Breathing pulses in the damped-soliton model for nerves

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(Received 17 August 2017; published 17 January 2018)

Unlike the Hodgkin-Huxley picture in which the nerve impulse results from ion exchanges across the cell membrane through ion-gate channels, in the so-called soliton model the impulse is seen as an electromechanical process related to thermodynamical phenomena accompanying the generation of the action potential. In this work, account is taken of the effects of damping on the nerve impulse propagation, within the framework of the soliton model. Applying the reductive perturbation expansion on the resulting KdV-Burgers equation, a damped nonlinear Schrödinger equation is derived and shown to admit breathing-type solitary wave solutions. Under specific constraints, these breathing pulse solitons become self-trapped structures in which the damping is balanced by nonlinearity such that the pulse amplitude remains unchanged even in the presence of damping.

DOI: 10.1103/PhysRevE.97.012211

I. INTRODUCTION

The action potential is most commonly described as a propagating form of the voltage difference across the nerve membrane [1–11]. This voltage difference traveling along the nerve axon as an electrical pulse, originates from unbalanced distributions of negative and positive ions on either side of the membrane. In past years a lot of effort has been devoted to understanding the mechanism underlying the generation and propagation of the action potential. Exploiting Bernstein's pioneer idea [5] of a key role held by membrane's permeability in ions flow across the nerve cell membrane, Hodgkin and Huxley suggested that the membrane contains proteins that selectively conduct sodium and potassium ions [1]. In the so-called Hodgkin-Huxley model [1–3] the nerve axon is treated as an electrical circuit in which proteins are represented as resistors, while the membrane is treated as a capacitor. In this picture, ion currents flowing through the membrane create a voltage pulse which propagates along the nerve axon.

The main characteristic feature of the Hodgkin-Huxley model is the propagation equation for the action potential, which is derived using Kirchhoff's laws and thus depends only on electrical parameters. Although the electrical-circuit representation describes rather satisfactorily several aspects of the action potential related to the electrical activities of the nerve, it has been unable to provide answers to mechanical and thermal responses accompanying the generation of the action potential. In fact, the mechanical and heat signatures in the generation of the action potential suggest that the nerve pulse should be an adiabatic and reversible phenomenon, reminiscent of a propagating mechanical wave.

The soliton model of the nerve impulse was proposed as an alternative to the electrical model, in which the action potential is described as an electromechanical process for which both mechanical and thermodynamical phenomena contribute. In this model the nerve impulse is associated with the change in the nerve membrane density and thus is a density pulse propagating along the membrane. Following its proposal, a great deal of theoretical interest has been devoted to the model; in particular, distinct types of solitonlike structures associated with the density change have been investigated. Namely, Heimburg and Jackson [12–14] obtained numerically bright soliton solutions to the model, while Contreras *et al.* [15] obtained analytically two forms of solutions for the density equation. In this last work the authors established that in the gel state of the nerve, the soliton model admits bright solitons, whereas in the liquid states they admit dark solitons [15]. Vargas *et al.* [16] observed that the dark and bright solitons previously proposed do not explain some important experimental features such as the hyperpolarization, the refractory periods, and stable periodic wave trains. Instead they investigated periodic wave trains under the assumption that the nerve density, as well as the overall length of the nerve, remain unchanged as the impulse propagates. As pointed out by Vargas *et al.* [16], such assumption is inconsistent in real nerves where the law of mass (and hence density) conservation does not hold, and the length also is not conserved because of the stretching and compression of the muscles attached to the nerve, which can drastically affect profiles of the propagating wave trains. The inconsistency of this assumption actually puts into evidence the need for a better description of the generation of soliton wave trains in the nerve, one possible route being the phenomenon of self-modulational instability. The phenomenon of self-modulational instability was proposed for the soliton model by Larios *et al.* [17], within the framework of the bifurcation analysis. On the other hand, studies of soliton collisions [18] in biomembranes and nerve have shown that an initial large-amplitude pulse introduced in the nerve will decay into a series of soliton pulses. These soliton pulses spread out, displaying an undershoot, hence suggesting that the modulational-instability phenomenon might be responsible for the formation of periodic pulse trains observed during nerve experiments, including the undershoot and refractory periods.

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More recently, theoretical studies have predicted that the propagating solitons contain two modes, namely, a transverse mode and a longitudinal mode. The transverse mode is characterized by the swelling and relaxation of the biomembrane and has a beating or symmetric wave profile, while the longitudinal mode is of a dark or bright-shape soliton type [19,20]. However, most works have focused only on the longitudinal solution of the Heimburg model of nerve. Also the physical context of a thick axon nerve, and of a myelinated nerve having a high conduction speed compared to a thin nerve as evidenced by the ions channel model, has not been considered within the framework of the Heimburg model. Last, the phenomenon of nerve signal blockage too has not been considered within the framework of the Heimburg model.

In this work, we address the above issues by considering the Heimburg nerve model with friction. We obtain analytical expressions for the breathing soliton modes, and show that the modulational-instability phenomenon in the nerve is associated with the changes in viscosity and might be responsible for stable periodic wave trains observed during experiments. The obtained damped breathing soliton is relevant for it encompasses all the mechanical changes occurring during the propagation of the action potential, including the nerve signal blockage and the high conduction speed due to changes in the thickness (viscosity) of the nerve. To obtain the breathing solution we shall use the method of multiple-scale expansion, combined with the reductive perturbation [21,22], which will enable us to obtain the evolution equation for the nerve impulse.

