Localized nonlinear excitations in diffusive Hindmarsh-Rose neural networks

F. M. Moukam Kakmeni,* E. M. Inack, and E. M. Yamakou

Complex Systems and Theoretical Biology Group, Laboratory of Research on Advanced Materials and Nonlinear Science (LaRAMaNS), Department of Physics, Faculty of Science, University of Buea, P.O. Box 63, Buea-Cameroon

(Received 2 January 2014; published 30 May 2014)

We study localized nonlinear excitations in diffusive Hindmarsh-Rose neural networks. We show that the Hindmarsh-Rose model can be reduced to a modified Complex Ginzburg-Landau equation through the application of a perturbation technique. We equally report on the presence of envelop solitons of the nerve impulse in this neural network. From the biological point of view, this result suggests that neurons can participate in a collective processing of information, a relevant part of which is shared over all neurons but not concentrated at the single neuron level. By employing the standard linear stability analysis, the growth rate of the modulational instability is derived as a function of the wave number and systems parameters.

DOI: 10.1103/PhysRevE.89.052919 PACS number(s): 05.45.Yv, 05.65.+b, 87.18.−h

I. INTRODUCTION

Nonlinear excitations of solitons type are localized solutions of a widespread class of weakly nonlinear dispersive partial differential equations. They were first observed by John Scott Russel [1–4] and turned out to originate from the balance between nonlinearity and dispersion. Their remarkable properties have been used to explain many yet unexplained phenomena such as the Fermi-Pasta-Ulam paradox [5] and to discover and establish new theories in many aspects of science and technology [2,4,6–9].

In the neuronal system, many studies have been carried out that noticed the presence of those peculiar nonlinear waves [10–13]. Note that the analysis of the mechanisms underlying spatial structures of activity in the neural tissue is important for understanding a wide range of both naturally occurring and pathological phenomena [14,15]. Thus, this work is motivated by experimental findings in the cortex and previous studies on diffusively coupled systems. For instance, in a nerve model with self-excitable membrane, localized short impulses were observed [16]. Modulated wave forms were obtained numerically from the time series of the membrane potential derived from the dynamical mechanisms of waxing and waning oscillations in thalamic relay neurons [17]; self-sustained oscillations were observed in real neural tissue [16,18]. In Ref. [19], it was shown numerically that the Hindmarsh-Rose (HR) neurons display nonlinear excitations in a spiking bursting behavior. All these studies report on the presence of nonlinear localized waves in a specific population of linked neurons under proper assumptions.

However, a clear analytical solution describing the dynamics of those diffusive nonlinear excitations has not yet been established. The form of the nerve impulse propagating in the neural network is very relevant because it stands as a signature to certain brain pathologies [20]. Moreover, knowing the conditions under which traveling waves of nerve activity can propagate in the cortical neural tissue is increasingly becoming an active area of research. This is due to the fact that, determining conditions under which cortical waves propagation occurs is primordial to comprehend the normal processing of sensory stimuli as well as more pathological forms of behavior such as migraines, epileptic seizures, Parkinson diseases etc. [20–23].

Consequently, the present work aims at using both analytical and numerical methods to study the dynamics of nonlinear excitations in a network of diffusively coupled HR neurons. We use the Liénard form of the diffusive HR model to come out with a modified complex Ginzburg-Landau (CGL) equation by means of a specific perturbation technique. The modified CGL equation is an equation that describes the evolution of modulated waves in this neural network. From there, we make use of the envelope soliton solution of the CGL established by Nozaki and Bekki [24] to obtain an expression of the nerve impulse. The modulational instability in the diffusive HR neural network is performed and it points to the fact that due to the combine effects from nonlinearity, dispersion, and dissipation, a small perturbation on the envelope of a nerve impulse plane wave may induce an exponential growth of its amplitude, resulting in the carrier-wave breakup into a train of localized waves [25–27].

Thus, this paper is organized as follows: Section II is devoted to the description of the coupled HR neural model. Section III aims at finding envelope solitons in the diffusive HR model by applying the multiple scale expansion in the semidiscrete approximation. In Sec. IV, we look for the conditions under which plane wave propagating in the diffusive neural network will become stable or unstable to small perturbation. We end the work with a conclusion in Sec. V.

II. THE HINDMARSH-ROSE COUPLED MODEL

There exists a variety of models used to mimic neuronal activities. We have the Hodgkin-Huxley model [28], Fitzhugh-Nagumo model [29], or more recently the ones of Morris-Lecar [30] and of Hindmarsh-Rose [31,32]. The later gives a wider view of the Fitzhugh-Nagumo model by taking into consideration bursting (series of spikes). Its primary goal is to study the spiking-bursting behavior of the membrane potential observed in experiments made with a single neuron. The Hindmarsh-Rose model is governed by a set of three nonlinear ordinary differential equations on the dimensionless variables $x(t)$, $y(t)$, and $z(t)$. The most important variable in this model is $x(t)$, which represents the membrane potential (nerve impulse).

*moukamkakmeni@gmail.com

The variable *y(t)* accounts for the measure of the rate at which transport of sodium and potassium ions is made through fast ion channels; it is also called the *spiking variable*. *z(t)* instead is the *bursting variable*, which takes into consideration the rate at which the transport of other ions (C_l^- and proteins anions) made through slow ions channels. It enables the model to control the rest period between two action potentials. These equations are a simple polynomial model of bursting in thalamic cells, which capture certain generic features of more complicated biophysical models [19,31,32]. Since neuronal activity takes all its significance when considered in a network, we are going to consider a network of N dynamical neurons.

The equation for a network of N Hindmarsh-Rose neurons coupled through the gap junction is given by

$$\begin{aligned} \dot{x_i} &= y_i - a{x_i}^3 + b{x_i}^2 - z_i + I, \\ &\quad + K(x_{i+1} - 2x_i + x_{i-1}) \\ \dot{y_i} &= c - d{x_i}^2 - e y_i, \\ \dot{z_i} &= r[s(x_i - x_0) - z_i], \end{aligned} \tag{1}$$

with $i = 1,...,N$.

The roles played by the system parameters are the following:

The parameter b allows one to switch between bursting and spiking behaviors; it also controls the spiking frequency. r controls the speed of variations of the bursting variable. s is the recovery variable. a is a unitary value of s that determines spiking behavior. x_0 is the equilibrium coordinate of the two-dimensional subsystem given by the first two equations of Eqs. (1) when $I = 0$ and $z_i = 0$. c, d, and e are constant parameters.

The coupling strength of the gap junction is K. In this paper, we have considered two nearest neighbors coupling in a weak coupling regime. From the biological point of view, neurons only make electrical connections with their nearest neighbors. Also a weak coupling between neighboring cells is a situation that arises in the study of bursting activity in the β-cell islets of the pancreas, which secrete insulin in response to glucose in the blood [19,33]. In normal physiological conditions, this variability may reflect different levels of expression of certain types of receptors or differences in regulatory effects induced by internal or external neuromodulatory processes. Some pathological conditions elicited by specific genetic mutations or by drug abuse are also known to be related to significant modifications of the level of neural membrane excitability [15].

I represents the stimulation current and it is the bifurcation parameter, determining the qualitative behavior of the neuron. In general, depending on the value of the stimulation current I the dynamical regime of the HR neuron can describes quiescence, subthreshold, suprathreshold, and chaotic bursting behaviors. For instance, in the case of an isolated HR neuron, when $I = 0$ there can be only one stable stationary solution and it corresponds to the stable quiescence behavior of the neuron. Furthermore, for $I \in (0,1.3)$ the HR neuron exhibits only subthreshold responses. However, for $I \in (2.92,3.40)$, and for the commonly used values of other parameters as given below, the HR model describes chaotic bursting, i.e., a series of spikes that are chaotically interspersed with refractory periods and quiescence behavior. The original values of the parameters that define the HR model are: $a = 1.0$, $b = 3.0$, $c = 1.0$, $d = 5.0$, $r = 0.008$, $s = 4.0$, $x_0 = -\frac{(1+\sqrt{5})}{2}$, and $e = 1.0$.

