

Minimal viscoelastic model for myocardial systems

Naoaki Bekki¹ and Yoshinori Nagai²

Abstract: We present a minimal viscoelastic model for cardiac dynamics in myocardial motion and extend the reductive perturbation method for an unstable dissipative system. We derive heuristically the one-dimensional Complex Ginzburg-Landau equation from the viscoelastic model with instabilities in a certain parameter region.

1. Introduction

In the previous century, molecular biology of the cell has been tremendously developed [1]. It is well known that the contraction of muscle occurs by the sliding mechanism between two types of myofilaments [2]. A theory based on the idea of a cross-bridge rotation mechanism was proposed by Huxley [3]. There exists the regulation of Ca^{2+} -release/uptake of sarcoplasmic reticulum (SR) due to the deformation of SR by the force generated by cross-bridges. Also, the auto-oscillatory properties inherent to the contractile system of striated muscle have been investigated [4]. Ishiwata et al found that the auto-oscillation of sarcomere length (SL) occurs under the steady conditions intermediate between full contraction and relaxation conditions. They named the phenomenon after the Spontaneous Oscillatory Contraction of myofibrils (SPOC) [4, 5].

On the other hand, a variety of complex patterns which include spiral waves [6] and the spontaneous response of the myocardium to electrical excitation have been observed in human heart by developing an ultrasonic noninvasive novel imaging modality with high temporal and spatial resolutions [7]. Visualizing the propagation of the myocardial response of the electric excitation in human hearts during systole, Kanai [8, 9] observed the velocity components toward the ultrasonic probe as waveforms and their instantaneous phases of 40 [Hz] components. A velocity component corresponding to the contraction was generated on the septum at a time of T-wave of ECG (end-systole), and propagated slowly in clockwise direction along the left ventricle circumferential direction. Thus, the behavior of phase defects in a human healthy heart is one of the most interesting subjects in biophysics. In order to explain its behavior in a human healthy heart, a certain model of explanation is therefore needed on the basis of the direct observation of phase singularities. Bekki et al [10] recently proposed a possible physical model explaining a part of cardiac dynamics of these phase jumps on the interventricular septum (IVS): they have shown that at least one of the phase defects in the nonlinear excited waves on a human cardiac IVS can be explained by the Bekki-Nozaki hole solution [11] in the complex Ginzburg-Landau equation (CGLE).

A wide class of nonlinear waves for strong dispersive and dissipative systems can be described by the one-dimensional nonlinear partial differential equations which are called the driven damped nonlinear Schrödinger equation [12, 13, 14] or CGLE [15, 16, 17, 18, 19]

$$i \frac{\partial}{\partial t} \psi(x, t) + p \frac{\partial^2}{\partial x^2} \psi(x, t) + q |\psi(x, t)|^2 \psi(x, t) = i \gamma \psi(x, t). \quad (1)$$

¹ College of Engineering, Nihon University, Koriyama, Fukushima 963-8642, Japan

² Faculty of Business, Kokushikan University, Setagaya, Tokyo 154-8515, Japan

Equation (1) is called the nonlinear Schrödinger equation for the case with coefficients $p_i = q_i = \gamma = 0$, and CGLE for the case with two complex constants ($p = p_r + ip_i$, $q = q_r + iq_i$) and real positive constant γ . Here ψ is a complex function of scaled time t and space x . Equation (1) is invariant under a global change of gauge which is the multiplication of ψ by $\exp(i\phi)$, as a consequence of translational invariance of the system. Equation (1) also reduces to the nonlinear Schrödinger equation in the limit $|p_r|, |q_r| \rightarrow \infty$, which soliton solutions are integrable. The CGLE, however, describes a complex nonlinear development in a nonintegrable dissipative open system and is not tractable for analytic investigations, despite its great importance. One of the exact solutions of CGLE connects two different patterns specified by the asymptotic wavenumbers and a phase-jump between two patterns, which is called the Bekki-Nozaki (BN) hole [11]. However, very few experimental investigations of BN hole have been reported up to now [21, 20]. Some localized amplitude holes have been observed in the hydrothermal nonlinear waves, and not completely compared with BN hole solution in CGLE [21]. Therefore, the estimation of the rescaled coefficients of CGLE from the experimental data is a difficult task.