II. MODEL AND DAMPED NONLINEAR SCHRÖDINGER EQUATION

The soliton model describes mechanical processes that occur in biomembranes. The model is based on the assumption that the nerve axon, seen as a cylindrical biomembrane, undergoes a phase transition from fluid to gel states at a temperature slightly below the body temperature. Heimburg and Jackson [12] observed that close to the melting transition, the speed of the propagating density pulse depends both on the membrane density and the propagation frequency. From this observation they suggested that the presence of nonlinearity, characterized by a density dependence on the speed of the density pulse, and of the dispersion resulting from the frequency dependence on density pulse, would introduce the possibility for localized excitations with soliton features. The phase transition can be initiated either by the propagation of action potential along the axoplasm [19], by a local cooling, or by mechanical perturbations [12]. They proposed that the density change responsible for the nerve impulse, as well as the associated mechanical responses, could be described by the following equation [12]:

$$\frac{\partial^2\Delta\rho^A}{\partial t^2}=\frac{\partial}{\partial x}\left(\left[c_0^2+\alpha\Delta\rho^A+\beta(\Delta\rho^A)^2\right]\frac{\partial\Delta\rho^A}{\partial x}\right)+\nu\frac{\partial^2}{\partial x^2}\left(\frac{\partial\Delta\rho^A}{\partial t}\right)-h\frac{\partial^4\Delta\rho^A}{\partial x^4}. \tag{1}$$

Explicitly, Eq. (1) is a Heimburg model with a damping term added to the system. It describes the propagation of an area density pulse $\Delta\rho^A$ through the nerve axon after taking into account the effect of damping. The equation assumes a propagation of the nerve impulse by compression and rarefaction of lipid molecules through a nerve axon, where x is the impulse position at time t. h and ν are parameters that measure the dispersion and friction of the nerve axon, respectively,; $\Delta\rho^A=\rho^A-\rho_0^A$ is the change in area density of the nerve axon between the gel-state density (ρ^A) and the fluid-state density (ρ_0^A); K_s^A accounts for a lateral compressibility of the axon while $c_0^2=\frac{1}{K_s^A\rho_0^A}$, $\alpha=-\frac{1}{K_s^A(\rho_0^A)^2}$, and $\beta=\frac{1}{K_s^A(\rho_0^A)^3}$.

While in most previous works the effect of friction has not been taken into consideration, in real membranes the movement of the muscle attached to the nerve can cause nerve compression, nerve stretching, and nerve friction, all of which affect the nerve impulse profile. Also the nerve membrane is not homogeneous, thus the composition of lipids and protein should vary and therefore the elastic constant of the nerve is expected to vary locally [12–14]. Since we consider the membrane as a viscous elastic fluid, the aspect of internal frictions due to vibrations of fluid particles cannot be neglected. The interaction between systems usually introduces the phenomena irreversibility and dissipation. There are numerous approaches in the literature that take into account these interactions [23–26]. The generalization of irreversible processes based uniquely and without additional assumptions or approximations on the nonlinear equation of motion, recently proposed by Baretta as the dynamical principle of quantum thermodynamics, as well as their possible adaptation to physical and nonphysical problems, can be found in Refs. [24,25]. Note also that in [23], the Heisenberg-Langevin equation was used to derive a Schrödinger equation for a Brownian particle interacting with a thermal environment. In the following, we apply the methods of reductive perturbation and multiple-scale expansion on Eq. (1), to obtain a damped nonlinear Schrödinger equation assumed to describe the dynamics of the amplitude of envelope solitons propagating within the nerve in the presence of friction. Consider the dimensionless variables u, z, and τ, defined as

$$u=\frac{\Delta\rho^A}{\rho_0^A},\quad z=\frac{c_0x}{\sqrt{h}},\quad \tau=\frac{c_0^2t}{\sqrt{h}}. \tag{2}$$

With these new variables, we obtain the following dimensionless density-wave equation:

$$\frac{\partial^2u}{\partial\tau^2}=\frac{\partial}{\partial z}[(1+pu+qu^2)u_z]-\frac{\partial^4u}{\partial z^4}+\mu\frac{\partial^3u}{\partial z^2\partial\tau}, \tag{3}$$

where

$$\mu=\frac{\nu}{\sqrt{h}},\quad q=\frac{\left(\rho_0^A\right)^2}{c_0^2}\beta,\quad p=\frac{\rho_0^A}{c_0^2}\alpha.$$

As we are interested in weakly nonlinear and weakly dissipative solution to Eq. (3), we apply the method of reductive perturbation. In this goal we introduce a small parameter ϵ ($\epsilon\ll1$), and proceed with the substitutions $p\to\epsilon^2p$, $q\to\epsilon^2q$, $\mu\to\epsilon\mu$, into (3). This leads to the following nonlinear damped equation for the nerve impulse in a one-dimensional cylindrical axon

$$\frac{\partial^2u}{\partial\tau^2}=\frac{\partial}{\partial z}[(1+\epsilon^2pu+\epsilon^2qu^2)u_z]-\frac{\partial^4u}{\partial z^4}+\epsilon\mu\frac{\partial^3u}{\partial z^2\partial\tau}. \tag{4}$$

Next consider the variable changes

$$y = \epsilon(z - \tau), \quad s = \epsilon^3 \tau, \tag{5}$$

where ϵ and ϵ^3 are chosen in such a way to balance the effects of nonlinearity and damping. Using this transformation, terms of order ϵ^4 give

$$\frac{\partial u}{\partial s} + \frac{1}{2}(apu + a^2 q u^2)u_y - \frac{1}{2}\frac{\partial^3 u}{\partial y^3} = a^2 \frac{\mu}{2}\frac{\partial^2 u}{\partial y^2}, \tag{6}$$

which is the Burgers-Korteweg–de Vries (BKdV) equation. Note that in Eq. (6) we proceeded with the substitutions $u \to au$ and $\mu \to a^2\mu$, after setting terms of order ϵ^4 to zero. It is also important to note here that this transformation did not fundamentally affect the structure of the system. Actually the choice of the order of perturbation a is such that nonlinearity and damping are not balanced in the BKdV equation (6).

