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Deterministic and stochastic bifurcations in the Hindmarsh-Rose neuronal model

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We analyze the bifurcations occurring in the 3D Hindmarsh-Rose neuronal model with and without random signal. When under a sufficient stimulus, the neuron activity takes place; we observe various types of bifurcations that lead to chaotic transitions. Besides the equilibrium solutions and their stability, we also investigate the deterministic bifurcation. It appears that the neuronal activity consists of chaotic transitions between two periodic phases called bursting and spiking solutions. The stochastic bifurcation, defined as a sudden change in character of a stochastic attractor when the bifurcation parameter of the system passes through a critical value, or under certain condition as the collision of a stochastic attractor with a stochastic saddle, occurs when a random Gaussian signal is added. Our study reveals two kinds of stochastic bifurcation: the phenomenological bifurcation (P-bifurcations) and the dynamical bifurcation (D-bifurcations). The asymptotical method is used to analyze phenomenological bifurcation. We find that the neuronal activity of spiking and bursting chaos remains for finite values of the noise intensity. © 2013 AIP Publishing LLC.

The rich and interesting dynamical behavior of the Hindmarsh-Rose (HR) neuronal model is one of the most studied in neurological sciences. The neuronal activity, which is substantially determined by a sufficient stimulus influence, is described by a system of ordinary differential equations. The model reveals a complex bifurcation solution reflecting the daintiness activity of a genuine neuron: a succession of a rest state, firing action potentials, and deactivation period. The action potentials firing and its nature (periodic or chaotic) depend on some key parameters of the model like, for example, the applied current $I$, which leads to the appearance of the neuronal activity via the instability of the equilibrium points. As many other natural systems, a genuine neuron belongs to an environment and is dominated by the presence of many fellow entities. The appropriated way to understand the neuronal activity is to include the stochastic influence that amounts to a non-linear noisy action. Two kinds of bifurcations occurring in the model show a close relationship with the deterministic dynamics. Our study reveals that under the influence of random Gaussian signal, the HR neuronal model becomes stable when the noise intensity increases.

I. INTRODUCTION

In recent years, mathematical and physical contributions to the flight of Neurological sciences have improved our understanding of neuronal behavior. Neuronal membranes display an excitatory electrical activity, in out-of-equilibrium conditions; they are maintained by the active ionic pumps. Under the action of external stimuli, the membrane potential can exhibit a complicated dynamical evolution in connection with variations of the intra-membrane ionic currents. Signals among neurons are probably transmitted as patterns of action potentials (spikes) of different complexity. The most common signals are constituted by continuous firing of action potentials (i.e., spiking dynamics) or by oscillations between tonic spiking and a silent (resting) state (i.e., bursting dynamics). Phenomenological and neurophysiological models developed to reproduce the electrical activity of cell membranes have provided many nontrivial examples of dynamical systems. The richness of behaviors exhibited by such models is often associated with extremely complicated bifurcation diagrams. The Hindmarsh-Rose (HR) neuronal model²,³ represents an example, for its ability to reproduce spiking or bursting dynamics upon variation of an external parameter (the dc current). This neuronal model²,³ may be seen either as a generalization of the Fitzhugh equations⁴ or as a simplification of the physiologically realistic model proposed by Hodgkin and Huxley.⁵ Besides the rich bifurcation diagram, the HR neuronal model displays two different chaotic regimes: bursts and spiking solutions. The characterization of these two dynamical states and of the transition between them is analyzed in Ref. 1.

Lindner et al.⁶ and Reinker et al.⁷ have reviewed the behavior of theoretical models of excitable systems driven by Gaussian white noise, with several applications in biophysics and lasers, together with cellular automata and phase models that considered prototypes of excitable stochastic dynamics. Various phenomena due to noise, such
as stochastic resonance,\textsuperscript{8} stochastic signal detection,\textsuperscript{9,10} stochastic synchronization,\textsuperscript{11} dynamical phase transitions,\textsuperscript{12} and delayed noise\textsuperscript{13,14} are also typical of excitable systems.