We present here a nonlinear viscoelastic model from which CGLE can be derived by use of a modified reductive perturbation method [22] instead of the center manifold theory for the chemical diffusion systems [23]. The CGLE (1) arising from unstable dissipative systems can be derived by different methods.

2. Basic Equations of Isotropic Elastic Body

A set of basic equations for a myocardial system assumed as an isotropic elastic body is given as follows:

$$\frac{\partial}{\partial t} \rho + \sum_j u_j \frac{\partial}{\partial x_j} \rho + \rho \sum_j \frac{\partial u_j}{\partial x_j} = 0, \quad (2)$$

$$\frac{\partial}{\partial t} u_i = \frac{1}{\rho} \sum_j \frac{\partial}{\partial x_j} P_{ij} + K_i, \quad (3)$$

$$\frac{\partial}{\partial t} P_{ij} = \lambda \sum_k \frac{\partial u_k}{\partial x_k} \delta_{ij} + \mu \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) = 0, \quad (4)$$

where ρ denotes a density of elastic body, u_j a velocity component and P_{ij} a stress tensor, respectively, λ and μ are Lamé elastic constants. Also, K_i ($i = 1, 2, 3$) is an external force taken into account of instability of the system. For simplicity, let us consider one-dimensional longitudinal viscoelastic waves, which is written in a matrix form:

$$\frac{\partial}{\partial t} U + A(U) \frac{\partial}{\partial x} U + B(U) = C(U) \frac{\partial^2}{\partial x^2} U, \quad (5)$$

where U is a column vector with 3 components, $U = (\rho, u, P_{11})$, and $B(U)$ is also a column vector; $A(U)$ and $C(U)$ are 3×3 matrices, which are functions of U , being assumed sufficiently smooth; x and t are the space and time coordinates, respectively. Here $B(U)$ is assumed to be a column vector containing an instability for the viscoelastic model. Many physically interesting problems involving strong dispersion and dissipation may be described by this model with instability.

The uniform state is given by a constant solution U_0 satisfying the algebraic equation

$$B^{(0)} = B(U_0) = 0, \quad (6)$$

where $U_0 = \begin{pmatrix} \rho_0 \\ 0 \\ 0 \end{pmatrix}$.

3. Modified Reductive Perturbation Method

Let us consider a nonlinear amplitude modulation of the nearly monochromatic plane wave subject to Eq. (5), that is,

$$U = U_0 + \delta U \exp[i(kx - \Omega t)] + \text{complex conjugation}, \quad (7)$$

where k is the real wavenumber and Ω the complex frequency near the critical wavenumber k_0 . Then, linearizing Eq. (5) about U_0 , we have the dispersion relation:

$$\det \left[-i\Omega I_{id} + ikA^{(0)} + \nabla B^{(0)} + k^2 C^{(0)} \right] = 0, \quad (8)$$

where

$$A^{(0)} = A(U_0),$$

$$\nabla B^{(0)} = \left[\frac{\partial B_i}{\partial u_j} \right]_{U=U_0},$$

$$C^{(0)} = C(U_0).$$

Above dispersion relation yields the domain of existence for the wave. Hereafter, we focus all our attention on the wave for the case of the small imaginary part of the complex frequency: $\Im(\Omega) = \gamma$. In $\Omega(k_0)$ thus obtained, we may assume for $|l| \neq 0$,

$$\det W_l \neq 0, \quad (9)$$

where the matrix W_l is defined by

$$W_l = -il\Omega I_{id} + ilkA^{(0)} + \nabla B^{(0)} + l^2 k^2 C^{(0)}. \quad (10)$$

According to the extended reductive perturbation method by Asano [22], we introduce the slow variables through the stretching

$$\begin{aligned} \xi &= \epsilon(x - v_g t), \\ \tau &= \epsilon^2 t, \end{aligned} \quad (11)$$

where the parameter v_g is determined through a compatibility condition as the complex group velocity

$$v_g = \frac{\partial \Omega}{\partial k}, \quad (12)$$

and ϵ is a parameter specifying the smallness of the amplitude.

