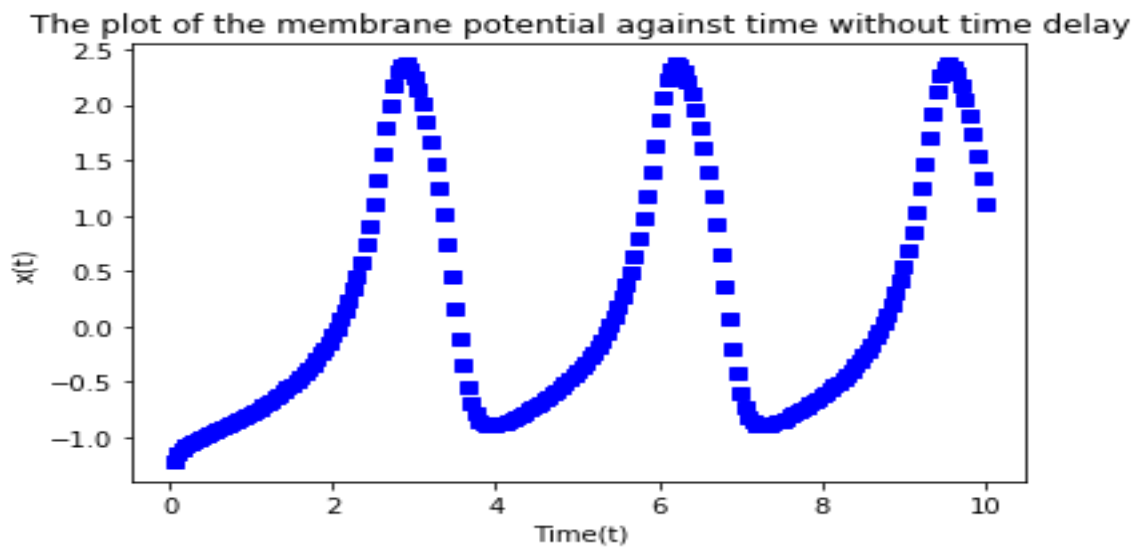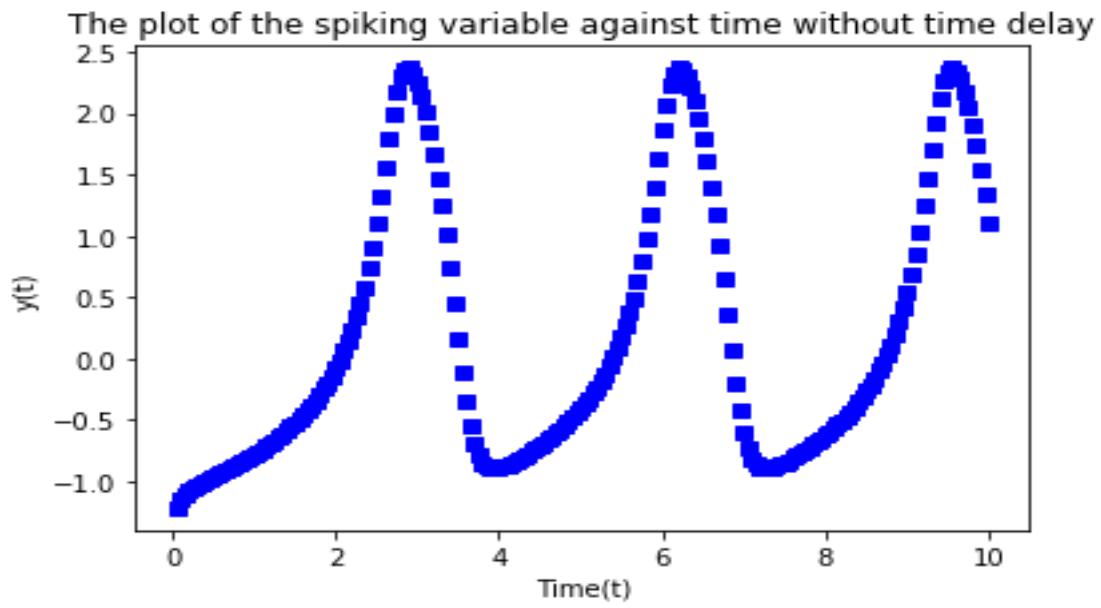

(b)