About the effects of additive noise in the HR neuronal model, Xia and Qi-Shao\textsuperscript{15} have studied the resonance effects and synchronization of HR neuronal model, and investigated the coherence resonance of a single HR neuron with Gaussian white noise added to the membrane potential. Wang \textit{et al.}\textsuperscript{16} found that the forced HR neuronal model with an additive Gaussian white noise leads to three kinds of firing modes: multi-modal firing, bi-modal firing, and intrinsic oscillation; and classified the relevant experimental observations on the periodically forced sensory neural systems through their dynamical status. Longtin and co-workers developed a bistable dynamic single neuron model embedded in external Gaussian white noise, and provided a simple and general explanation of the skipping phenomenon by comparing the multi-modal interspike interval histogram to the histogram of the residence time distribution of the noisy bistable system.\textsuperscript{17,18} Their results indicate that the mechanism of the skipping phenomenon is analogous to stochastic resonance.\textsuperscript{19} Our aim is to find the conditions (i.e., the type of bifurcation) that make the equilibrium points unstable. About noise, we will investigate if the bursting-spiking dynamics is smeared by random fluctuations.

In this paper, we consider the 3 dimensional version of the \textit{Hindmarsh-Rose (3D HR) neuronal model with and without a random signal. Our aim is to find the bifurcations structures of the model and the effects of the random signal. The paper is organized as follows: Sec. II presents the 3D HR neuronal model without random signal. Section III deals with the deterministic bifurcation structures, analyse the equilibrium solutions, and their stability. Moreover, various bifurcations that lead to chaotic states and the transitions between bursting chaos and spiking chaos are also analyzed. In Sec. IV, we numerically integrate the stochastic 3D HR neuronal model. The approach is based on stochastic ordinary differential equations and the corresponding \textit{Fokker-Planck} equations, treated by both analytical manipulations and numerical simulations. We find stochastic bifurcations, such as \textit{P-bifurcation} and \textit{D-bifurcation} structures. Section V concludes.

II. THE 3D HINDMARSH-ROSE NEURONAL MODEL

The 3D \textit{HR} neuronal model,\textsuperscript{3} a simplified version of the \textit{Hodgkin-Huxley} neuronal model and a modification of the \textit{FitzHugh} equations, has been originally proposed to model the synchronization of firing of two snail neurons. It can be formulated by means of the following equations:

\[
\begin{align*}
\dot{x} &= y - x^3 + \alpha x^2 - z + I, \\
\dot{y} &= 1 - dx^2 - y, \\
\dot{z} &= r[s(x - x_1) - z].
\end{align*}
\]

(1)

Here, \(x(t)\) represents the neuronal membrane potential, \(y(t)\) and \(z(t)\) are the recovery variables to account for the fast and slow ions currents fluxes across the cytoplasm, respectively. The parameter \(r\) dictates the difference scale between the fast and the slow dynamics. The applied current \(I\), which distinguishes normal physiological activity, is the fluxes of ionic charges across the cytoplasm occurring when a sufficient stimulus is applied to the neuron (that is, in fact an electrical potential). The fluxes can be controlled during the experiments to leap out their influence on the action potential on the neuronal activity. Moreover, the flux due to \(z(t)\) is a slow current whose rate of change is of the order of the small parameter \(r\) \((0 < r < 1)\) that governs the bursting and adaptation behavior of the model, while \(s\) governs adaptation. Through this study, we set the parameter of the 3D \textit{HR} neuronal model as follows: \(a = 3\), \(d = 5\). The equilibrium of the 2 dimensional \textit{HR} system, \(x_1 = -\frac{1}{3}(1 + \sqrt{5})\), corresponds to the threshold potential to trigger bursts. The 3D \textit{HR} neuronal model is known to demonstrate many types of robust activities that are generic for most \textit{Hodgkin-Huxley} neuronal models. It allows also for some regulation of the bursting activity that is referred to the square-wave bursting\textsuperscript{23,25} in neuronal models.\textsuperscript{26-33}

Neuronal activity is a mixture of two phases: (1) the rest phase, when the neuron does not emit any action potential (also known as the slow neuronal dynamical behavior); (2) the spiking or fast dynamic phase characterized by a repeated emission of action potential. These two phases lead to the bursting phase, the fast repeated emission of action potential, separated by slow periods. In Eq. (1), the fast dynamics is given by the variables \(x(t)\) and \(y(t)\). The main variable \(z(t)\) induces the slow neuronal dynamic in the 3D \textit{HR} neuronal model, which depends on the parameter \(r\), and is generally of the order \(r = 0.001\).

III. DETERMINISTIC BIFURCATIONS IN THE 3D \textit{HR} NEURONAL MODEL

A. Stability of the equilibrium solutions

In this section, we perform the deterministic bifurcation analysis of the model. First, we find the equilibrium solutions as a function of the neuronal parameters \(I\) and \(s\). Various dynamical structures can be observed for the action potential, \(x(t)\).\textsuperscript{34} We find that the action potential decays to a constant value, as well as oscillatory solutions: be periodic firings of a single spike, periodic firings of well-defined bursts of spikes, or chaotic firings of spikes and bursts of spikes.