In the present work, we are looking for solitons made up of carrier waves modulated by envelope signal, which is called breathing pulses or solitons. This type of soliton appears naturally for most weakly nonlinear systems which are described by a wave equation in the small amplitude limit. Since we are studying low amplitude nonlinear excitations in a weakly dissipative soliton model, it is adequate to use a multiple-scale expansion method. The multiple-scale expansion is a perturbation technique in which both the carrier waves and the amplitude are treated in the continuum limit [21,22]. It is thus incumbent on us to use this technique to obtain the evolution equation of the BKdV equation, when nonlinearity and damping are now balanced. The method involves introducing two time and spatial scales, i.e., the fast time and spatial scales for the oscillations and the slow time and spatial scales for the envelope amplitude. This method has been used to derive the nonlinear Schrödinger equation from the KdV equation in Ref. [21]. Proceeding with calculations, we introduce new time scale $s_i = a^i s$ and space scale $y_i = a^i y$, and assume a solution of the form

$$u(y,s) = \sum_{i=0}^{\infty} a^i u(s_0, s_1, s_2, y_0, y_1), \tag{7}$$

where each value of s_i and y_i is treated as an independent variable. This leads to a perturbation series of operators from all independent variables

$$\frac{\partial}{\partial s} = \frac{\partial}{\partial s_0} + a\frac{\partial}{\partial s_1} + a^2\frac{\partial}{\partial s_2}. \tag{8}$$

An important feature of the multiple-scale method is the fact that the solution of the original problem will only be obtained, if a multidimensional space is generated by the new sets of variables of s_i and y_i that come from the physical line

$$s_0 = s, \quad s_1 = as, \quad s_2 = a^2 s,$$

and

$$y_0 = y, \quad y_1 = ay, \quad y_2 = a^2 y.$$

Proceeding similarly, we obtain the operator for the spatial scale

$$\frac{\partial}{\partial y} = \frac{\partial}{\partial y_0} + a\frac{\partial}{\partial y_1}. \tag{9}$$

Next we replace the above operators into the different terms of the BKdV equation, and then group terms of the same order of a to obtain a system of equations. Each of these equations will correspond to each approximation having harmonics of a specific order. But one of the major difficulties of this method lies in the choice of the ansatz used, since the ansatz varies with the model. Thus one usually guesses the appropriate form of the ansatz for a given model; in our specific case we pick the following operators:

$$\frac{\partial^2}{\partial y^2} = \frac{\partial^2}{\partial y_0^2} + 2a\frac{\partial^2}{\partial y_0 \partial y_1} + a^2\frac{\partial^2}{\partial y_1^2}. \tag{10}$$

According to the multiple-scale expansion method, the ansatz for the solution u must be consistent with the series expansion of differential operators in powers of the small parameter a [21,22] adopted in (10). Let us therefore write u as a perturbative series, and consider only terms to the first order in a, i.e.,

$$u = Ae^{i\theta} + A^* e^{-i\theta} + a(C + Be^{2i\theta} + B^* e^{-2i\theta}), \tag{11}$$

where $\theta = (ky_0 - \omega s_0)$ and the amplitudes A, B, and C correspond, respectively, to (s_1, y_1, y_2). We then substitute equations (8), (9), (10), and (11) into (6), and look for relations between terms of same orders in a with terms in $e^{\pm i\theta}$, $e^{\pm 2i\theta}$ without an exponential dependence set to zero. To the order a^0, the annihilation of terms in $e^{\pm i\theta}$ gives the dispersion relation of linear waves, i.e.,

$$\omega = \frac{k^3}{2}. \tag{12}$$

To the order a^1, the cancellation of terms in $e^{\pm i\theta}$ gives

$$\frac{\partial A}{\partial s_1} + v_g \frac{\partial A}{\partial y_1} = 0, \tag{13}$$

where v_g is the group velocity defined as

$$v_g = \frac{\partial \omega}{\partial k} = \frac{3k^2}{2}. \tag{14}$$

Setting terms in $e^{\pm 2i\theta}$ to zero gives

$$B = -\frac{pk}{6k^3}A^2. \tag{15}$$

To the second order in the perturbation, terms with zero exponential dependence yield

$$\frac{\partial C}{\partial s_1} - \frac{p}{2}\frac{\partial |A|^2}{\partial y_1} = 0. \tag{16}$$

Now consider the transformation $y = (y_1 - v_g s_1)$ and $\tau = s_1$, and comparing the result with (13) we obtain the relation

$$C = \frac{p}{3k^2}|A|^2. \tag{17}$$

To the second order in the perturbation, terms of order $e^{\pm i\theta}$ give the damped nonlinear Schrödinger equation

$$i\frac{\partial A}{\partial s_2} + P\frac{\partial^2 A}{\partial y_1^2} + Q|A|^2 A + iRA = 0, \tag{18}$$

which describes the evolution of the envelope amplitude of the density pulse u in the one-dimensional cylindrical nerve axon.

Here the nonlinearity or (self-trapping), the damping and the dispersive coefficients Q, R, and P, respectively, are real and are defined in terms of membrane parameters as

$$Q = \left(\frac{\rho_0^A}{2c_0}\right)^2\left(\frac{\alpha^2}{3c_0^2k^2} - k\beta\right), \quad P = \frac{3k}{2}, \quad R = \frac{\nu k^2}{2\sqrt{h}}. \tag{19}$$

The imaginary term in the nonlinear Schrödinger equation causes the damping of the amplitude and shows the irreversibility of the time evolution but not any effect of dissipation of energy. A similar Schrödinger equation with an imaginary (even nonlinear) added term was proposed by Beretta [25], introducing irreversible time evolution toward a state of maximum entropy, but without dissipation of energy.

Remark that in Ref. [9], it was shown that the evolution of nerve signal in the Hindmash-Rose model can be described by a similar equation but with complex coefficients then referred to as complex Ginzburg-Landau equation. Electrical solitons in dendrites and axons rely not on nonlinear inductance or capacitance, but on an active membrane. This result suggested that the brain may actively work not only in time domain but also effectively use the spatial dimension for information processing. In the present case, the damped nonlinear Schrödinger equation obtained from the Heimburg soliton model in neuroscience, clearly indicates that information encoding and transmission in the form of an electromechanical wave traveling along the axon can also emerge as modulated structures, and not only in the form of nerve pulse as observed in Refs. [12–18]. A biophysical model for the mechanical action waves that accompanies action potential with a similar profile was also proposed by Hady and Machta [20].