Phase potrait

(c)

Fig.4.1: Solution trajectories without delay evaluated around P_1. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable, (c): Phase portrait of the membrane potential against the spiking variable.
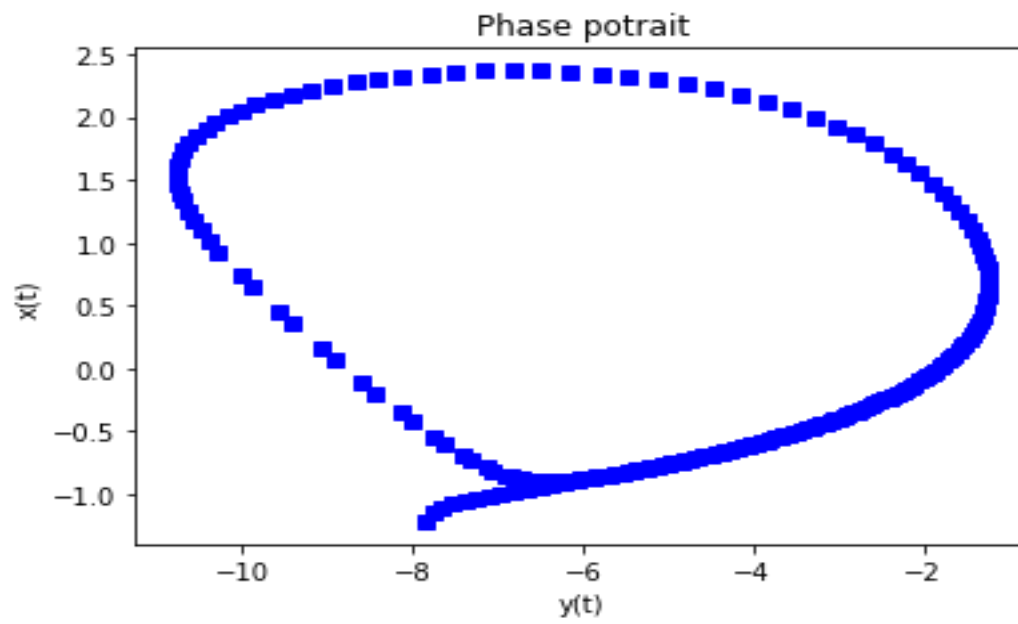
Case2: The HR fast sub-system with $\tau = 0$ and $z = 0$ $evaluated$ $around$ P_2

The plot of the membrane potential against time without time delay
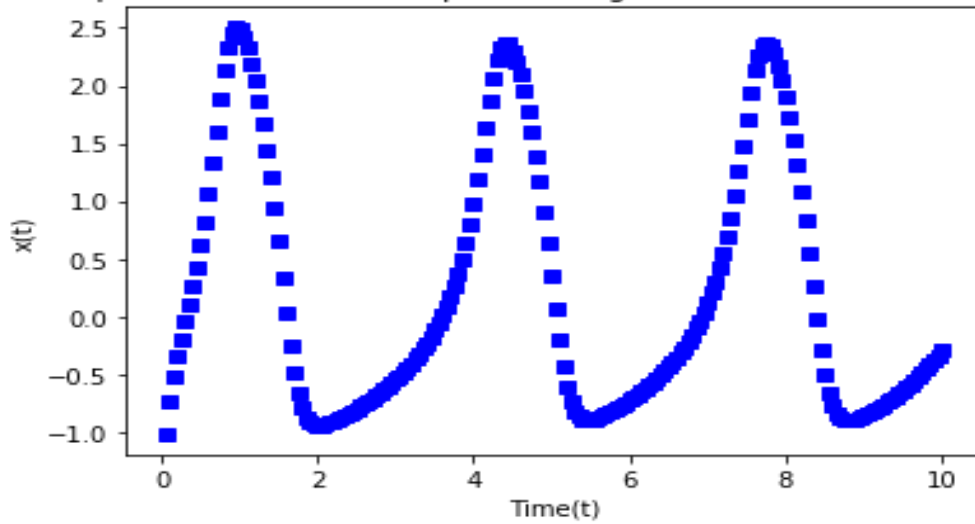
The plot of the spiking variable against time without time delay

Phase potrait

Fig 4.2: Solution trajectories without delay evaluated around P_2. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable, (c): Phase portrait of the membrane potential against the spiking variable.