Typically, large scale neuronal networks can exhibit a number of spatially structured activity states. Oscillations, waves, and spatial structure of diffusively coupled neurons systems have been observed through numerical simulations [10–13,19]. Numerical simulations, however, typically show complex dynamics because a large number of tilted waves can be excited and compete with others. We therefore use the semidiscrete approach to analytically derive the type of localized excitations that propagate in network of coupled HR neuron model. In order to apply this method, it is convenient to transform the system into the wave form. To do so, we reduce the first and second equations in Eqs. (1) into a second-order ODE in x_i. We achieve this by differentiating the first equation in Eqs. (1) and substituting $\dot{y}_i$ into the obtained second-order ODE. From the biological standpoint, single cell models of bursting consist of a set of ordinary differential equations governing the behavior of the fast variable and at least one slow variable [19]. The above transformations allow us to conveniently write Eqs. (1) in a Liénard form that is a second-order differential equation with a small damping term. The governing equations then become

$$\begin{aligned} &\ddot{x_i} + {\Omega_0}^2 x_i + \left(\gamma_0 + \gamma_1 x_i + \gamma_2 {x_i}^2\right)\dot{x_i} + \lambda_1 {x_i}^2 \\ &+ \frac{\gamma_2}{3}{x_i}^3 + \lambda_3 z_i + I_0 = D_0(x_{i+1} - 2x_i + x_{i-1}) \\ &+ D_1(\dot{x}_{i+1} - 2\dot{x}_i + \dot{x}_{i-1}) \\ &\dot{z_i} = r[s(x_i - x_0) - z_i] \end{aligned} \tag{2}$$

where $\Omega_0, \gamma_0, \gamma_1, \gamma_2, \lambda_1, \lambda_3, I_0, D_0$, and D_1 are constant parameters of different order to be determined during the application of the semidiscrete approach.

It is important to note that the transformation did not fundamentally affect the structure of the system. It is thus still possible to decompose the obtained system, activity into a fast subsystem described by x, which treats the slow variables as parameters, and a slow subsystem defined by z. Equation (2) is a set of coupled nonlinear ODEs similar to those that generally describe the dynamics of atomic chain. It is almost impossible to solve these equations analytically; however, nearly exact solutions can be obtained using perturbation methods. In order to determine the order of the different terms, we introduce the variables

$$x_i = \epsilon \varphi_i, \tag{3}$$

$$z_i = \epsilon \psi_i, \tag{4}$$

where $\epsilon \ll 1$. In Eqs. (2), the parameter λ_3 couples the equations of the membrane potential to those of the bursting variable. However, due to the fact that the variation of the bursting variable is slower than the one of the membrane potential, we can consider λ_3 to be a perturbed parameter of order ϵ^2. Also, since we are looking for solution in a weakly dissipative medium, the parameters γ_0 and D_1 are also considered to be perturbed at the order ϵ^2. Keeping the first two nonlinear terms of the development, the system of Eqs. (2)

becomes

$$\ddot{\varphi}_i + {\Omega_0}^2\varphi_i + \epsilon\left(\epsilon\gamma_0 + \gamma_1\varphi_i + \epsilon\gamma_2{\varphi_i}^2\right)\dot{\varphi}_i + \epsilon\lambda_1{\varphi_i}^2 + \epsilon^2\frac{\gamma_2}{3}{\varphi_i}^3 + \epsilon^2\lambda_3\psi_i = D_0(\varphi_{i+1} - 2\varphi_i + \varphi_{i-1}) + \epsilon^2 D_1(\dot{\varphi}_{i+1} - 2\dot{\varphi}_i + \dot{\varphi}_{i-1}), \quad (5)$$

$$\dot{\psi}_i - r\psi_i - {\Omega_0}^2\varphi_i = 0. \quad (6)$$

Equation (5) is therefore the equation regulating the dynamics of the membrane potential in the HR coupled model, and it resembles the one governing the nondimensional dynamics of the potential difference across the cell membrane for a FitzHugh-Nagumo model [34]. The second equation, Eq. (6), is the coupling term describing the dynamic of the wave due to the burst variable of the HR neural model.

As stated earlier in this work, we are looking for solitons made up of carrier waves modulated by envelope signal, which are called envelope solitons. This type of solitons appears naturally for most weakly dispersive and nonlinear systems, which are described by a wave equation in the small amplitude limit [2,3]. Since we are studying low-amplitude nonlinear excitations in a weakly diffusive neural network, it is adequate to use *multiple scale expansions*, which can be applied to the *semidiscrete approximation*. Thus, the aim of the following section is to find analytically and numerically envelope solitons of the diffusive Hindmarsh-Rose model. We will start by describing the multiple scale method in the semidiscrete approximation, then we will apply it to our model in order to obtain a modified complex Ginzburg-Laudau (CGL) equation. Analytical and numerical solutions of this equation will be established.

III. MULTIPLE SCALE EXPANSION IN THE SEMIDISCRETE APPROXIMATION

The semidiscrete approximation is a perturbation technique in which the carrier waves are kept discrete while the amplitude is treated in the continuum limit. Applying this method allows one to study the modulation of a plane wave caused by nonlinear effects.

We proceed by making a change of variables according to the new space and time scales $U_i = \epsilon^i u$ and $T_i = \epsilon^i t$, respectively. It should be noted that the principal purpose of using the multiple scale expansion is to find a solution $x(u,t)$ depending on these new sets of variables as a perturbation series of functions. We will consider here that

$$x(u,t) = \sum_{i=1}^{\infty}\epsilon^i\varphi_i(U_0,U_1,U_2,...,T_0,T_1,T_2,...), \quad (7)$$

$$z(u,t) = \sum_{i=1}^{\infty}\epsilon^i\psi_i(U_0,U_1,U_2,...,T_0,T_1,T_2,...), \quad (8)$$

where each U_i and T_i is treated as an independent variable. Thus, we also obtain a perturbation series of operators from all independent variables:

$$\frac{\partial}{\partial t} = \frac{\partial T_0}{\partial t}\frac{\partial}{\partial T_0} + \frac{\partial T_1}{\partial t}\frac{\partial}{\partial T_1} + \frac{\partial T_2}{\partial t}\frac{\partial}{\partial T_2} + ...,$$

implying that

$$\frac{\partial}{\partial t} = \frac{\partial}{\partial T_0} + \epsilon\frac{\partial}{\partial T_1} + \epsilon^2\frac{\partial}{\partial T_2} + ... \quad (9)$$

An important feature of this method is that the solution of the original problem will only be obtained if the multidimensional space generated by the new sets of variables X_i and T_i come from the physical line [2,3]: $T_0 = t$; $T_1 = \epsilon t$; $T_2 = \epsilon^2 t$. The greatest asset of these auxiliary variables is that they permit us to impose appropriate conditions to the system, thus assuring that the asymptotic expansion converges uniformly for small values of ϵ [2,3]. Using the notation $D_i = \frac{\partial}{\partial T_i}$, we thus obtain

$$\frac{\partial}{\partial t} = D_0 + \epsilon D_1 + \epsilon^2 D_2 + ... \quad (10)$$

And similarly, the spatial derivatives can be expressed by

$$D_{U_i} = \frac{\partial}{\partial U_i},$$
$$\frac{\partial}{\partial u} = D_{U_0} + \epsilon D_{U_1} + \epsilon^2 D_{U_2} + ... \quad (11)$$

The following analytical procedure consists of replacing the new form of the nerve impulse and of the bursting variable (the one depending on new variables) and the derivatives in the different terms of the membrane potential equation of motion. We then group terms in the same power of ϵ, which leads us to a system of equations. Each of those equations will correspond to each approximation for specific harmonics.