III. LOCALIZED PERIODIC WAVE TRAINS IN THE NERVE: MODULATIONAL INSTABILITY

Before seeking for soliton solutions to the damped nonlinear Schrödinger equation (18), it is useful to first examine the stability of plane waves in the system. With this goal in mind, we carry out a modulational-instability analysis [9,27] of small-amplitude impulse signals propagating in the dissipative nerve. As shown by Benjamin and Feir [28] in the context of fluids dynamics, such instability can be a precursor of localized periodic wave trains in systems exhibiting weak nonlinearity.

The plane-wave solution to the damped nonlinear Schrödinger (18) can be written in the general form

$$A = A_0 e^{i(\xi y_1 - \varphi s_2)} e^{-Rs_2}, \tag{20}$$

where ξ is the wave number, φ is the frequency, and A_0 is the amplitude of the plane wave. The dispersion relation associated with the plane wave (20) is given by

$$\varphi = P\xi^2 - QA_0^2 e^{-Rs_2}. \tag{21}$$

Now consider small perturbations $a_1(y_1,s_2)$ and $\phi(y_1,s_2)$ on the amplitude and phase, respectively, of the plane wave such that the solution (20) can be rewritten

$$A(y_2,s_1) = [A_0 + a_1(s_2,y_1)]e^{i(\xi y_1 - \varphi s_2) + \phi(s_2,y_1)} e^{-Rs_2}. \tag{22}$$

Replacing (22) in the damped nonlinear Schrödinger equation (18), and retaining only linear terms in the perturbations, a separation of real part from imaginary part leads to the following two coupled linear differential equations:

$$\frac{\partial a_1}{\partial s_2} + 2\xi P\frac{\partial a_1}{\partial y_1} + PA_0\frac{\partial^2\phi}{\partial y_1^2} = 0 \tag{23}$$

and

$$-A_0\frac{\partial\phi}{\partial s_2} + P\frac{\partial^2 a_1}{\partial y_1^2} - 2\xi A_0 P\frac{\partial\phi}{\partial y_1} + \left(2QA_0^2 - \xi^2 + \varphi\right)a_1 = 0. \tag{24}$$

Suppose the solutions to the system (23) and (24) are

$$a_1 = a_0 e^{i(Ky_1 - \Omega s_2)} \tag{25}$$

and

$$\phi = \phi_0 e^{i(Ky_1 - \Omega s_2)}, \tag{26}$$

where a_0 and ϕ_0 are constants, and K and Ω are the modulation wave vector and frequency, respectively. Substituting (25) and (26) into (23) and (24) gives rise to

$$i(-\Omega + 2PK\xi)a_0 - 2PA_0K^2\phi_0 = 0, \tag{27}$$

$$\left(-PK^2 + 2QA_0^2 - \xi^2 P + \varphi\right)a_0 + iA_0(\Omega - 2P\xi K)\phi_0 = 0. \tag{28}$$

Equations (27) and (28) can be represented as a 2×2 matrix, for which the two possible eigenvalues are two distinct dispersion relations for the perturbations, i.e.,

$$\Omega(K) = 2P\xi K \pm \sqrt{P^2K^4 + QA_0^2K^2P(e^{-Rs_2} - 2)}. \tag{29}$$

For the plane wave to be modulationally unstable the modulation frequency Ω must be a complex function. We define the modulation gain as $G = 2\mathrm{Im}[\Omega(K)]$ [29]; it follows then that a plane wave will be unstable if

$$P^2K^4 + QA_0^2K^2P(e^{-Rs_2} - 2) < 0. \tag{30}$$

For the ideal Heimburg model, where there is no damping, i.e., $R = 0$, the condition (30) reduces to

$$\frac{Q}{P} > \frac{K^2}{A_0^2}. \tag{31}$$

For very large values of A_0 the relation (31) reduces to

$$PQ > 0, \tag{32}$$

which is the Benjamin-Feir instability criteria for the nonlinear Schrödinger equation, or the ideal Heimburg model [9,21]. When this condition is fulfilled, the amplitude of perturbation grows exponentially and plane waves are therefore unstable. In Fig. 1, we plotted the modulation gain G as a function of the modulation wave number K for different values of the viscosity ν [Fig. 1(a)], and as a function of both the modulation wave number and the viscosity [Fig. 1(b)].

We observe that changes in the nerve viscosity affect the modulation gain in the system; in fact, G is seen to grow with an increase in the viscosity meaning that the viscosity increases the modulational instability in the nerve. As a consequence the plane-waves solution to the damped nonlinear Shrödinger equation become unstable, and will eventually break up into

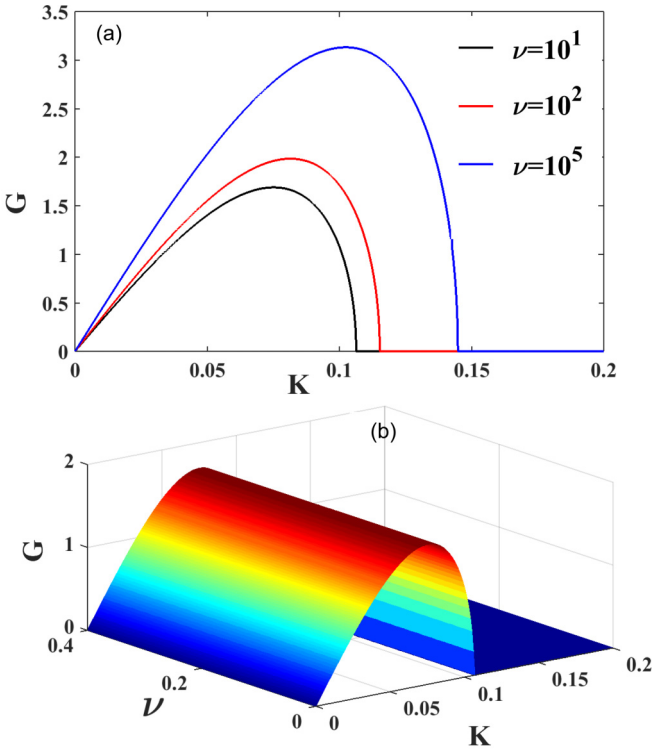

FIG. 1. (a) Gain spectrum for different values of viscosity. (b) Three-dimensional representation of the gain. Parameters chosen are $\xi = 40$ and $a = 0.0002$, $A_0 = 0.2$, and $t = 10^{-4}$.

filaments of stable periodic wave trains. Note that the stability of solitons in the presence of dissipations in the Heimburg-Jackson model has been studied numerically [18]. It was shown that large-amplitude solitons decay into a series of several pulses in the presence of damping effects [18]. This result has been obtained from a generalization of the Boussinesq equation, proposed to be relevant for pulse propagation in biomembranes and nerves. They found that solitons retain their characteristic properties even in the presence of relatively strong damping. On the contrary, for low-amplitude soliton solutions obtained after reducing the Boussinesq equation into the nonlinear Schrödinger equation, the presence of damping increases the amplitude of the perturbation growth rate.