At the equilibrium point \(E = (x_e, y_e, z_e)\), the variables \((x(t), y(t), z(t))\) reach a stationary state, while the equilibrium point corresponding to \(\dot{x} = \dot{y} = \dot{z} = 0\), depends on the parameters \(I\) and \(s\), and also on the variable \(\Delta\):

\[
\Delta = a^2 + \frac{11}{27}p^3
\]

\[
= I^2 - 2I \left[ -\frac{11}{27} - s \left( x_1 + \frac{11}{27} \right) \right]
\]

\[
+ \left[ -\frac{11}{27} - s \left( x_1 + \frac{2}{3} \right) \right] + \frac{4}{27} \left( s - \frac{4}{3} \right)^3. \quad (2)
\]
One finds that the number of equilibrium points depends on the sign of $\Delta$. If $\Delta$ is positive, there is one equilibrium solution; for $\Delta = 0$, there are two equilibrium solutions; finally for $\Delta < 0$, three equilibrium solutions are possible.

We focus now on the sign of $\Delta$, by consider the new variable $\Delta_\Delta$, the discriminant of Eq. (2):

$$\Delta_\Delta = \frac{16}{27} \left( \frac{4}{3} - s \right)^3, \quad \text{with} \quad s \leq \frac{4}{3}. \quad (3)$$

The two solutions $I_1$ and $I_2$ of the equation $\Delta = 0$ are

$$\begin{align*}
I_1 &= -\frac{11}{27} - \left( x_1 + \frac{2}{3} \right) s - 2 \left[ \frac{1}{3} \left( \frac{4}{3} - s \right) \right]^2, \\
I_2 &= -\frac{11}{27} - \left( x_1 + \frac{2}{3} \right) s + 2 \left[ \frac{1}{3} \left( \frac{4}{3} - s \right) \right]^2.
\end{align*} \quad (4)$$

The dependence of the parameters $I$ and $s$ on the sign of $\Delta$ are shown in Fig. 1. It appears that in the region defined by (1a) and (1b), $\Delta > 0$ and the 3D HR neuronal model has one equilibrium point, $E^1(x_r, y_r, z_r)$ defined by

$$E^1 \begin{cases} 
x_r = \frac{\sqrt{-q + \Delta}}{2} + (y_r - 5x_r^2, \\
y_r = 1 - 5x_r^2, \\
z_r = s(x_r - x_1),
\end{cases} \quad (5)$$

while in the region (2), one finds three equilibrium points $E^3(x_r, y_r, z_r)_{(i=a,b,c)}$ of the 3D HR neuronal model ($\Delta < 0$)

$$E^3 \begin{cases} 
x_a = \frac{i\sqrt{3}}{2} \cos \left[ \frac{\arccos \left( -\frac{q}{2}\sqrt{27} - \frac{27}{-p^3} \right)}{3} \right], \\
y_a = 1 - 5x_a^2, \\
z_a = s(x_a - x_1),
\end{cases} \quad E^3 \begin{cases} 
x_b = \frac{i\sqrt{3}}{2} \cos \left[ \frac{\arccos \left( -\frac{q}{2}\sqrt{27} + \frac{27}{-p^3} \right) + \frac{2\pi}{3}}{3} \right], \\
y_b = 1 - 5x_b^2, \\
z_b = s(x_b - x_1),
\end{cases} \quad E^3 \begin{cases} 
x_c = \frac{i\sqrt{3}}{2} \cos \left[ \frac{\arccos \left( -\frac{q}{2}\sqrt{27} + \frac{4\pi}{3} \right) + \frac{2\pi}{3}}{3} \right], \\
y_c = 1 - 5x_c^2, \\
z_c = s(x_c - x_1),
\end{cases} \quad (6)$$

The case $\Delta = 0$ corresponds to the boundary between the two above cases, see Fig. 1, where the 3D HR neuronal model has two equilibrium points $E^2(x_r, y_r, z_r)_{(i=a,b)}$

$$E^2 \begin{cases} 
x_a = \frac{3q - 2p}{-2p}, \\
y_a = 1 - x_a^2, \\
z_a = s(x_a - x_1),
\end{cases} \quad E^2 \begin{cases} 
x_b = \frac{3q - 2p}{2p}, \\
y_b = 1 - x_b^2, \\
z_b = s(x_b - x_1),
\end{cases} \quad (7)$$