A. Equation of motion of the amplitude

Now, let us consider the following solutions of our coupled HR model:

$$\varphi_i = B_i e^{i\theta_i} + B_i^* e^{-i\theta_i} + \epsilon(C_i + D_i e^{2i\theta_i} + D_i^* e^{-2i\theta_i}) + O(\epsilon^2), \quad (12)$$

$$\psi_i = F_i e^{i\theta_i} + F_i^* e^{-i\theta_i} + \epsilon(G_i + H_i e^{2i\theta_i} + H_i^* e^{-2i\theta_i}) + O(\epsilon^2), \quad (13)$$

with $\theta_i = qu_i - wt$ where q is the normal mode *wave vector* and w is the *angular velocity* of the wave.

Here, we look for nerve impulses having the form of nonlinear localized excitations of the diffusive neuronal network. The fact that there are nonlinear terms in Eq. (5) [respectively, Eq. (6)] incites one to predict that through frequencies superpositions, the first harmonics of the wave will contain terms in $e^{\pm 2i\theta_i}$ as well as terms without any exponential dependence. We will equally consider that the amplitudes *B, C, D*(respectively, *F, G, H*) change slowly in space and time. That is why for them, we are going to do a continuum limit approximation and a multiple scale expansion. To sum up, we are going to deal with the amplitude in the continuum limit while keeping the carrier wave discrete.

In this semidiscrete approximation, *B, C, D* (respectively, *F, G, H*) are supposedly independent of the "fast" variables t and u. Instead, they depend on the "slow" variables $U_1 = \epsilon u$, $U_2 = \epsilon^2 u$, $T_1 = \epsilon t$, $T_2 = \epsilon^2 t$. Applying now the continuum limit approximation on these amplitudes

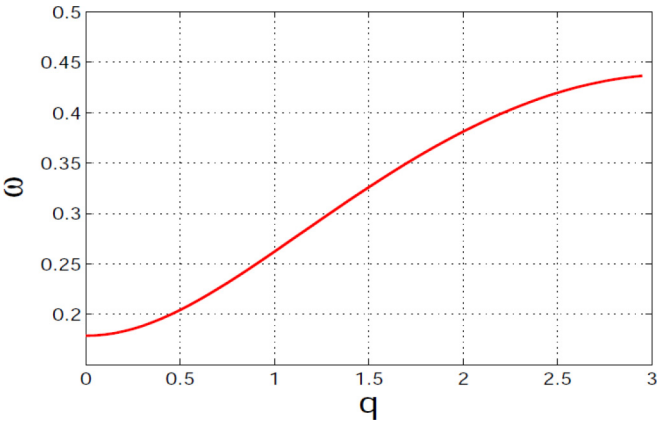

FIG. 1. (Color online) The dispersion relation of the nerve impulse. $D_0 = 0.04$, $\Omega_0^2 = 0.032$.

will yield to $B_i(t)$, $C_i(t)$, and $D_i(t)$ becoming, respectively, $B(U_1,U_2,T_1,T_2)$, $C(U_1,U_2,T_1,T_2)$, and $D(U_1,U_2,T_1,T_2)$. $B_{i\pm1}$ is obtained at ϵ^2 by a Taylor expansion,

$$B_{i\pm1} = B \pm \epsilon \frac{\partial B}{\partial U_1} \pm \epsilon^2 \frac{\partial B}{\partial U_2} + \frac{\epsilon^2}{2}\frac{\partial^2 B}{\partial U_1^2} + O(\epsilon^3),$$

and its temporal derivative is given by

$$\frac{\partial B_i}{\partial t} = \epsilon \frac{\partial B}{\partial T_1} + \epsilon^2 \frac{\partial B}{\partial T_2} + O(\epsilon^3).$$

An identical process is done for C_i and D_i (respectively, F_i, G_i, H_i); therefrom, we substitute the above equations in Eqs. (5) and (6). We look for relations at different order of ϵ for which terms in $e^{\pm i\theta_i}$, $e^{\pm 2i\theta_i}$ and terms without exponential dependence cancel out.

At the order ϵ^0, the annihilation of terms in $e^{\pm i\theta_i}$ give the *dispersion relation* of linear waves of the system made up of Eqs. (5) and (6) (See Fig. 1):

$$w^2 = \Omega_0^2 + 4D_0 \sin^2\frac{q}{2}. \tag{14}$$

As expected, this is indeed the dispersion relation of a discrete model. The axonal waves dispersion is related to the system parameters of the neuronal network.

At the order ϵ^1, the cancellation of terms in $e^{i\theta_i}$ gives

$$\frac{\partial B}{\partial T_1} + v_g \frac{\partial B}{\partial U_1} = 0, \tag{15}$$

where

$$v_g = \frac{D_0 \sin q}{w} \tag{16}$$

is the *group velocity*.

We observe that the velocity of axonal waves depends on the diffuseness of the plasma membrane. The more diffusive the axon, the faster the nerve impulses. Resulting in a quick movement of the ions across the ion pumps and ion channels of the nerve cell membrane.

Terms without exponential dependence give the relation

$$C = -\frac{2\lambda_1}{\Omega_0^2} B B^*. \tag{17}$$

Terms with $e^{i2\theta_i}$ give the relation

$$D = \frac{(\lambda_1 - iw\gamma_1)}{3} \frac{B^2}{\Omega_0^2 + \frac{16}{3}D_0 \sin^4\frac{q}{2}}. \tag{18}$$

At the second order of perturbation, terms with $e^{i\theta_i}$ dependence give the relation for Eq. (6):

$$F = -\frac{\Omega_0^2(r - iw)}{r^2 + w^2} B. \tag{19}$$

This relationship comes from the coupling between the action potential and the bursting variable in the neural network. We note that it is complex and depends on system parameters. Still, at this order, terms depending on $e^{i\theta_i}$ yield the following relation for Eq. (5):

$$\begin{aligned}\frac{\partial^2 B}{\partial T_1^2} - 2iw\frac{\partial B}{\partial T_2} &= iw\gamma_0 B + (iw\gamma_1 - 2\lambda_1)(BC + B^* D) \\ &\quad + (iw - 1)\gamma_2 |B|^2 B + \frac{\lambda_3\left(r - iw\Omega_0^2\right)}{r^2 + w^2} B \\ &\quad + 4iwD_1 \sin\frac{q}{2}^2 B + 2iD_0 \sin q \frac{\partial B}{\partial U_2} \\ &\quad + D_0 \cos q \frac{\partial^2 B}{\partial U_1^2}.\end{aligned} \tag{20}$$