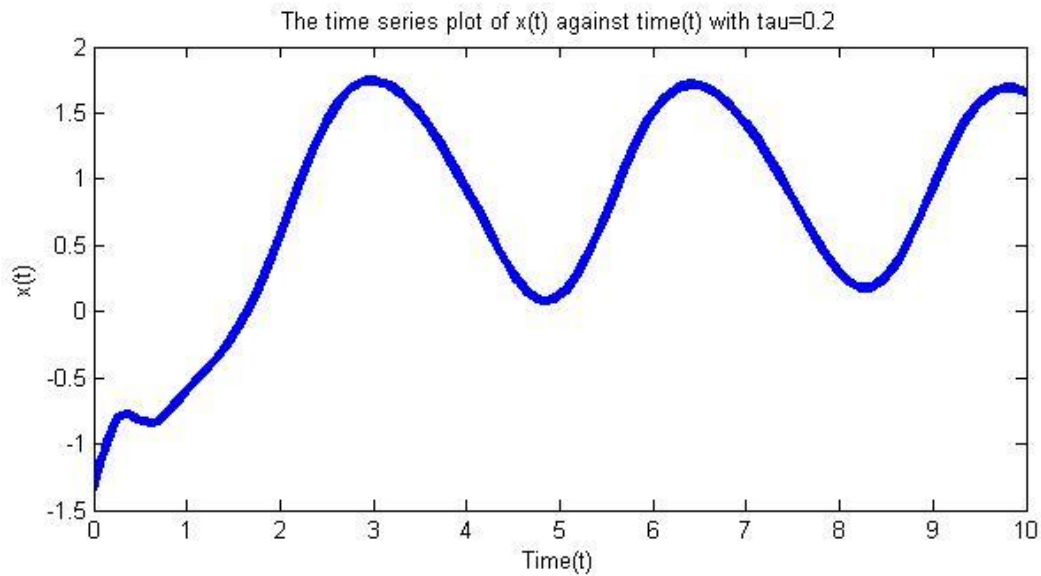Case 3: The HR fast subsystem in the presence of delay evaluated at P_2

The time series plot of x against t when tau =0.1

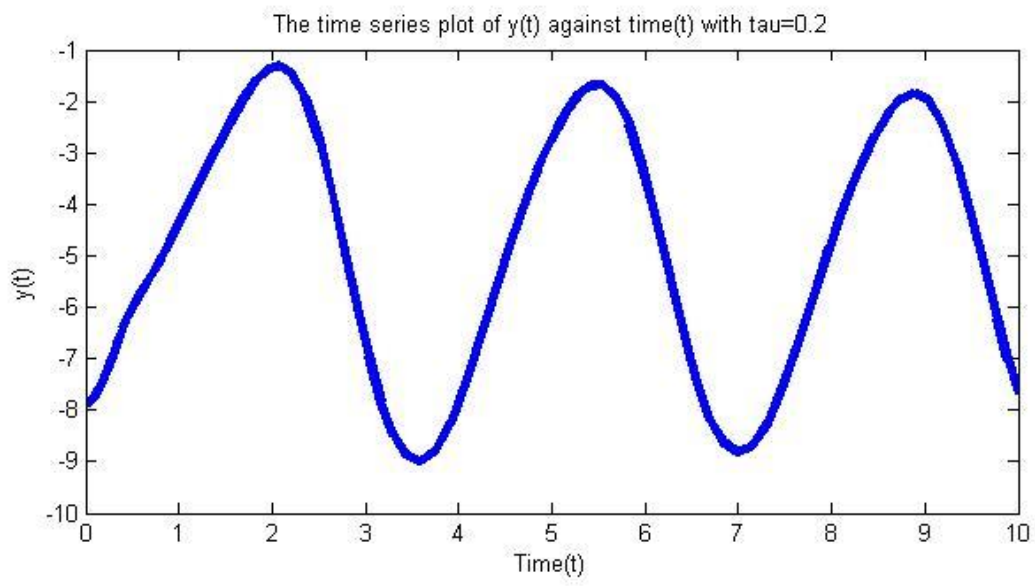(a)