To understand the dynamics of the 3D HR neuronal model described by Eq. (1), it is interesting to investigate the nature of the equilibrium points, as a function of the neuronal parameters $I$ and $s$, through the eigenvalues of the Jacobian matrix $J$, i.e., small perturbations around the equilibrium $(x_r, y_r, z_r)$. Setting $\delta \mathbf{X} = (\delta x, \delta y, \delta z)^T$ and $\mathbf{X} = (x, y, z)^T$, Eq. (1) lead to

$$\delta \mathbf{X} = J(x_r, y_r, z_r) \delta \mathbf{X},$$

where $J$ reads

$$J(x_r, y_r, z_r) = \begin{pmatrix} 6x_r - 3x_r^2 & 1 & -1 \\
-10x_r & -1 & 0 \\
rs & 0 & -r \end{pmatrix}. \quad (9)$$

The equilibrium point is stable if all the real parts of the eigenvalues $\lambda = \mu + j\nu$ ($\nu^2 = -1$) of $J$ are negative. The associated characteristic equation is

$$-\lambda^2 + \lambda^2(-3x_r^2 + 6x_r - 2) + \mu[-6x_r^2 + 22x_r - r(1 + s)] + r(-3x_r^2 + 16x_r - s) = 0. \quad (10)$$

Let us consider in the $(I, s)$-plane a point $P(I, s)$, and the corresponding equilibrium points $E(x_r, y_r, z_r)$. In the region of a single equilibrium solution, for example, the point $P_1(I, s) = (-1, \frac{11}{12} + (x_1 + \frac{2}{3} + \frac{1}{3}))$, and the corresponding equilibrium solution $E^1(1.115, -5.218, -2.733) \equiv E_1$ is a

![FIG. 1. Map in the plan (s,I) showing the number of equilibrium points. In the regions (1.a) and (1.b), the neuronal model has one equilibrium point, while in the region (2), one finds three equilibrium points. The boundary between the above two regions corresponds on the case of two equilibrium points.](image-url)
saddle-focus, since the corresponding eigenvalues are \( \lambda_1 = 0.9 + 2.6j, \lambda_2 = 0.9 - 2.6j, \lambda_3 = -0.0009 \). The bifurcation scenario for the equilibrium point \( E_1^0 \) presented in Fig. 2(i), leads to a series of spikes after a short transition period. When Eq. (1) admits two equilibrium points, the set \( P_2(s, I) \) belongs to the boundary \( \Lambda \equiv 0 \), the behavior becomes singular with the appearances of non-hyperbolic equilibria which drive the system into saddle-node bifurcations. Fig. 2(ii) illustrates the phase portrait of the saddle-node bifurcation for \( (s, I) = [0, -\frac{11}{2} - 2(\frac{1}{2})^3] \). The two equilibrium points are \( E_2^0(-2, -19, 0) \equiv E_2 \) and \( E_3^0(0, 1, 0) \equiv E_3 \). The eigenvalues of the characteristic Eq. (2) for \( E_2^0 \) and \( E_3^0 \) are \( (\lambda_1 = -24.839, \lambda_2 = -0.161, \lambda_3 = -0.001) \) and \( (\lambda_1 = 0, \lambda_2 = -1, \lambda_3 = -0.001) \), respectively. Since \( E_2^0 \) is stable and \( E_3^0 \) is unstable, all the trajectories converge to the stable point as it is clear in Fig. 2(ii). The bifurcation scenario in which the ionic fluxes are shut away in some fixed values making the emission of a constant value of the action potential even the applied current variation lay in a huge domain.

Thirdly, we consider the case of three equilibrium solutions, see region (2) of Fig. 1. With the set \( P_3(s, I) = \{0, -\frac{11}{2} - 1, \frac{1}{2}\} \), the equilibrium solutions are \( E_4^0(0, 3, 0, 2, 0) \equiv E_4 \), \( E_5^0(1.9, -17.1, 0) \equiv E_5 \), and \( E_6^0(-0.4, -0.1, 0) \equiv E_6 \), which are a focus-saddle, a stable node, and a saddle solution, for the eigenvalues obtained for each equilibrium \( E_4^0, E_5^0, E_6^0 \). The two equilibrium points \( a \) and \( b \) of Fig. 6. One also finds that chaotic behavior appears before another chaotic region. As \( s \) increases further, a reverse period doubling transition takes place. Thus, the influence of \( s \) through the product \( r \) accentuates, maintains, even destroys the difference between the slow and the fast dynamic of the neuronal model. The variation of \( s \) can drive the system into several unpredictable changeovers. The variation of the Lyapunov exponent and the bifurcation diagram versus \( s \) are showed in Fig. 6. One also finds that chaotic behavior appears on the 3D HR neuronal model.