Let us assume the following solution;

$$U = U_0 + \sum_{\alpha=1}^{\infty} \epsilon^\alpha U^{(\alpha)}, \quad (13)$$

where

$$U^{(\alpha)} = \sum_{l=-\infty}^{\infty} U_l^{(\alpha)}(\xi, \tau) X_l,$$

$$X_l = \exp[il(kx - \Omega t)].$$

As the reality condition, we have

$$U_l^{(\alpha)} = U_{-l}^{(\alpha)*}, \quad (14)$$

where the asterisk denotes the complex conjugation.

The expansions of $A(U)$, $B(U)$ and $C(U)$ can be given in terms of U as follows;

$$A = A^{(0)} + \epsilon \nabla A^{(0)} U^{(1)} + \epsilon^2 \left(\nabla A^{(0)} U^{(2)} + \frac{1}{2} \nabla \nabla A^{(0)} : U^{(1)} U^{(1)} \right) \dots, \quad (15)$$

where

$$\nabla \nabla A^{(0)} : U^{(1)} U^{(1)} = \sum_{i,j} \left[\frac{\partial^2 A}{\partial u_i \partial u_j} \right]_{U=U_0} u_i^{(1)} u_j^{(1)}.$$

The expansions of matrices B and C are similar to A .

Substituting these expansions into Eq. (5), and equating the various powers of ϵ of the l -th harmonics to zero, we get

$$W_l U_l^{(1)} = 0, \quad (16)$$

$$\begin{aligned} W_l U_l^{(2)} + \left(-v_g I_{id} + A^{(0)} - 2ilkC^{(0)} \right) \frac{\partial}{\partial \xi} U_l^{(1)} + \left\langle \nabla A^{(0)} U^{(1)} \sum_{l'} il'k U_{l'}^{(1)} X_{l'} \right\rangle_l \\ + \frac{1}{2} \left\langle \nabla \nabla B^{(0)} : U^{(1)} U^{(1)} \right\rangle_l + \left\langle \nabla C^{(0)} U^{(1)} \sum_{l'} (l'k)^2 U_{l'}^{(1)} X_{l'} \right\rangle_l = 0, \end{aligned} \quad (17)$$

$$W_l U_l^{(3)} + \left(-v_g I_{id} + A^{(0)} - 2ilkC^{(0)} \right) \frac{\partial}{\partial \xi} U_l^{(2)} + I_{id} \frac{\partial}{\partial \tau} U_l^{(1)} - C^{(0)} \frac{\partial^2}{\partial \xi^2} U_l^{(1)}$$

$$\begin{aligned}
 & + \left\langle \sum_{l'} (A^{(0)} - 2il'k\nabla C^{(0)}) U^{(1)} \frac{\partial}{\partial \xi} U_{l'}^{(1)} X_{l'} \right\rangle_l + ik \left\langle \sum_{l'} l' A^{(0)} (U^{(1)} U_{l'}^{(2)} + U^{(2)} U_{l'}^{(1)}) X_{l'} \right\rangle_l \\
 & \quad + \frac{1}{2} ik \left\langle \sum_{l'} l' (\nabla \nabla A^{(0)} : U^{(1)} U^{(1)}) U_{l'}^{(1)} X_{l'} \right\rangle_l + \left\langle \nabla \nabla B^{(0)} : U^{(1)} U^{(2)} \right\rangle_l \\
 & \quad + \frac{1}{6} \left\langle \nabla \nabla \nabla B^{(0)} : U^{(1)} U^{(1)} U^{(1)} \right\rangle_l + k^2 \left\langle \sum_{l'} (l')^2 \nabla C^{(0)} (U^{(1)} U_{l'}^{(2)} + U^{(2)} U_{l'}^{(1)}) X_{l'} \right\rangle_l \\
 & \quad + \frac{1}{2} k^2 \left\langle \sum_{l'} (l')^2 (\nabla \nabla C^{(0)} : (U^{(1)} U^{(1)}) U_{l'}^{(1)} X_{l'} \right\rangle_l = 0, \tag{18}
 \end{aligned}$$

where the angle bracket denotes the coefficient of the l -th harmonics, that is, for any function F ,

$$F = \sum_{l=-\infty}^{\infty} \langle F \rangle_l X_l.$$

Since we consider the nonlinear amplitude modulation of the plane wave with the complex frequency Ω and the wave number k , $U_l^{(1)}$ must be zero for $|l| \neq 1$. therefore, we have

$$U_l^{(1)} = 0 \quad \text{for } |l| \neq 1, \tag{19}$$

while

$$U_1^{(1)} = \phi R \quad \text{for } l = 1, \tag{20}$$

where R is the right eigenvector of W_1 ,

$$W_1 R = 0, \tag{21}$$

and ϕ is a scalar function of the slow variables to be determined later.