IV. SOLITON SOLUTIONS TO THE ENVELOPE EQUATION

A. General soliton solutions

We now turn to large-amplitude nerve impulse signals. In general, the damped nonlinear Schrödinger equation (18) admits several distinct types of nonlinear traveling-wave solutions, including dark and bright soliton solutions. These solutions depend on the sign of coefficients P and Q, which are functions of k and of the system parameters α, β, and h. When $PQ > 0$, Eq. (18) admits an envelope soliton solution which has a vanishing amplitude as $|y_1| \to \infty$, and corresponds to a small-amplitude breathing pulse. However, if $PQ < 0$, a dark (envelope hole) soliton will propagate with finite amplitude as $|y_1| \to \infty$. In this section, we shall be interested in a solitary-wave solution to Eq. (18), obtained under the condition $PQ > 0$.

It is well known [21] that when $PQ > 0$, the nonlinear Schrödinger equation (18) without the damping term admits a modulated pulse-shaped solitary-wave solution of the form

$$A(y_1,s_2) = A_0\,\mathrm{sech}[B(y_1 - u_e s_2)]e^{-i(k_0 y_1 - \omega s_2)}, \tag{33}$$

where the pulse amplitude A_0, inverse width B, velocity u_e, and modulation wave vector k_0 and frequency ω, are all constant both in time and space. It should be noted here that this ansatz is valid for trivial boundary conditions (i.e., $y_1 \to \infty$, then $A \to 0$ and $\frac{dA}{dy_1} = 0$). Let us postulate that in the presence of a weak damping, the pulse shape is preserved but its characteristic parameters (in particular the pulse amplitude and width) can vary. In terms of this assumption, we consider the solution of Eq. (18) to be of the following general form [30–32]:

$$A(y_1,s_2) = A_1\mathrm{sech}(\tau)e^{-i(k_0 y_1 - \omega s_2)}, \tag{34}$$

where A_1 is the pulse amplitude, and $\tau = B(y_1 - u_e s_2)$ with $B = 1/l_e$ (l_e is the pulse width to be defined below). Substituting the general pulse solution (34) into leads to the following more explicit solution to the damped nonlinear Schrödinger equation

$$A = A_1\mathrm{sech}\left[(y_1 - u_e s_2)\sqrt{\frac{Q}{2P}}A_1 e^{-Rs_2}\right] \times i(u_e/2P)(y_1 - u_c s_2), \tag{35}$$

where the pulse amplitude A_1, the pulse width l_e, and the carrier speed u_c are given, respectively, by

$$A_1 = \sqrt{\frac{u_e^2 - 2u_e u_c}{2PQ}}e^{-Rs_2}, \tag{36}$$

$$l_e = \frac{2Pc_0}{\sqrt{h\left(u_e^2 - 2u_e u_c\right)}}, \tag{37}$$

and

$$u_c = \frac{4P^2 - 2u_e^2 l_e^2}{2u_e l_e}. \tag{38}$$

It follows from Eqs. (36) and (37) that for the width and amplitude of the damped soliton to be real, the velocities and the nonlinear and dispersion coefficients must satisfy

$$u_c < \frac{u_e}{2}, \quad PQ > 0. \tag{39}$$

Figure 2 displays time evolutions of the amplitude A_1 [Fig. 2(a)], the width l_e [Fig. 2(b)], and the carrier speed u_c [Fig. 2(c)], for some values of the damping coefficient ν.

As expected, when $\nu = 0$ (corresponding to an ideal nerve) the three characteristic parameters are constant in time. However, for nonzero values of ν the envelope amplitude decays exponentially while its width grows exponentially in time. The carrier speed grows with time and saturates at a finite threshold value which is increased as viscosity (i.e., the damping) increases. The latter observation is reminiscent of the well-established fact that the conduction velocity of the nerve impulse depends on the size of an axon, and on the thickness (and hence the viscosity) of its myelin sheath [33,34]. In Ref. [34] it was pointed out that the speed of the action potential also depends on the stiffness of the axolemma. Furthermore, the myelinization of an axon by a tight wrapping

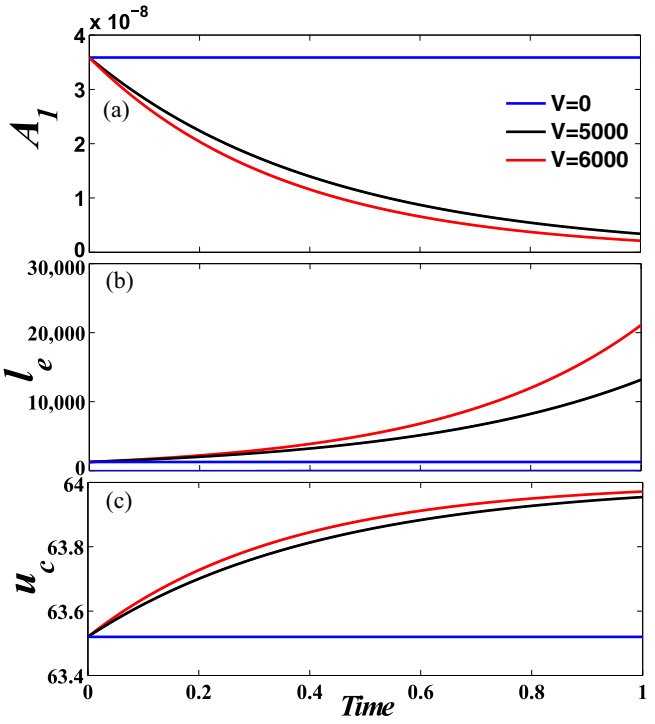

FIG. 2. Time variations of the amplitude A_1 (a), width l_e (b), and carrier speed u_e of the envelope soliton (c) for $a = 0.2$ and $k = 1$. Parameters are taken from the works of Heimburg and Jackson on unicellular DPPC vesicles [12].