The solvability condition written in the mobile reference frame $\xi_i = U_i - v_g T_i$ and $\tau_i = T_i$ with velocity v_g yields the equation of evolution of the envelope function,

$$i\frac{\partial B}{\partial \tau_2} + \frac{P}{2}\frac{\partial^2 B}{\partial \xi_1^2} + Q|B|^2 B + i\frac{R}{2}B = 0, \tag{21}$$

where the coefficients P, Q, and R are given by

$$P = \frac{D_0 w^2 \cos q - D_0^2 \sin^2 q}{w^3}, \tag{22}$$

$$Q = Q_r + iQ_i, \tag{23}$$

$$R = R_r + iR_i. \tag{24}$$

Q_r and Q_i are the real and imaginary parts of the nonlinearity coefficient. The same terminology is applied for the dissipation coefficient:

$$Q_r = \frac{1}{w}\left[\frac{2\lambda_1^2}{\Omega_0^2} + \frac{\gamma_1^2 w^2 - 2\lambda_1^2}{6\left(\Omega_0^2 + \frac{16}{3}D_0 \sin^4\frac{q}{2}\right)} - \frac{\gamma_2}{2}\right], \tag{25}$$

$$Q_i = \frac{\lambda_1\gamma_1}{6\left(\Omega_0^2 + \frac{16}{3}D_0 \sin^4\frac{q}{2}\right)} - \frac{\lambda_1\gamma_1}{\Omega_0^2} + \frac{\gamma_2}{2}, \tag{26}$$

$$R_r = \gamma_0 + 4D_1 \sin^2\frac{q}{2} - \frac{\lambda_3\Omega_0^2}{r^2 + w^2}, \tag{27}$$

$$R_i = -\frac{r\lambda_3\Omega_0^2}{w(r^2 + w^2)}. \tag{28}$$

This equation shows that the evolution of modulated waves in this neural network model is described by the *modified complex Ginzburg-Landau* equation where the *nonlinearity*

and *dissipation coefficients* Q and R, respectively, are complex and the *dispersion coefficient* P is real [35].

The generalized complex Ginzburg-Landau equation is of paradigmatic importance to many field in physics. It is of tremendous importance in nonlinear optics, where it describes the full spatiotemporal optical solitons [36]. The study of the envelope soliton dynamics in monoatomic and diatomic lattices pays special attention in the last case to dependence of such dynamics on the width of the gap of the spectrum and to the companion modes [37]. The fact that one-dimensional nonlinear atomic chains support propagation of the so-called envelope solitons has been realized rather long ago and today they have become a subject of numerous studies (see, e.g., Ref. [2] and references therein). Such excitations are characterized by small amplitudes and involve quasiharmonic oscillations of many atoms. They display a rather stable behavior and in the leading approximation are described by the nonlinear CGL equation. In some cases, when the dissipation is not considered in the model, the modified complex Ginzburg-Landau equation reduces to the nonlinear Schrödinger equation in the case of diatomic lattices. For example, this is a fact that C. Tchawoua investigated when he studied the dynamics of solitons in diatomic nonlinear networks with a double-well site potential and a cubic coupling potential between nearest neighbors [3]. There, the motion of modulated waves were proven to be described by the complex Ginzburg-Landau equation. He showed that if the diatomic chain had to be substituted into a monoatomic chain without taking into account dissipation, the CGL equation would reduce to a nonlinear Schrödinger equation. However, that is not the case for the model under consideration in the present work; because even when the dissipation is neglected, the nonlinearity coefficient of the CGL equation remain complex. Thus, the equation of motion will always be modeled by the complex Ginzburg-Landau equation. To our knowledge, this is the first research work that attempts to describe the evolution dynamical behavior of solitons in networks of coupled HR neurons. From the biological point of view, this result suggests that neurons can participate in a collective processing of long-scale information, a relevant part of which is shared over all neurons but not concentrated at the single neuron level. Thus, the brain may actively work not only in time domain but also effectively use the spatial dimension for information processing [11,38–40]. The neuronal chain can process stimulus differently or identically in different circumstances. For example, waves of neural activity, functionally related to behaviors and global dynamics, have been found in visual, sensory-motor, auditory, and olfactory cortices [11,41].

The variations of constants P, Q_r, Q_i and of the product PQ_r with respect to the wave vector q are represented in Fig. 2. Whereas the variations of real and imaginary dissipative coefficients are presented in Fig. 3. Note that the real dissipative coefficient is positive, whereas the imaginary one is negative and very small.

Since the dispersion coefficient is real, the modulational instability depends on the sign of PQ_r. According to Benjamin-Feir instability, plane waves are unstable for positive values of PQ_r, while they are stable for negative values. Note that this stability criterion does not depend on the manner with which the wave propagates. Thus, one can expect to find in the diffusive neural network spatially localized nerve impulses for any wave carrier whose wave vector is in the positive range of PQ_r.

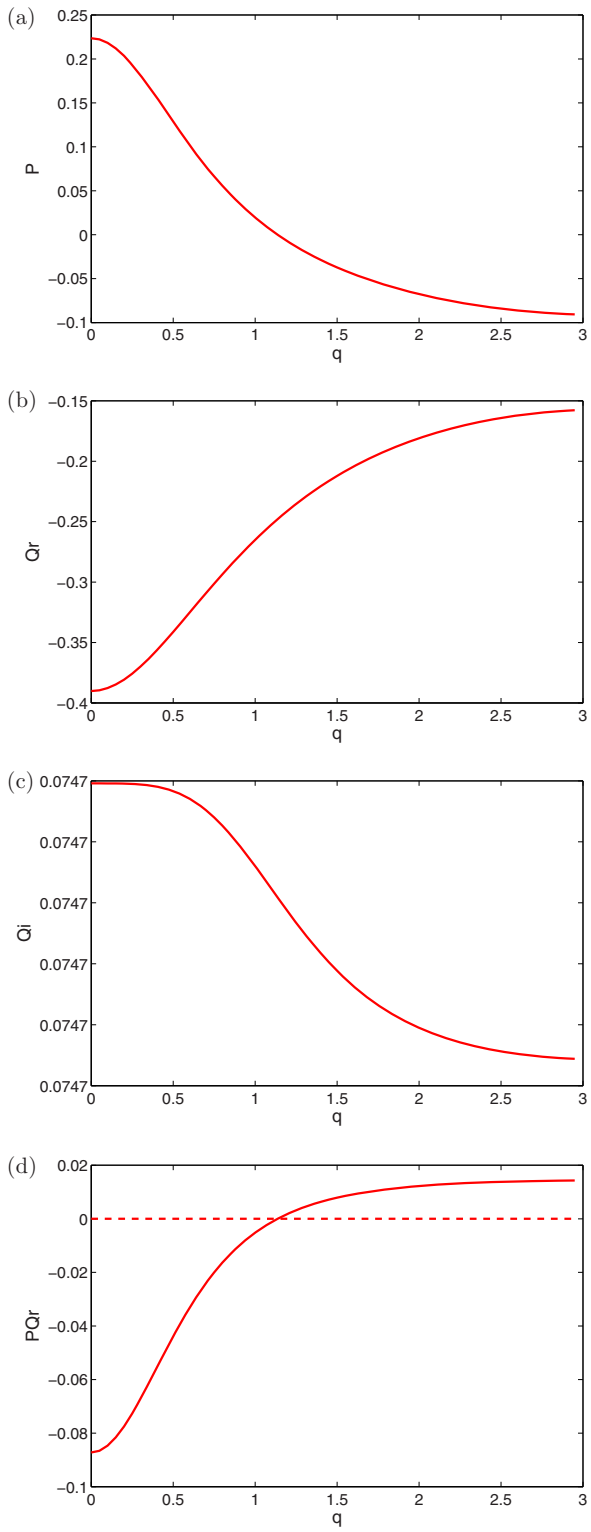

FIG. 2. (Color online) Variations of coefficients (a) P, (b) Q_r, (c) Q_i, (d) the product PQ_r in terms of the wave vector q of the carrier wave. $D_0 = 0.04$, $\Omega_0^2 = 0.032$, $\lambda_1 = 0.01$, $\gamma_1 = 0.001$, $\gamma_2 = 0.15$, $r = 0.008$.