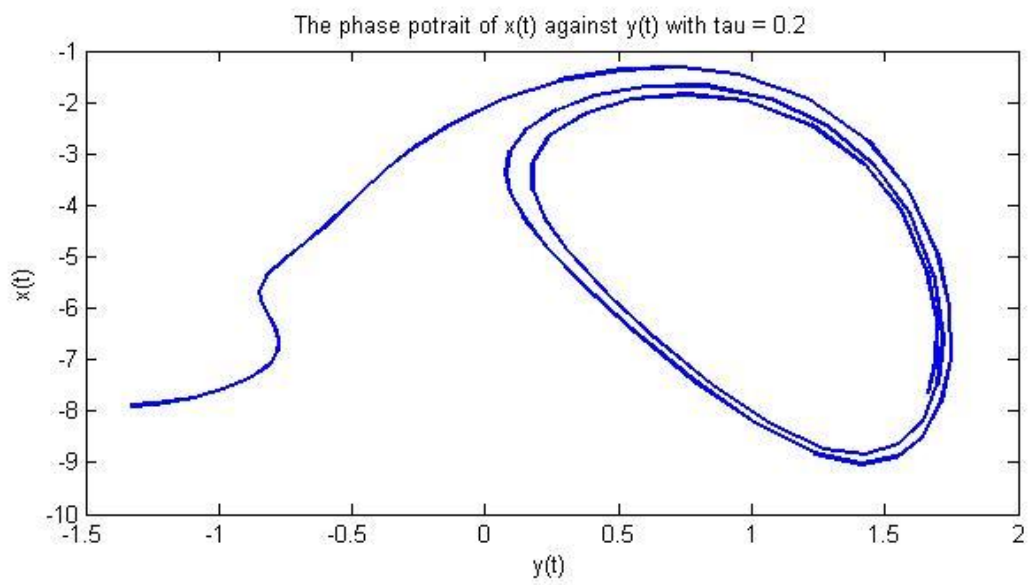The time series plot of y against t when tau =0.1