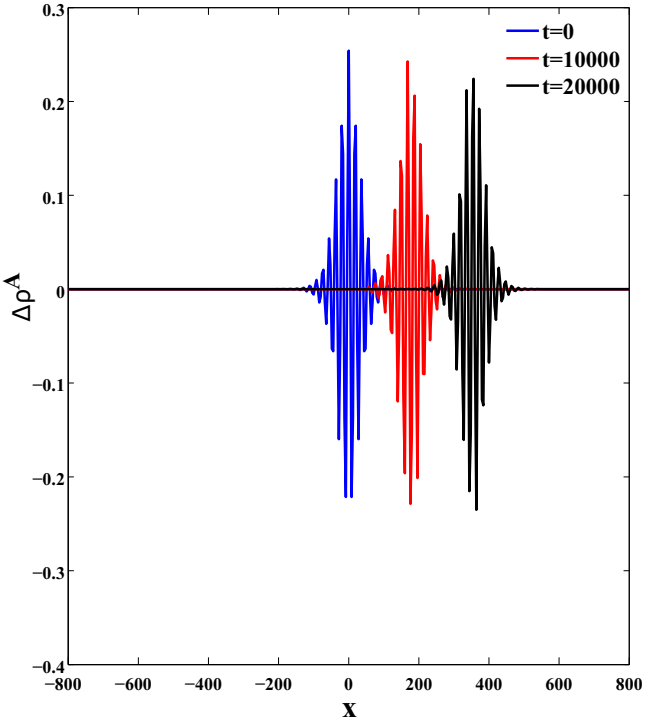

FIG. 3. Evolution of the breathing soliton ($\Delta\rho^A$) along the nerve (x), at different times. Parameters were chosen as $\varepsilon = 0.02$, $l_e = 0.8$, $a = 0.0001$, $k = 0.008$, $u_e = 100$, $u_c = 10$, $v = 0.05$, $h = 2.22$.

of Schwann cells around the axon in a spiral manner, is aimed at increasing the rigidity of the axon membrane and therefore will increase the conduction speed if the viscosity of the nerve is assumed constant. However, temperature changes have been shown to affect the viscosity, and consequently the speed of propagation of the impulse. It should be noted that the viscosity increases with a decrease in temperature [35]; an increase in the viscosity means a damping of the electromechanical waves at all frequencies and consequently an increase in the speed of the action potential [34].

B. Breathing-mode solitons

The damped nonlinear Schrödinger equation (18) does not govern the spatiotemporal evolution of the nerve impulse, but rather of the amplitude of one term in the series representing the overall solution to Eq. (1). To find the general solution representing the wave form associated with the nerve impulse in the soliton-model picture, we need to substitute (35) into (11). Proceeding with and transforming into original coordinates, we obtain

$$\Delta\rho^A = \left(2a\rho_0^A A_1\right)\mathrm{sech}\,\epsilon\left(\frac{x - V_e t}{l_e}\right)\cos(Kx - \Omega t) + \left(\frac{a^2\rho_0^A p A_1^2}{2v_g}\right)\mathrm{sech}^2\epsilon\left(\frac{x - V_e t}{l_e}\right) - \left(\frac{a^2\rho_0^A p A_1^2}{2\omega}\right)\mathrm{sech}^2\,\epsilon\left(\frac{x - V_e t}{l_e}\right)\cos 2(Kx - \Omega t), \tag{40}$$

where

$$V_e = ac_0(1 + a\epsilon u_e), \tag{41}$$

$$K = \frac{c_0}{\sqrt{h}}\left(k + \frac{au_e}{2P}\right), \tag{42}$$

and

$$\Omega = \frac{c_0^2}{\sqrt{h}}\left(k + \epsilon^2\omega + \frac{au_e}{2P} + \frac{u_e u_c a^2\epsilon^3}{2P}\right). \tag{43}$$

The solution formula (40) represents a breathing soliton displaying two important physical features, namely, the hyperpolarization and the refractory periods, observed in experiments. Figure 3 represents the evolution of the solution (40) along the nerve at different times. In the figure, the undershoot characterizing the two features is manifest through a phenomenon by which part of the nerve input signal goes below zero. Note that throughout this work, we used experimental values of the parameters proposed by Heimburg and Jackson [12] and given by $c_0 = 176.6$ m/s, $\alpha = -16.6c_0^2/\rho_0^A$, $\beta = 79.5c_0^2/(\rho_0^A)^2$, $\rho_0^A = 4.035 \times 10^{-3}$ g/m^3.

It is worthwhile stressing that the solution found in the present work [i.e., in (40)] is quite different from the Bell solitons proposed by Heimburg and Jackson [12], in which the features of hyperpolarization and refractory periods were not accounted for. To highlight the consistency of the breathing-soliton solution (40) with experimental observations, let us explore some of its relevant profiles for specific parameter sets. Consider first of all the effect of varying the magnitude of the perturbation parameter a, on the breathing-soliton profile.