**B. Deterministic bifurcation and Chaotic states**

Deterministic bifurcation occurs for some values of the neuronal parameters. For illustration, we numerically solve Eq. (1) and the corresponding variational equations, with a standard 4th order Runge–Kutta method with time step size 0.001, and plot the resulting bifurcation diagram as a function of \( r, s \), and \( I \). Fig. 3 shows a chaotic phase portrait (Fig. 3(i)) and the evolution of the action potentials (Figs. 3(ii)–3(iv)), while Figs. 4–6 show the bifurcation diagrams and the corresponding Lyapunov exponents. The Lyapunov exponent being defined by

\[
\lambda_{\text{max}} = \lim_{t \to \infty} \frac{1}{t} \ln \left| \frac{\delta X(t)}{\delta X(0)} \right| = \lim_{t \to \infty} \frac{1}{t} \ln |D\Phi(X)|, \tag{11}
\]

where \( \Phi \) is the flow of the HR neuronal model and \( D\Phi(X) \), the Jacobian of \( \Phi \) defined at the state \( X \). We focus on the parameter \( r \) which measures the difference between the fast dynamic and the slow dynamic. We observe in Fig. 4 the quasi-periodic oscillations for \( r < 0.0078 \) where it bifurcates in chaotic state. At \( r = 0.0099 \), a tiny multiperiodic window appears before another chaotic region. As \( r \) increases further, a reverse period doubling transition takes place. Thus, the parameter \( r \) induces a complex sequence of period. In Fig. 5, for the applied current \( I \in [1.345, 4] \), we find a quasi periodic behavior for \( I \in [1.345, 3.282] \) and a periodic behavior for \( I \in [3.322, 4] \) with a small chaotic region for \( I \in [3.282, 3.322] \). Fig. 5 shows 23 regular spikes per burst before entering the chaotic phase. The influence of \( s \) through the product \( r \) accentuates, maintains, even destroys the difference between the slow and the fast dynamic of the neuronal activity. The variation of \( s \) can drive the system into several unpredictable changeovers. The variation of the Lyapunov exponent and the bifurcation diagram versus \( s \) are showed in Fig. 6. One also finds that chaotic behavior appears on the 3D HR neuronal model.
FIG. 3. Strange attractor of the 3D HR neuronal model. The parameters used are $r = 0.008001$, $s = 4$, and $I = 3.322$. In (i), we show the phase portrait. In (ii), (iii), and (iv), we show the action potential, the fast ionic fluxes, and the slow ionic fluxes.

FIG. 4. Bifurcation diagram and variation of the maximum Lyapunov exponent versus the parameter $r$; the parameters used are $s = 4; I = 3.25$.

FIG. 5. Bifurcation diagram and variation of the maximum Lyapunov exponent versus the parameter $I$. The parameters used are $r = 0.001; s = 4$. 
The quaint sight of the bifurcation diagram associated with the variation of the external current reveals a complex evolution from bursting to spiking solutions. The transition is chaotic from bursting chaos to spiking chaos. Different states occurring within this type of transition can be found in Refs. 1, 25, and 34. Examples of the time evolution of action potential of the neuronal activity are shown in Fig. 7.

IV. STOCHASTIC BIFURCATIONS IN THE 3D HR NEURONAL MODEL WITH NOISE

Stochastic bifurcation may be defined as a sudden change in character of a stochastic attractor when the bifurcation parameter of the system passes through a critical value. It is found that under certain conditions, a stochastic bifurcation always occurs when a stochastic attractor collides with a stochastic saddle. We will investigate if such phenomena occur and lead to the disappearance of the dynamics described in Sec. III.