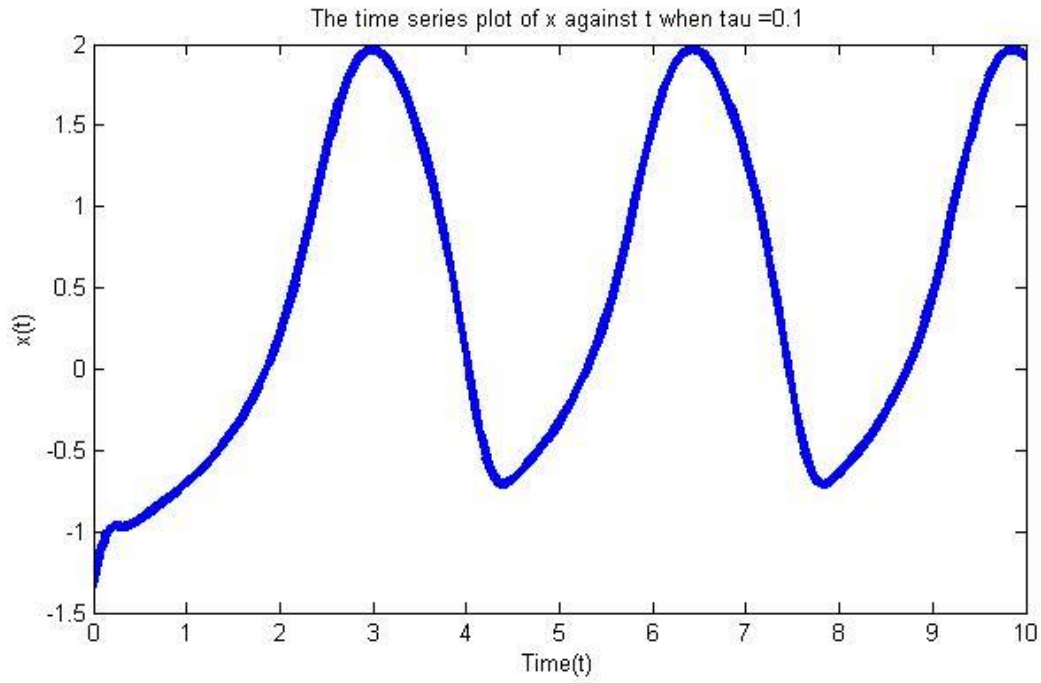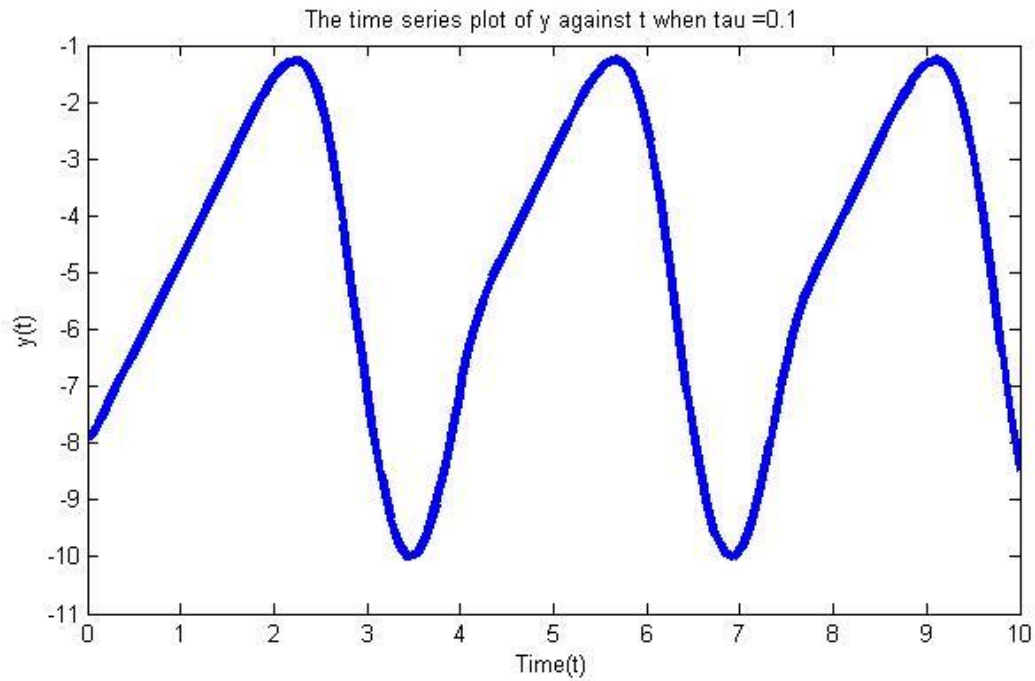
(b)
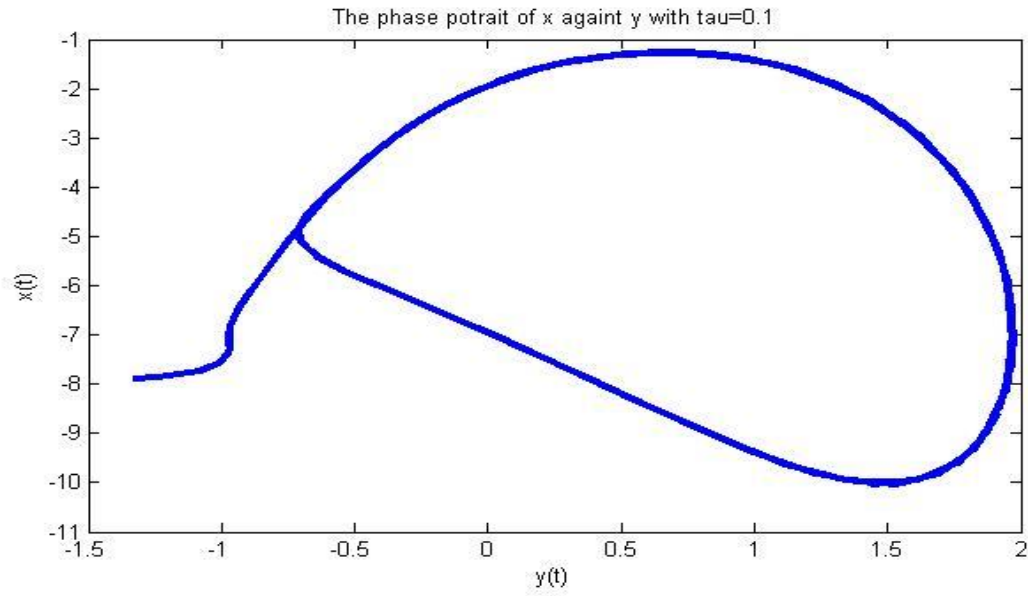
(c)