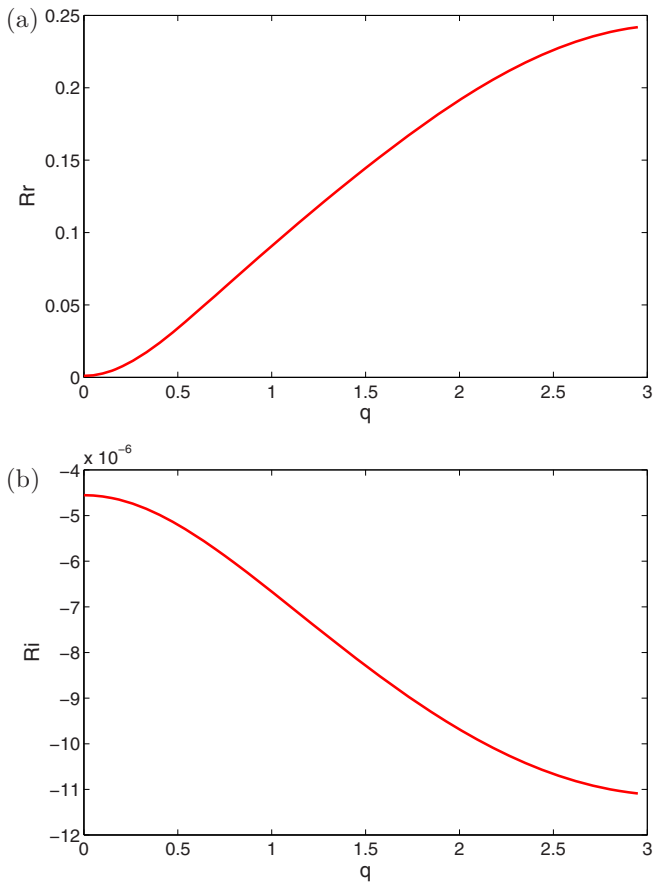

FIG. 3. (Color online) Variations of dissipation coefficients (a) R_r and (b) R_i in terms of the wave vector q of the carrier wave. $D_1 = 0.04$, $\Omega_0^2 = 0.032$, $\lambda_3 = 0.01$, $\gamma_0 = 0.1$, $r = 0.008$.

B. Nonlinear solution of the equation of motion

If we consider the dissipation term to be purely real ($R_i = 0$), that is we neglect the motion of ions and protein anions across slow ions channels, the form of the envelope soliton solution of Eq. (21) will be given by [3,24,42]

$$B(\xi_i,t) = \frac{B_0 e^{\phi}}{1 + e^{(\phi+\phi^*)^{(1+i\alpha)}}}, \tag{29}$$

where

$$\phi = q\xi_i - w\tau, \tag{30}$$

$$\alpha = \beta \pm (2+\beta^2)^{\frac{1}{2}}, \tag{31}$$

$$\beta = \frac{3Q_r}{2Q_i}. \tag{32}$$

After some computations, one can easily find that

$$B = B_r + iB_i, \tag{33}$$

with

$$B_r = B_0 \frac{e^{-\phi} + \cos 2\alpha\phi e^{\phi}}{2(\cosh 2\phi + \cos 2\alpha\phi)}, \tag{34}$$

$$B_i = -B_0 \frac{\sin 2\alpha\phi e^{\phi}}{2(\cosh 2\phi + \cos 2\alpha\phi)}. \tag{35}$$

From Eq. (12), we obtain

$$\varphi = 2(B_r\cos\theta - B_i\sin\theta) + \epsilon[C + 2(D_r\cos 2\theta - D_i\sin 2\theta)] + O(\epsilon^2). \tag{36}$$

D_r and D_i are, respectively, the real and imaginary parts of D.

Now if we suppose that

$$\begin{aligned} D &= (a_1 - ia_2)B^2 \\ &= (a_1 - ia_2)(B_r + iB_i)^2 \\ &= \left[a_1\left(B_r^2 - B_i^2\right) + 2a_2B_rB_i\right] \\ &+ i\left[a_2\left(B_i^2 - B_r^2\right) + 2a_1B_rB_i\right], \end{aligned} \tag{37}$$

where

$$a_1 = \frac{\lambda_1}{3\Omega_o^2 + 16D_0\sin^4\frac{q}{2}}, \tag{38}$$

$$a_2 = \frac{w\gamma_1}{3\Omega_o^2 + 16D_0\sin^4\frac{q}{2}}. \tag{39}$$

Using Eqs. (36) and (37), then inserting the expression $x_i = \epsilon\varphi_i$, we obtain

$$\begin{aligned} x_i = {} & \epsilon B_0\left[\frac{\cos(\theta_i - 2\alpha\phi_i)e^{\phi_i} + \cos\theta_i e^{-\phi_i}}{(\cosh 2\phi_i + \cos 2\alpha\phi_i)}\right] + \epsilon B_0^2\left[-\frac{\lambda_1}{\Omega_0^2(\cosh 2\phi_i + \cos 2\alpha\phi_i)}\right] \\ & + \epsilon^2 B_0^2\left[(a_1\cos 2\theta_i + a_2\sin 2\theta_i)\left(\frac{2\cos 2\alpha\phi_i + \cos 4\alpha\phi_i e^{2\phi_i} + e^{-2\phi_i}}{2(\cosh 2\phi_i + \cos 2\alpha\phi_i)^2}\right)\right] \\ & + \epsilon B_0^2\left[(a_1\sin 2\theta_i - a_2\cos 2\theta_i)\left(\frac{2\sin 2\alpha\phi_i + \sin 4\alpha\phi_i e^{2\phi_i}}{2(\cosh 2\phi_i + \cos 2\alpha\phi_i)^2}\right)\right]. \end{aligned} \tag{40}$$

Figure 4 shows the nerve impulse localized in the neural network having the form of an *asymmetric envelope soliton*.

In Fig. 5 we observe how the nerve impulse evolves in the diffusive network with respect to time. It is clear from there that as time evolves, the form of the asymmetric envelope soliton changes; it is structurally unstable. Therefore, the axonal waves are strongly nonlinear envelope solitons having an up-and-down asymmetry in amplitude. This phenomenon has been observed in a modified Toda lattice model [43]. However, the variation of the parameter ϵ does not affect the form of the wave but its amplitude. From Fig. 6, we notice that the terms resulting from the superposition of harmonics in θ, namely C and D, do affect the wave amplitude. As the perturbation increases so does the amplitude of the nerve

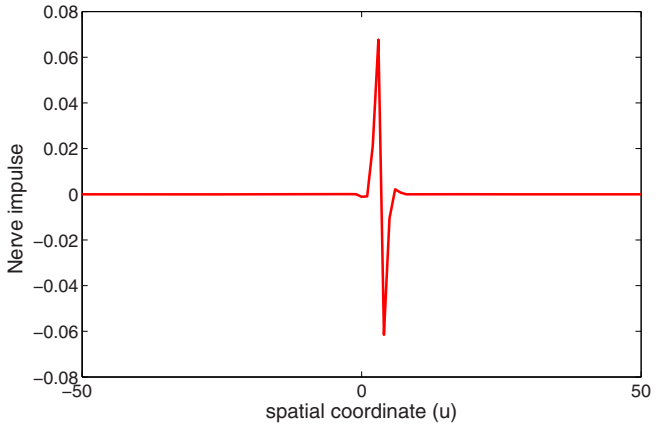

FIG. 4. (Color online) The nerve impulse localized in space. $D_0 = 0.04$, $\Omega_0^2 = 0.032$, $\lambda_1 = 0.01$, $\gamma_1 = 0.001$, $\gamma_2 = 0.15$, $b_0 = 1.0$, $r = 0.008$, $q = 1.5$, $t = 10$, $\epsilon = 0.0001$.

impulse. Therefore, the more random are the ions shared across the plasma membrane the narrower is the width of the action potential and the larger its amplitude is. This phenomenon can be caused by fluctuations in the sequence of neuronal firing times [22,23].