A. The 3D HR neuronal model with random signal

It is known that the emission of the action potential can occur be made if there is a sufficient stimulus applied to the neuron. Thus, the propagation of the potential to another neuron depends on the depolarization induced by the prior neuron. A neuron can be upriver linked to one or many other neurons with different ionic fluxes across their cytoplasm, the effective stimulus injected to the nervous cell can be considered a random term which is the result of many unlike applied currents. Gaussian white noise statistical features seem appropriated to mimic the complex reality exhibited by a neuron under the influence of the environment and the other neurons. Here, we only consider the case where the white noise is added through the electrical potential of the membrane, i.e., the effective current imposed to the nervous cell contains a random term

$$I_{eff} = I + e(t),$$

where $e(t)$ is the stochastic process, a Gaussian white noise with amplitude $D$ and defined by the properties

$$\langle e(t) \rangle = 0,$$
$$\langle e(t), e(t') \rangle = 2D \delta(t - t'),$$

which completely determines its statistical features. Hence, the stochastic 3D HR neuronal model reads

$$\begin{align*}
\dot{x} &= y - x^3 + ax^2 - z + I_{eff} \\
\dot{y} &= 1 - dx^2 - y \\
\dot{z} &= r[s(x - x_1) - z].
\end{align*}$$

There are main two definitions of stochastic bifurcation: one is based on a sudden change of the steady-state probability density function, called the P-bifurcation; and the other is based on a change of the sign of the maximum Lyapunov exponent (MLE), called D-bifurcation. To analyze the stochastic bifurcations in the HR neuronal model, we therefore compute numerically the probability density functions - P-Bifurcation and the Lyapunov exponent - D-Bifurcation.

B. P-bifurcation and algorithm of numerical simulations

1. Definition: P-bifurcation

Phenomenological bifurcation is concerned with the change in the shape of density (stationary probability density) of a family of random dynamical systems as the change of the parameter. Here, the P-bifurcation is the change in the shape of the density of the HR neuronal model as the intensity of the noise changes.

2. Algorithm of numerical simulations

The evolution of the probability density function of the neuron state is made here by means of the solution of the Fokker-Planck equation associated with Eq. (14). As usual, the Fokker-Planck equation leads to a stochastic partial derivatives equation with three variables, whose analytical solution is difficult to find. Therefore, only the numerical analysis of this partial derivative equation is tackled. The finite difference numerical method allows us to find the bifurcations of the probability density. The slow dynamical
subsystem which is governed by the $z$-variable of the 3D HR model is neglected, the parameter $r$ is small and according to the asymptotical method one variable can be neglected, i.e., $\dot{z} \approx 0$. Thus, Eq. (14) can be rewritten as

$$\begin{align*}
\dot{x} &= y - x^3 + ax^2 + I + \epsilon(t) \\
\dot{y} &= 1 - dx^2 - y.
\end{align*}$$

(15)

The probability density function $P(t,x,y)$, satisfies the equation

$$\begin{align*}
\frac{\partial P(t,x,y)}{\partial t} &= \sum_{i=1}^{2} \frac{\partial \left[ -f_{i}(x,y)P(t,x,y) \right]}{\partial x_i} \\
&\quad + \sum_{i=1}^{2} \sum_{j=1}^{2} \frac{\partial^2 \left[ D_{ij}P(t,x,y) \right]}{\partial x_i \partial x_j},
\end{align*}$$

(16)

where $(x_1, x_2) \equiv (x, y)$ and $D_{ij}$ is the diffusion matrix given by

$$D_{ij} = \begin{pmatrix}
D & 0 \\
0 & 0
\end{pmatrix}.$$  

(17)

So, the Fokker-Planck equation is

$$\begin{align*}
\frac{\partial P(t,x,y)}{\partial t} &= \frac{\partial \left[ -f_{1}(x,y)P(t,x,y) \right]}{\partial x} \\
&\quad + \frac{\partial \left[ -f_{2}(x,y)P(t,x,y) \right]}{\partial y} + \frac{\partial^2 P(t,x,y)}{\partial x^2} + D^2 \frac{\partial^2 P(t,x,y)}{\partial x^2}.
\end{align*}$$

(18)

The set of parameters used is $a = 3$, $d = 5$, $I = 3.322$ and the diffusion coefficient $D = 0.1$. Using the time splitting method, Eq. (8) is reformulated in terms of splitting operators $L_i (i = 1, 2, 3)$ as

$$\frac{\partial P}{\partial t} = L_1 P + L_2 P + L_3 P.$$  

(19)

$L_i$ operators introduction simplifies the linear equations system construction by breaking up Eq. (19) into three directions of a progressive resolution:

$$\begin{align*}
L_1 u &= -\frac{\partial}{\partial x} [f_1 u] \\
L_2 u &= -\frac{\partial}{\partial y} [f_2 u] \\
L_3 u &= \frac{\partial^2}{\partial x^2} [D u].
\end{align*}$$

(20)

Equation (18) is numerically solved by the implicit stable pattern for each splitting operator with the Von Neumann stability analysis. The spatial grid is made of $(2N + 1) \times (2N + 1)$ nodes with $N = 100$ and the time step is fixed to $\Delta t = 0.001$.