Fig 4.3: Solution trajectories with tau=0.1 evaluated around P_2. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable, (c): Phase portrait of the membrane potential against the spiking variable.


(a)

The time series plot of y(t) against time(t) with tau=0.2

(b)

The phase potrait of x(t) against y(t) with tau = 0.2

(c)

Fig 4.4: Solution trajectories with tau=0.2 evaluated around P_2. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable, (c): Phase portrait of the membrane potential against the spiking variable.
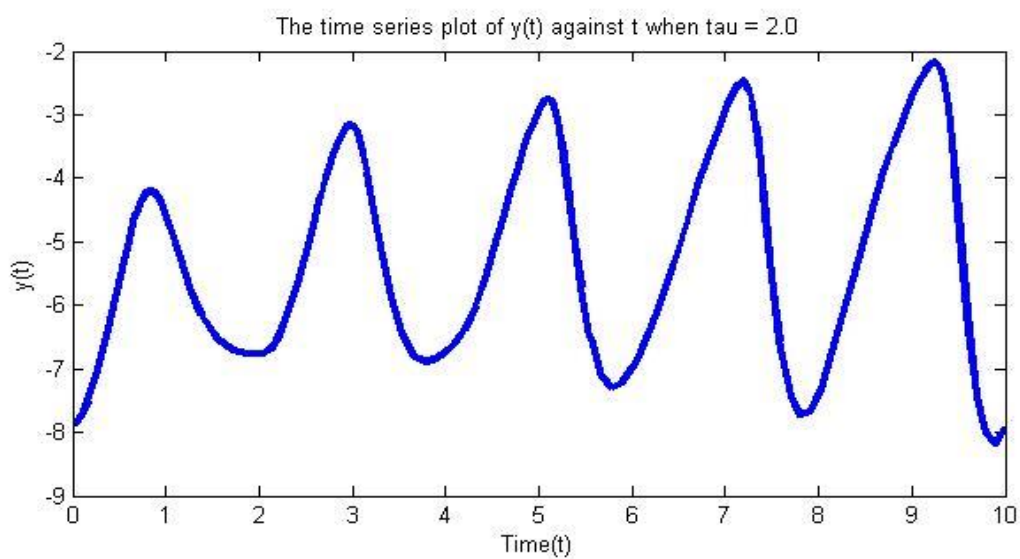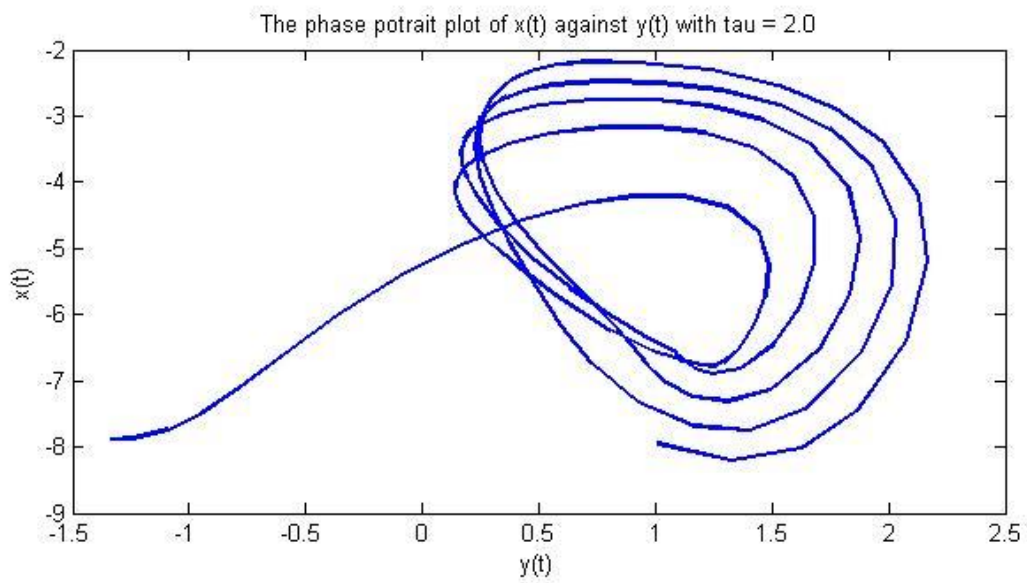