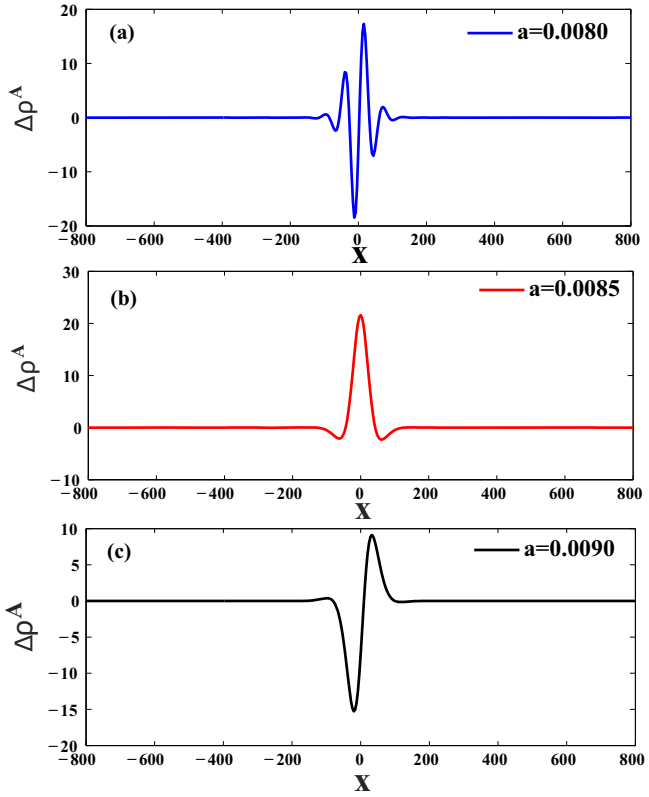

FIG. 4. Evolution of the breathing soliton ($\Delta\rho^A$) along the nerve (x), for different values of the perturbation parameter a and for $t = 0.1, l_e = 0.8, \varepsilon = 0.02, k = 0.008, u_e = 100, u_c = 10, \nu = 0.05$, $h = 2.22$. The profile (a) represents a typical breathing mode soliton while (b) and (c) are typical longitudinal and transverse profiles, respectively.

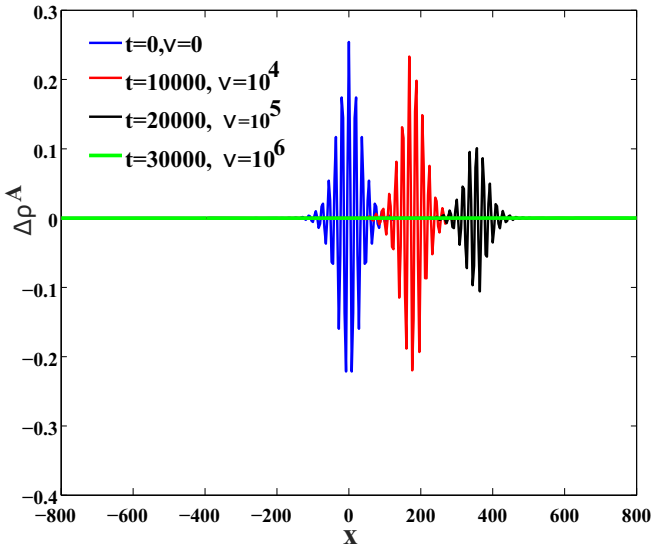

FIG. 5. Evolution of the nerve impulse nerve with changes in viscosity at different time intervals. The nerve impulse is completely damped due to an increase in the viscosity. Parameters were chosen as $\varepsilon = 0.02$, $l_e = 0.8$, $a = 0.0001$, $k = 0.008$, $u_e = 100$, $u_c = 10$, $\nu = 0.05$, $h = 2.22$.

Figure 4 shows changes in the spatial profile of the impulse as a is changed, suggesting a great variety of shape profiles for the nerve impulse when the perturbation parameter is varied (but remaining small).

The second feature reflected by the analytical solution (40) is the phenomenon of nerve-impulse blockage, associated with an increase of viscosity. In Fig. 5 we represented profiles of the impulse signal along the nerve at different times, for different values of the damping coefficient ν. As one can see, the breathing pulse soliton is highly sensitive to changes in the viscosity of the nerve. Namely, when changes in the viscosity are very large the impulse signal becomes completely damped, as illustrated in Fig. 5. This phenomenon, reflecting a blockage of the nerve signal due to changes in the viscosity, is very useful in the development of mechanical anesthesia. Such mechanical anesthesia might be advantageous to conventional chemical anesthesia in that it can easily be reversible, by reducing external pressure on the nerve. Although no change in the chemical composition of the nerve is expected, high pressure may, however, cause nerve damage. Nerve-signal blockage has been observed using different models of the nerve. For instance, in Ref. [36] Shneider and Pekker have shown, using the Hodgkin-Huxley model, that an increase in the perturbation amplitude will lead to initiation of the action potential. They stressed that a further increase may cause blockage of the nerve impulse in a region of depleted channel density. In [37], Novacek *et al.* used the Fitzhugh-Nagumo neuron model in a numerical study showing that an application of high-frequency stimulations on the nerve leads to nerve blockage, whereas low-frequency simulations of the nerve would favor propagation of the nerve impulse.

In general, viscosity is related to how thick a fluid is; it reflects a resistance of the fluid against its flow or motion. A number of factors can change the viscosity of a fluid, as for instance the change in temperature: the nerve is more viscous at low temperature than at high temperature. High pressure on the nerve also can change the internal friction (viscosity), leading to nerve-impulse blockage.

C. Self-trapping solitons

During experiments on the nerve, external fields such as ultrasound, microwave [36], pressure, or electrical signals [38] are usually utilized to excite the nerve. In general, these signals are periodic, of high amplitudes and unstable. In the nerve, they are trapped and propagate along with the solitons. To be able to stabilize these unstable external fields the nerve must possess certain properties, and the resulting stable signals can be referred to as self-trapped signals.

For self-trapping to occur in a nonlinear system such as the damped nonlinear Schrödinger equation, the nonlinearity coefficient Q must be greater than zero. This condition implies that the nonlinearity coefficient Q has the main stabilizing effect on the breathing pulse signal, which can be overcome only when the frequency of the input signal is sufficiently large [39,40]. Applying the condition $Q > 0$ to our problem, with Q defined in (19), yields the condition $\alpha^2 > 3c_0^2k^3\beta$ for the trapping and propagation of periodic waves along the nerve.