The external perturbation is a Gaussian white noise, and the initial probability density $P(t = 0, x, y)$, is a Gaussian function whose peak should not be very high, therefore we chose

$$P(t = 0, x, y) = \exp \frac{-(x - \mu_1)^2}{2\sigma_1^2} \exp \frac{-(y - \mu_2)^2}{2\sigma_2^2}.$$  

(21)

the parameters $\sigma_1 = \sigma_2 = 0.9$, $\mu_1 = \frac{\lambda_{max} - \lambda_0}{2}$ and $\mu_2 = \frac{\gamma_{max} - \gamma_0}{2}$. Here, $\lambda_{max}, \lambda_0$, $\gamma_{max}$, and $\gamma_0$ are the boundaries of the spatial domain.
3. P-Bifurcation

The initial repartition is the Gaussian function defined in Eq. (21). The probability density function rapidly evolves, first decreasing and then stretching out in the initial domain, up to the amplitude of neuronal spiking firing (Figs. 8(i) and 8(ii) at $t = 0.025$ and $t = 0.05$, respectively). As a result, an enlargement of the initial domain must be considered to get a clear visualization. It is crucial to underline that the diffusion coefficient effect gains advantage over the $I$-effect, that never causes considerable changeovers. Then, in Figs. 9(i)–9(viii), we shut the evolution times going from $t = 0.15$ to the steady state $t = 0.5$. The probability density function to find the neuron emission of action potential and ionic fluxes decreases while spreading over a huge domain (something like $[-50; 50] \times [-3000; 3000]$), with however, tiny peak values of the order of $10^{-14}$. Thus, the diffusion effect on the neuronal behavior is the most prominent and it can drive the system to a very large range of firing response.

In conclusion, we observe that as the noise intensity increases, the shape of the steady-state probability density function changes and a stochastic P-bifurcation occurs. We analyze in Sec. IV C, the D-bifurcation phenomenon, resulting on a sudden change of the MLE.

C. D-bifurcation and algorithm of numerical simulations

1. Definition: D-bifurcation

Dynamical bifurcation is concerned with a family of random dynamical systems which is differential and has the invariant measure $\mu_z$. If there exists a constant $z_D$ satisfying in any neighbourhood of $z_D$, there exists another constant $z$ and the corresponding invariant measure $\mu_z \neq \mu_z$ satisfying $\nu_z \rightarrow \mu_z$ as $z \rightarrow z_D$. Then, the constant $z_D$ is a point of dynamical bifurcation.

2. Algorithm of numerical simulations

To generate numerically the Gaussian white noise, we start from two random numbers $b_1$ and $b_2$ which are uniformly distributed on the unit interval $[0;1]$ and, with the Box-Mueller algorithm, we generate a standardized Gaussian-distributed sequence. We use the simple Euler algorithm to integrate Eq. (14); the numerical scheme is

$$\begin{align*}
\epsilon_{\mid t+\Delta t} &= \sqrt{-4D\Delta t \ln(s)} \cos(2\pi b_2) \\
x_{\mid t+\Delta t} &= x + (y - x^3 + ax^2 - z + f)\Delta t + \epsilon_{\mid t} \\
y_{\mid t+\Delta t} &= y + (1 - dx^2 - y)\Delta t \\
z_{\mid t+\Delta t} &= z + (r[s(x - x_1) - z])\Delta t.
\end{align*}$$

The main purpose is to determine the critical value of the diffusion coefficient (or noise amplitude) $D$ at which the neuronal behavior becomes periodic or stable, according to the sign of the maximum Lyapunov exponent $\lambda_{\text{max}}$ (see Eq. (11)) averaged over 50 realizations. If $\lambda_{\text{max}} < 0$, the neuronal activity is stable or periodic; if not the neuronal activity is simply unpredictable.