(a)

(b)

The phase potrait plot of x(t) against y(t) with tau = 2.0
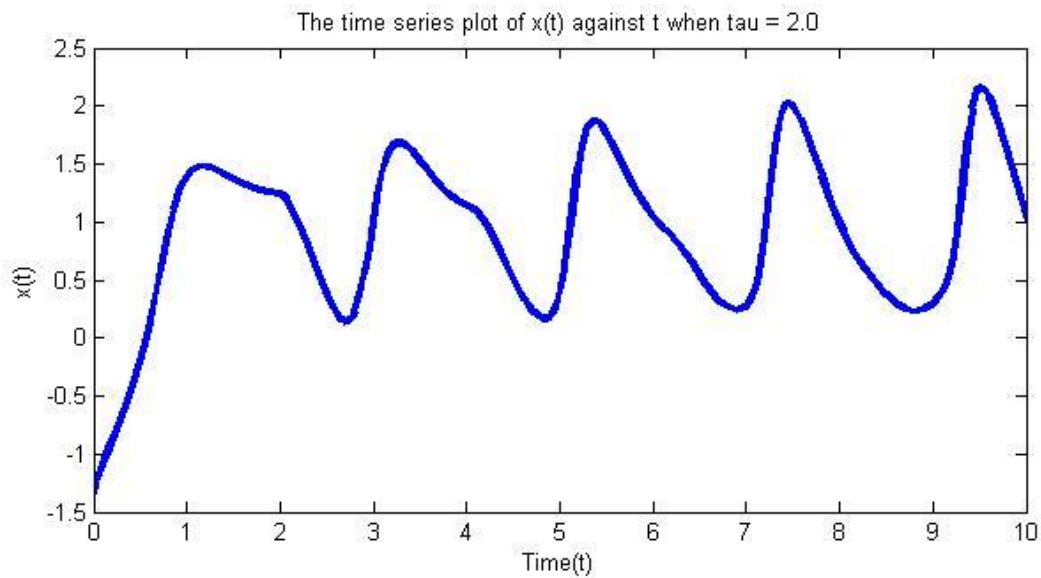
(c)