We have also performed numerical simulation of the CGL equation [Eq. (21)] using a Runge-Kutta method with fixed step size. For the initial condition considered as in Eqs. (34) and (35), the profile of the analytical envelope solution is presented in Fig. 7. As expected the solution $|B(\xi,t)|^2$ of the equation is an asymmetric envelope soliton that keeps its shape.

We now suppose that the dissipation term has both its real and imaginary parts ($R_i \neq 0$). In order to find the new form of the solution, we consider the change of variable $M = Be^{i\sigma\tau}$, where M is a solution of the modified CGL equation. Inserting it in Eq. (21) yields

$$\sigma = -\frac{R_i}{2}, \tag{41}$$

thus we obtain

$$M = Be^{-\frac{iR_i}{2}\tau}. \tag{42}$$

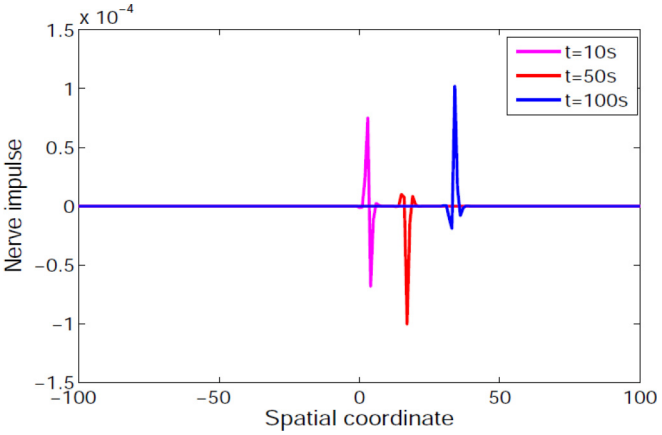

FIG. 5. (Color online) The evolution of the nerve impulse at different times. $D_0 = 0.04$, $\Omega_0^2 = 0.032$, $\lambda_1 = 0.01$, $\gamma_1 = 0.001$, $\gamma_2 = 0.15$, $b_0 = 1.0$, $r = 0.008$, $q = 1.5$.

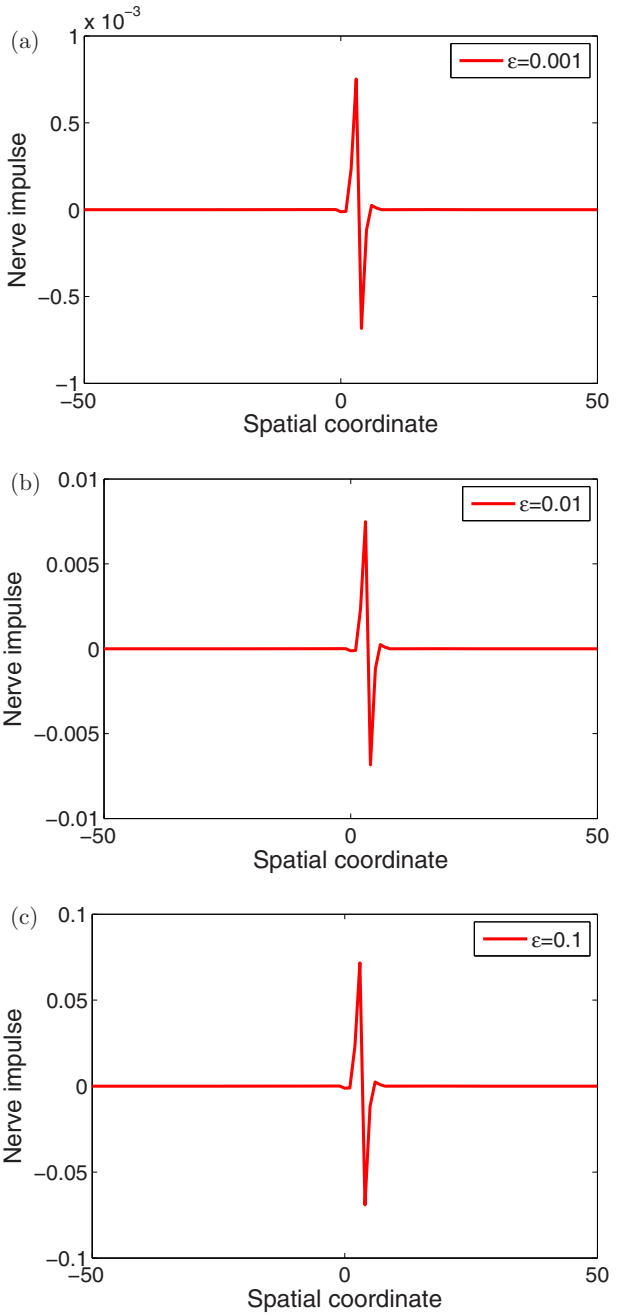

FIG. 6. (Color online) Effects of small perturbations on the nerve impulse. $D_0 = 0.04$, $\Omega_0^2 = 0.032$, $\lambda_1 = 0.01$, $\gamma_1 = 0.001$, $\gamma_2 = 0.15$, $b_0 = 1.0$, $r = 0.008$, $q = 1.5$, $t = 10$. (a) $\epsilon = 0.001$, (b) $\epsilon = 0.01$, (c) $\epsilon = 0.1$.

We observe that the imaginary term of the dissipation acts only on the wave phase. The amplitude of the nerve impulse is not altered.

IV. MODULATIONAL INSTABILITY IN THE NEURAL NETWORK

In the previous section, we saw that the equation of evolution of the membrane potential amplitude admits spatially localized wave solutions; this was under the constraint of PQ_r

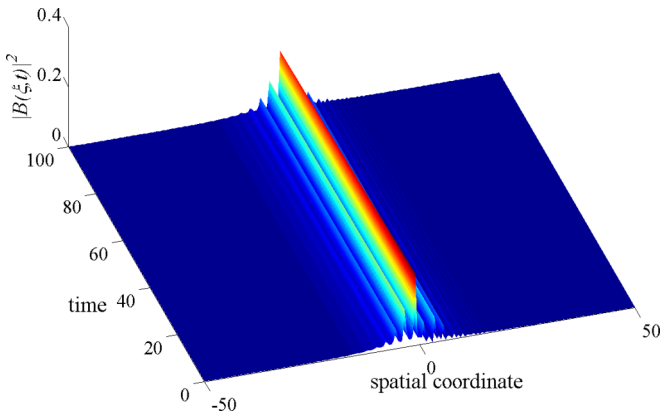

FIG. 7. (Color online) Profile of analytical envelope solution $|B(\xi,t)|^2$ of Eq. (21) with initial conditions Eqs. (34) and (35).

being strictly positive. This situation occurs when the energy tends to localize itself in the system thanks to modulational instability.

Although the modified CGL equation has an envelope soliton as solution, it also admits a plane-wave solution. In this section, we are looking for conditions under which a plane wave propagating in the diffusive neural network will become stable or unstable to a small perturbation. The instability of the plane wave will generate amplitude modulated waves. Hence, we are searching for a plane wave in the form

$$B(\xi_i,t) = B_0 e^{i(\nu\xi_i - \Lambda t)}, \tag{43}$$

where ν is the *wave number* and Λ the wave *angular frequency*.

Reporting Eq. (43) in Eq. (21) gives for the real part

$$\Lambda = \frac{P}{2}\nu^2 + \frac{R_r}{2} - Q_r B_0^2, \tag{44}$$

and the imaginary part

$$Q_i B_0^2 + \frac{R_i}{2} = 0. \tag{45}$$

Equation (44) is the nonlinear dispersion relation of the plane wave; it shows that the angular frequency depends on both the wave number and the wave amplitude.