3. D-bifurcation

We firstly investigate the influence of the noise intensity $D$ on the small parameter $r$, seeking for sudden changes of the maximum Lyapunov exponent, or D-Bifurcations. In Fig. 10, we plot the average of the Lyapunov exponent $\lambda_{\text{max}}$ versus $D$ for three different values of $r$. The critical values of the noise intensity $D_{\text{cr}}$, a point of Dynamical bifurcation, for the transition between chaotic and stable states, are $D_{\text{cr}} = 1.27$ for $r = 0.0010$, $D_{\text{cr}} = 1.99$ for $r = 0.0080$, and $D_{\text{cr}} = 1.27$ for $r = 0.0150$. For instance, we find that before $r \simeq 0.0157$, the behavior of the 3D HR neuronal model is deterministically chaotic. Therefore, for a value of $r$ laying in this interval, the noise (whose first effect is to disturb the neuronal activity) increases the chaotic state before the nervous cell reorganizes its firing dynamics. The time spent by the neuron under the diffusion effects in chaotic states increases with the value of $D$. In contrast, above $r \simeq 0.0157$, the neuronal activity is periodic: it spends no time in a chaotic state because his originally dynamic becomes stable. This is why for $r = 0.001; 0.008001$ and 0.045, the point of Dynamical bifurcation $D_{\text{cr}}$ increases from 1.27 to 1.99 before decrease to 1.27. This behavior is general and the details of the $r$-dependence of the critical $D$ value are shown in Fig. 11. This figure shows the stable domain (gray area) and the
unstable domain (white area) of the neuronal activity. The point \( D_{cr} \) decreases to zero \( (r \simeq 0.11) \), for \( r \) stabilizes the neuronal activity. The decrease in \( D_{cr} \) shows the noise effect on the neuronal activity. As it is illustrated in Fig. 12, the \( I \)-effect on the neuronal activity is totally different: the unstable domain is no more limited. This could be the consequence of the effective applied current in Eq. (14). The parameter contribution cooperates with the noise to increase somehow the diffusion and disturbs the neuron natural activity. Thus, when \( I \) increases, the noise effect increases, and the time spent by the neuron in an unpredictable activity also increases, as found by the raising of the \( D_{cr} \). Fig. 13 presents

FIG. 9. Evolution of the probability density function showing a P-bifurcation for \( t = 0.15 \) in (i), \( t = 0.2 \) in (ii), \( t = 0.25 \) in (iii), \( t = 0.3 \) in (iv), \( t = 0.35 \) in (v), \( t = 0.4 \) in (vi), \( t = 0.45 \) in (vii), and for \( t = 0.5 \) in (viii). At \( t \simeq 0.5 \), the system reaches the steady state. The parameters used are defined in Fig. 5.
V. CONCLUSIONS

We have studied the bifurcation structures of a 3D Hindmarsh-Rose neuronal model, with and without a random component. We have first found that the number of equilibrium points depends on $I$ and $s$. Although the map (Fig. 1) obtained is not large enough to predict all the equilibrium points, we observe the existence of unstable and even non-hyperbolic equilibria. This leads to a potential dynamical activity in the 3D Hindmarsh-Rose neuronal model Eq. (1). In fact, the deterministic bifurcations presented by the neuron reveal a complex structure when the parameters ($I; r; s$) are changed. Variations of the maximum Lyapunov exponent, bifurcations diagrams, phase portraits, and time series allowed us to show and characterize such a behavior. This complex bifurcation is uplifted by the existence of two types of chaotic response (bursting chaos and spiking chaos, which are a transition between two periodic activities named bursting and spiking) with the switching values of the applied current $I$. The effect of additive random component has also been studied. We observe the phenomenon of stochastic bifurcation: a sudden change in character of a stochastic attractor when the bifurcation parameter of the system passes through a critical value. Two types of stochastic bifurcations have found: the P-bifurcation and D-bifurcation, which demonstrate that the firing action potentials might be observed for finite values of the noise intensity. We conclude that large noise always leads to the suppression of the neuronal activity (bursting or spiking) activity. For a finite (and relatively low) value, the behaviors of system under noise are not dissimilar from the deterministic counterpart. The neuronal natural activity is always reinstated in the neuronal behavior after a disordely transient time, under the diffusion, if the parameters $r$ and $s$ are the $s$-influence on the critical $D$ value. The scenario resembles the behavior of Fig. 11 because the parameter $s$ acts in a way much similar to $r$, through the product $r \cdot s$ in the 3D HR neuronal model Eq. (1). The main difference is that the unstable domain as a function of $s$ is much larger than the domain of Fig. 11.
only variables. For $I$, the diffusion maintains an increasing impact on the neuronal activity.

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