Fig 4.5: Solution trajectories with tau=2.0 evaluated around P_2. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable, (c): Phase portrait of the membrane potential against the spiking variable.
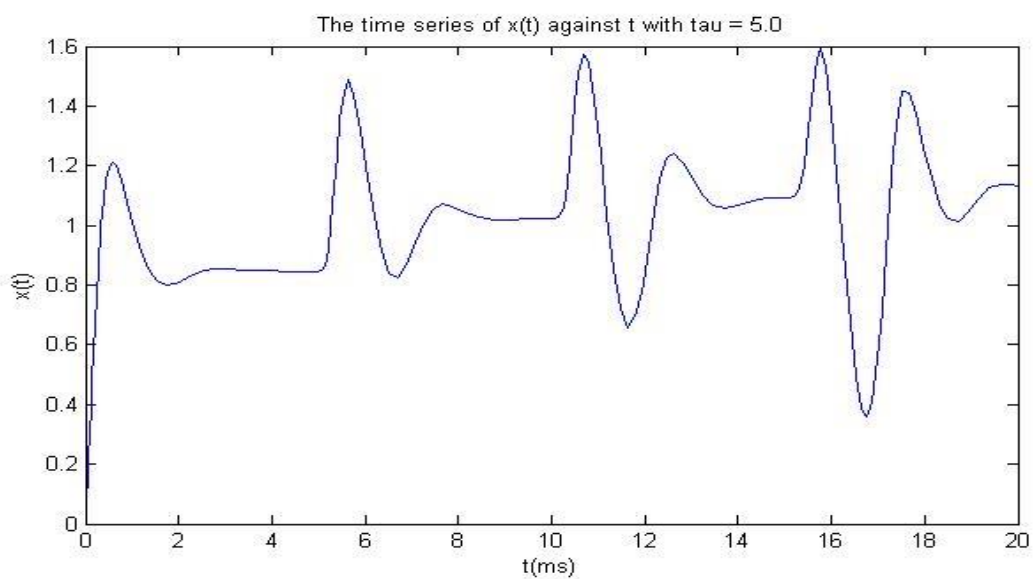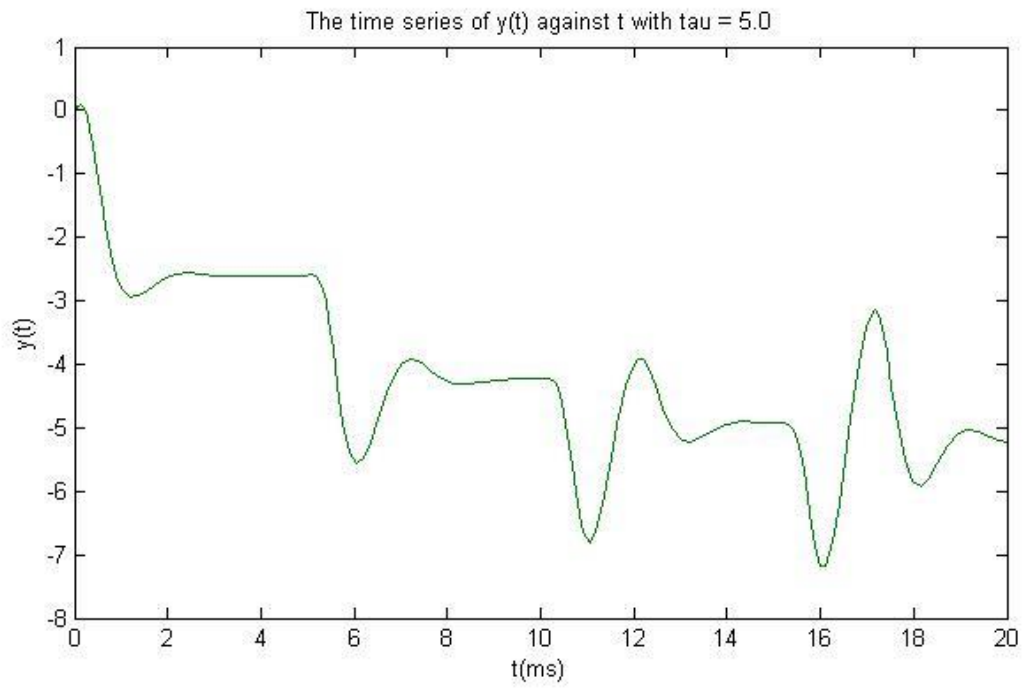

The time series of x(t) against t with tau = 5.0

(a)

The time series of y(t) against t with tau = 5.0

(b)

Fig 4.6: Solution trajectories with tau=5.0 evaluated around P_2. (a): Time series of the membrane potential, (b): Time series plot of the spiking variable.

5. CONCLUSION

The dynamical behaviour of the Hindmarsh-Rose model with time delay was intensively investigated. By applying the Descartes' sign rule, which is a powerful tool for real polynomials with constant coefficients, we have determined the number of real zeroes of a polynomial function. Classifications of the imaginary roots of the characteristic equation were presented. It was proved that the Hindmarsh-Rose system was absolutely stable for any time delay less than the critical delay. Time series curves and phase portrait were drawn using dde23 tool. It reveals that the equilibrium point 1 is neutral stable and 2 is asymptotically stable. We have seen that there exist a critical value of the delay where the asymptotically stable system becomes unstable called the bifurcation point/value (at $\tau = 0.2$). Therefore, for $\tau > 0.2$, the system is unstable.

The time delay can induce various dynamical transitions in HR model system, in particular, medium time delay can lead to the transition from bursting oscillation to relaxation oscillation and complex oscillation, and large time delay can result in chaotic bursting oscillation.

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FUTURE WORK

- Incorporating the model with double time delay.

- Considering the model with a time dependent delay.

- Considering the case of a coupled neuron with both dependent and independent time delay.

- Describe the local kinetics of the neuronal network and the memristive electromagnetic induction current with time delay.