To examine the linear stability of the plane wave, we look for a solution in the form

$$B(\xi_i,t) = [B_0 + b(\xi_i,t)]e^{i[\nu\xi_i - \Lambda t + \theta(\xi_i,t)]}, \tag{46}$$

where the perturbation amplitude $b(\xi_i,t)$ is supposed to be small in comparison to the plane wave amplitude.

Inserting Eq. (46) into Eq. (21), neglecting the nonlinear terms and considering terms that annihilate, yields to the following equations for real and imaginary parts:

$$-B_0\theta_t + \frac{P}{2}b_{\xi\xi} - PB_0\nu\theta_\xi + 2Q_r B_0^2 b = 0, \tag{47}$$

$$b_t + \frac{P}{2}B_0\theta_{\xi\xi} + P\nu b_\xi - R_i b = 0. \tag{48}$$

The above system of equations admits solutions of the form

$$b = b_o e^{i(\delta\xi - \eta t)} + \text{c.c.}, \tag{49}$$

$$\theta = \theta_o e^{i(\delta\xi - \eta t)} + \text{c.c.}, \tag{50}$$

where δ is an arbitrary real wave number of the perturbation and η is its corresponding propagation frequency that is generally complex.

Substituting these solutions in the system of equations that describe the evolution of the perturbation yields to the following linear homogeneous system for b_o and θ_o:

$$\left(2QrB_0^2 - \frac{P}{2}\delta^2\right)b_o + iB_0(\eta - P\nu\delta)\theta_o = 0, \tag{51}$$

$$(-R_i - i(\eta - P\nu\delta))b_o - \frac{P}{2}B_0\delta^2\theta_o = 0, \tag{52}$$

which can be written in the matrix form as

$$\begin{pmatrix} 2QrB_0^2 - \frac{P}{2}\delta^2 & iB_0(\eta - P\nu\delta) \\ -R_i - i(\eta - P\nu\delta) & -\frac{P}{2}B_0\delta^2 \end{pmatrix}\begin{pmatrix} b_o \\ \theta_o \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}.$$

This system will allow nontrivial solutions if the matrix determinant is equal to zero. The characteristic equation is given by

$$(\eta - P\nu\delta)^2 = \frac{P^2\delta^2}{4}\left(\delta^2 - \frac{4Qr}{P}B_0^2\right) + iR_i(\eta - P\nu\delta). \tag{53}$$

In the above dispersion relation of the perturbation, we notice that the behavior of the angular frequency η depends on the sign of $\frac{Qr}{P}$ for a given value of the wave number δ.

Let $z = \eta - P\nu\delta$, then we have

$$z^2 - iR_i z - \frac{P^2\delta^2}{4}\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right) = 0. \tag{54}$$

The discriminant of this equation is

$$\Delta = P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right) - R_i^2. \tag{55}$$

: − If $\Delta > 0$, i.e., $P^2\delta^2(\delta^2 - \frac{4Q_r}{P}B_0^2) > R_i^2$.

$$z = i\frac{R_i}{2} \pm \frac{\sqrt{R_i^2 - P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right)}}{2}. \tag{56}$$

The imaginary part of z which is the imaginary part of η is given by $\frac{R_i}{2}$. In this region, the wave oscillates about its original value and after a certain period of time the perturbations will extinguish [this is because $R_i < 0$ see Fig. 3(b)]. The plane wave is therefore *stable*.

: − If $\Delta = 0$, i.e., $P^2\delta^2(\delta^2 - \frac{4Q_r}{P}B_0^2) = R_i^2$, $z = i\frac{R_i}{2}$.

The imaginary part of η is still given by $\frac{R_i}{2}$. Thus, as seen above, the plane wave remains *stable*.

: − If $\Delta < 0$, i.e., $P^2\delta^2(\delta^2 - \frac{4Q_r}{P}B_0^2) < R_i^2$,

$$z = i\frac{R_i}{2} \pm i\frac{\sqrt{R_i^2 - P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right)}}{2}. \tag{57}$$

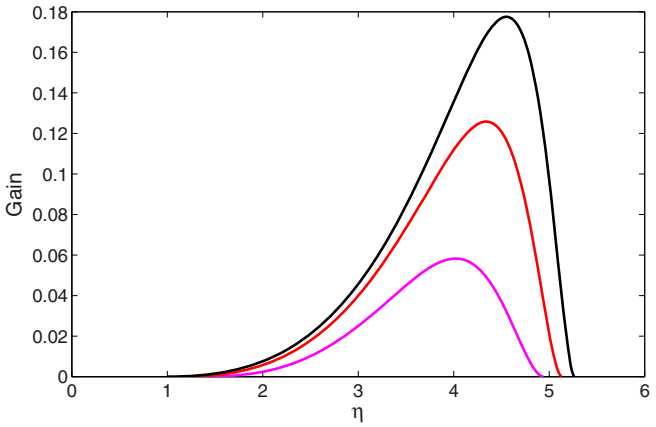

FIG. 8. (Color online) Instability growth rate according to Eq. (2.56) for three values of the diffusive coupling strentgh $D_0 = 0.01$ (magenta line), $D_0 = 0.03$ (red line), $D_0 = 0.06$ (black line). $b_0 = 0.04$.

Let's discuss the conditions for which the plane wave is stable, which is the same as discussing the sign of the imaginary part of z.

: $-$ If $Imz < 0$, i.e., $R_i < \sqrt{R_i^2 - P^2\delta^2(\delta^2 - \frac{4Q_r}{P}B_0^2)}$
$\Rightarrow P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right) > 0.$

This is true if $\frac{Q_r}{P} < 0$ (i.e., $PQ_r < 0$). The plane wave solution of the modified CGL equation is therefore *stable* when $PQ_r < 0$. The perturbations b and θ merely oscillate at a constant amplitude without damping.

: $-$ If $Imz > 0$, i.e., $R_i > \sqrt{R_i^2 - P^2\delta^2(\delta^2 - \frac{4Q_r}{P}B_0^2)}$
$\Rightarrow P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right) < 0.$

Here, $\frac{Q_r}{P} > 0$ (i.e., $PQ_r > 0$). The perturbations grow exponentially with time resulting in the plane wave being *unstable*. The *local growth rate of the modulational instability* or the *gain* is then given by

$$g = |Im\eta| = \frac{1}{2}\left[R_i + \sqrt{R_i^2 - P^2\delta^2\left(\delta^2 - \frac{4Q_r}{P}B_0^2\right)}\right]. \quad (58)$$

When the gain is maximal, $g = \frac{1}{2}[R_i + \sqrt{R_i^2 + 3Q_r^2B_0^2}]$, the plane wave tends to modulate itself with the corresponding wave number $\delta = B_0\sqrt{\frac{Q_r}{P}}$ (see Fig. 8). We observe that the growth rate of the modulational instability depends on the amplitude of the plane wave, the nonlinear and dissipative coefficients of the modified CGL equation. Whereas, in the case of the nonlinear Schrödinger equation (NLS), the gain is exclusively dependent of the wave amplitude and the nonlinear coefficient of the NLS equation [2]. We equally notice that if $R_i = 0$, we obtain the same expression for the gain established by Peyrard and Dauxois for a NLS equation [2].

In the domain of existence of solitons, the modulated phase of the wave (as observed during the linear stability analysis of the small perturbations) precedes a phase where the wave amplitude will vanish in particular areas of the network. This is the generation of envelope solitons. This analysis corroborates the fact that solitons exist unrestrainedly in nonlinear, dissipative, and dispersive medium.