On this same issue, Tappert and Varma [39] suggested another condition for self-trapping in the nonlinear Schrödinger

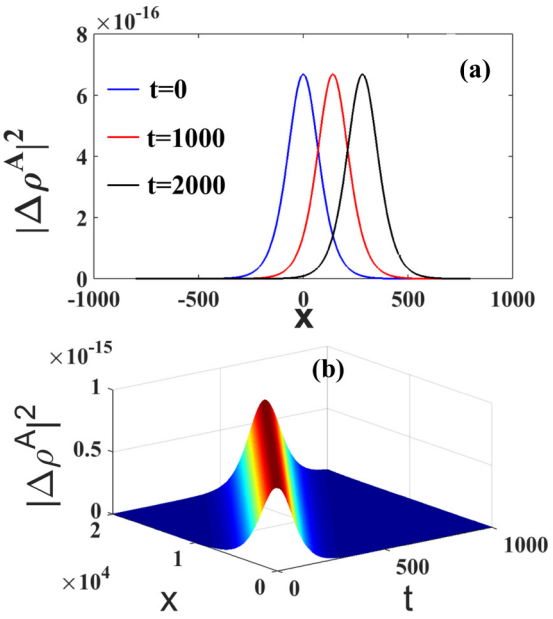

FIG. 6. (a) Amplitude of the soliton envelope at different times t. (b) Three-dimensional propagation of the nerve impulse for $\varepsilon = 0.02$, $l_e = 0.8$, $a = 0.0001$, $k = 0.008$, $u_e = 100$, $u_c = 10$, $\nu = 0.05$, $h = 2.22$. The signal propagates with constant profile without dissipation of energy.

equation. According to them, for a pulse of width l_e to undergo self-trapping its amplitude $|u|^2 = 2\varepsilon^2 a^2 |A|^2$ must exceed some critical threshold, i.e.,

$$|u^2| > |u^2|_c = \frac{rh^2}{Ql_e^2}, \tag{44}$$

where r is a perturbation parameter and h is the dispersion coefficient. Once the condition (44) is satisfied, the damped nonlinear Schrödinger equation admits stationary solution. Stationary solutions are solutions in which the dispersion and nonlinearity are exactly balanced; such solutions are called envelope solitons [39] and these are strictly the types of waves described by Heimburg and Jackson in Ref. [12]. Expressed in terms of the original coordinates, the envelope sSoliton in this case can be written

$$|(\Delta\rho^A)|^2 = 2a^2\epsilon^2 A_1^2 \left(\rho_0^A\right)^2 \mathrm{sech}^2\epsilon\left(\frac{x - V_e t}{l_e}\right). \tag{45}$$

More precisely, Eq. (45) is the amplitude of the self-trapping solution of the damped nonlinear Schrödinger equation. This solution shows that periodic unstable external stimulations can propagate as stable envelope soliton pulses, moving with constant profile even in the presence of damping. The self-trapping soliton solution is plotted in Fig. 6, where it is seen to be similar (the modulus) to that obtained numerically by Heimburg and Jackson [12] on one hand, and to the bright soliton obtained analytically by Contreras *et al.* in [15] without considering the damping, on the other hand.

V. DISCUSSION AND CONCLUSION

The propagation of action potential along the nerve can be accompanied by important mechanical responses, such as the change in the axonal radius, the change in nerve pressure, and a shortening of the axon. These mechanical responses have been predicted and well accounted for by the so-called Heimburg model of the nerve [12], in which the nerve impulse generation is regarded as a thermodynamic phenomenon related to characteristic properties of lipid membranes. The model considers the nerve as a long and narrow cylinder, in which ions flow across the membrane and the motion of the density difference on the two sides of the membrane is typical of long-wavelength (i.e., soundlike) waves.

In this work we considered the Heimburg model in the presence of damping, the aim being to investigate the effects of viscosity on soliton propagation along the nerve. We established that in the regime when nonlinearity and dispersion are balanced, the nerve-impulse propagation is described by the KdV-Burgers equation. A multiple-scale expansion method on the KdV-Burgers equation led to a damped nonlinear Schrödinger equation, whose solution was found to be a modulated pulse with a damped amplitude and time-varying width. It was shown that under specific constraints these breathing-type solitons could become self-trapped structures in which the damping is balanced by nonlinearity, such that the pulse amplitude and width remain unchanged even in the presence of damping.

The breathing-soliton solution found in this work reveals a number of interesting features which would provide relevant insight into a better understanding of the propagation of the nerve impulse in a damped nerve. Namely, we obtained that the breathing soliton oscillates while changing profile as the result of changes in the perturbation parameters, assumed to represent the soliton interaction with its environment. The breathing soliton does not break down into a series of sinusiodal waves in the presence of damping. However, at large values of the damping coefficient, corresponding to a relatively strong viscosity effect, the breathing soliton will be completely damped. This behavior is relevant in the development of a possible mechanism for mechanical anesthesia.

Although typically assumed to degrade performance, random fluctuations or noise can sometimes improve information processing in nervous system. Understanding the constructive role of noise in the context of neuronal oscillations in the brain using the soliton model for nerves can facilitate our mastering of signal processing in the neural system [41]. In the present work, we have studied the dynamics of a simple damped soliton model for nerves pulse without considering the effect of noise. However, there is a possibility of adding a noise term to the partial differential equation, describing the dynamics of the nerve pulse in the framework of the soliton model. A stochastic KdV-Burgers equation may be derived by employing some suitable transformations. Such stochastic KdV-Burgers equation was a subject for much research in recent years [42,43]. For example, the invariance of white noise under the flow of a stochastic KdV-Burgers equation, and the local and global well-posedness of the stochastic KdV-Burgers equation with white noise was analyzed by Richards in [42,43]. The effect of thermal fluctuations and/or noise sources on the formation and propagation of solitons on long Josephson junctions was also analyzed in [44–46]. In the context of nervous systems, however, the issue of noise in the nerve-impulse dynamics requires a lot of caution: noise in

the nerve system can come from several and distinct origins and a proper identification of its nature would be necessary before embarking on its treatment. An external stress, for instance, will not be treated the same way we would treat a disturbance in the nerve-impulse propagation caused by chemical reactions between enzymes, or between the ionic species flowing across the membrane and substances "foreign" to the neuronal environment. Noise will be considered in our future work in the framework of the soliton model for biomembranes and nerves after a proper identification of the nature of noise likely to affect the nerve-impulse propagation and the results obtained will be published in due time.

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