V. CONCLUSION

The main objective of this work was to study localized nonlinear excitations in diffusive Hindmarsh-Rose neural networks. In order to carry out that purpose, we have shared our work into three sections. In the first section, we reduced the diffusive coupled Hindmarsh-Rose model system formed by three nonlinear ordinary differential equations to two differential equations with the equation governing the motion of the transmembrane voltage being in a Liénard form. The second section was dedicated to find envelope solitons in the diffusive Hindmarsh-Rose neural model. The necessity to find in our model nonlinear excitations of weak amplitude led us to the use of the multiple scale expansion in the semidiscrete approximation. At the first and second order of approximation, we obtain that both the angular frequency and the group velocity of the action potential depend on characteristic features of the neural tissue. At the third order of approximation, we obtain from the membrane potential equation of motion, an equation describing the evolution of modulated waves, a modified complex Ginzburg-Landau equation. We therefore made numerical simulations of the envelope solitons solutions of that modified CGL equation by using the solution form proposed by Nozaki and Bekki. It is described by asymmetric envelope solitons unstable in their dynamics down the axon, however, keeping the asymmetric envelope soliton shape in the presence of disturbances. A study of the modulational instability of a plane wave propagating in the network was also made in Sec. IV. Via the linear stability analysis of the plane wave, we verify as one could expect that the expression of the growth rate of the modulational instability of the modified CGL equation reduces to the one of a nonlinear Schrödinger equation if the dissipation term is neglected. The Benjamin-Feir instability was checked to be true in our neural network.

These results have several interesting aspects. In the first place, they describe the way of obtaining the type of localized structures that propagate in polynomial form of equation of neurons using fairly standard perturbation technique. Second, bursting in a neuronal system is a recurrent alternation between active phases (large amplitude oscillations) and quiescent phases (small amplitude oscillations). Studying the properties of regularly and irregularly oscillating localized structures may be fruitful for fundamental insights into spatiotemporal dynamics and chaos and possible interest for neuronal population information encoding and transmission where several neurons fire within a population.

ACKNOWLEDGMENT

This work was supported by the Academy of Sciences for the Developing World (TWAS), under the Research Grant Agreement (RGA) No. 10-071 RG/PHYS/AF/AC-G-UNESCO FR 3240245983.

[1] M. Remoissenet, *Waves Called Solitons Concepts and Experiments, 3rd ed.* (Springer, New York, 2003).

[2] T. Dauxois and M. Peyrard, *Physics of Solitons* (Cambridge University Press, Cambridge, 2006).

[3] C. Tchawoua, Ph.D. thesis, Université de Yaoundé I, 2005.

[4] *Dissipative Solitons*, edited by N. Akhmediev and A. Ankiewicz (Springer, New York, 2005).

[5] R. H. Enns, *It's a Nonlinear World* (Springer, New York, 2011).

[6] M. Brambilla, L. A. Lugiato, F. Prati, L. Spinelli, and W. J. Firth, Phys. Rev. Lett. **79**, 2042 (1997).

[7] P. L. Ramazza, E. Benkler, U. Bortolozzo, S. Boccaletti, S. Ducci, and F. T. Arecchi, Phys. Rev. E **65**, 066204 (2002).

[8] B. Schäpers, M. Feldmann, T. Ackemann, and W. Lange, Phys. Rev. Lett. **85**, 748 (2000).

[9] P. V. Paulau, A. J. Scroggie, A. Naumenko, T. Ackemann, N. A. Loiko, and W. J. Firth, Phys Rev. E **75**, 056208 (2007).

[10] J. A. Villacota-Atienza, M. G. Velarde, and V. A. Makarov, Biol. Cybern. **103**, 285 (2010).

[11] J. A. Villacota-Atienza and V. A. Makarov, PLoS ONE **8**, e57440 (2013).

[12] M. Argentina, P. Coullet, and V. Krinsky, J. Theor. Biol. **205**, 47 (2000).

[13] E. A. Ermakova *et al.*, PLos ONE **4**, e4454 (2009).

[14] S. Brunak and B. Lautrup, *Neural Networks* (World Scientific Publishing, Singapore, 1990).

[15] R. A. Stefanescu and V. K. Jirsa, PLoS Comput. Biol. **4**, e1000219 (2008).

[16] A. M. Dikandé and G. A. Bartholomew, Phys. Rev. E **80**, 041904 (2009).

[17] A. Destexhe, A. Babloyantz, and T. J. Sejnowski, Biophys. J. **65**, 1538 (1993).

[18] W. R. Lieb and W. D. Stein, *Transport and Diffusion Across Cell Membranes* (Academic Press, San Diego, 1986).

[19] S. Raghavachari and J. A. Glazier, Phys. Rev. Lett. **82**, 2991 (1999).

[20] Z. P. Kilpatrick and P. C. Bressloff, Physica D **239**, 547 (2010).

[21] D. D. Clarke and L. Sokoloff, in *Basic Neurochemistry: Molecular, Cellular and Medical Aspects*, edited by G. J. Siegel *et al.* (Lippincott-Raven, Philadelphia, 1999).

[22] S. E. Folias and P. Bressloff, SIAM J. Appl. Math. **65**, 2067 (2005).

[23] S. E. Folias and P. Bressloff, SIAM J. Appl. Dynam. Syst. **3**, 378 (2004).

[24] K. Nozaki and N. Bekki, J. Phys. Soc. Jpn. **53**, 1581 (1984).

[25] A. Kenfack-Jiotsa and E. Tala-Tebue, J. Phys. Soc. Japan **80**, 034003 (2011).

[26] Y. S. Kivshar and M. Peyrard, Phys. Rev. A **46**, 3198 (1992).

[27] C. B. Tabi, A. Mohamadou, and T. C. Kofane, Eur. Phys. J. B. **74**, 151 (2010).

[28] A. L. Hodgkin and A. F. Huxley, J. Physiol. (London) **117**, 500 (1952).

[29] R. FitzHugh, Biophys. J. **1**, 445 (1961).

[30] C. Morris and H. Lecar, Biophys. J. **35**, 193 (1981).

[31] J. L. Hindmarsh and R. M. Rose, Nature (London) **296**, 162 (1982).

[32] J. L. Hindmarsh and R. M. Rose, Proc. R. Soc. London, Ser. B **221**, 87 (1984)

[33] M. Perez-Armandriz, M. C. Roy, D. C. Spray, and M. V. L. Bennet, Biophys. J. **59**, 76 (1991).

[34] D. Tatchim Bemmo, M. Siewe Siewe, C. Tchawoua, Phys. Lett. A **375**, 1944 (2011).

[35] N. Dror and B. A. Malomed, Phys. Rev. E **79**, 016605 (2009).

[36] Y. S. Kivshar and B. A. Malomed, Rev. Mod. Phys. **61**, 763 (1989).

[37] S. Jiménez and V. V. Konotop, Phys. Rev. B **60**, 6465 (1999).

[38] C. D. Negro, C.-F. Hsiao, S. Chandler, and A. Garfinkel, BioPhys. J. **75**, 174 (1998).

[39] C. M. Pedroarena, I. E. Pose, J. Yamuy, M. H. Chase, and F. R. Morales, J. Neurophysiology **82**, 1465 (1999).

[40] A. K. Al Azad and P. Ashwin, SIAM J. Appl. Dyn. Syst. **9**, 261 (2010).

[41] J. Y. Wu, X. Huang, and C. Zhang, Neuroscientist **14**, 487 (2008).

[42] N. R. Pereira and L. Stenflo, Phys. Fluids **20**, 1733 (1977).

[43] Daisuke Yagi and Takuji Kawahara, Wave Motion **34**, 97 (2001).