The third, fourth and fifth terms on the left hand side of Eq. (17) result from the self-interaction of the fundamental mode and it is obvious that they are non-vanishing only for $|l| = 2$ and $l = 0$. Hence, for $l = 1$, Eq. (17) becomes

$$W_1 U_1^{(2)} + \left(-v_g I_{id} + A^{(0)} - 2ikC^{(0)} \right) R \frac{\partial}{\partial \xi} \phi = 0. \tag{22}$$

As Eq. (22) must be algebraically solvable for $U_1^{(2)}$, the compatibility condition is required as follows,

$$L \left(-v_g I_{id} + A^{(0)} - 2ikC^{(0)} \right) R = 0, \tag{23}$$

where L is the row left eigenvector of W_1 , that is,

$$L W_1 = 0. \tag{24}$$

The solution $U_1^{(2)}$ of Eq. (22) is written in terms of an arbitrary function $\phi^{(2)}(\xi, \tau)$ as follows

$$U_1^{(2)} = R\phi^{(2)} - i\frac{\partial R}{\partial k}\frac{\partial}{\partial \xi}\phi. \quad (25)$$

Further, the component with $l = 0$ of Eq. (17) becomes

$$\begin{aligned} W_0 U_0^{(2)} + ik \left(\nabla A^{(0)} U_{-1}^{(1)} U_1^{(1)} - c.c. \right) + \frac{1}{2} \left(\nabla \nabla B^{(0)} : U_{-1}^{(1)} U_1^{(1)} + c.c. \right) \\ + k^2 \left(\nabla C^{(0)} U_{-1}^{(1)} U_1^{(1)} + c.c. \right) = 0, \end{aligned} \quad (26)$$

and yields $U_0^{(2)}$ in the form

$$U_0^{(2)} = R_0^{(2)} |\phi|^2, \quad (27)$$

where $R_0^{(2)}$ is a column vector, satisfying

$$\begin{aligned} W_0 R_0^{(2)} + ik \left(\nabla A^{(0)} R^* R - c.c. \right) + \frac{1}{2} \left(\nabla \nabla B^{(0)} : R^* R + c.c. \right) \\ + k^2 \left(\nabla C^{(0)} R^* R + c.c. \right) = 0. \end{aligned} \quad (28)$$

If get $W_0 \neq 0$, we have

$$\begin{aligned} R_0^{(2)} = -W_0^{-1} \left[ik \left(\nabla A^{(0)} R^* R - c.c. \right) + \frac{1}{2} \left(\nabla \nabla B^{(0)} : R^* R + c.c. \right) \right. \\ \left. + k^2 \left(\nabla C^{(0)} R^* R + c.c. \right) \right], \end{aligned} \quad (29)$$

where W_0^{-1} denotes the inverse matrix of W_0 . Other components of $U^{(2)}$ are similarly given by

$$U_2^{(2)} = R_2^{(2)} (\phi)^2, \quad (30)$$

$$U_l^{(2)} = 0 \quad \text{for } |l| \geq 3, \quad (31)$$

with

$$R_2^{(2)} = -W_2^{-1} \left[ik \nabla A^{(0)} R R + \frac{1}{2} \nabla \nabla B^{(0)} : R R + k^2 \nabla C^{(0)} R R \right], \quad (32)$$

where W_2^{-1} stands for the inverse matrix of W_2 . It is notable that the nonlinear terms in Eq. (18) do not contain $\phi^{(2)}$ for $|l| = 1$.

4. Derivation of CGLE from Nonlinear Viscoelastic Model

We are now ready to determine $\phi(\xi, \tau)$. Multiply Eq. (18) for $l = 1$ by the left eigenvector L from the left, and substitute the solutions of U into Eq. (18) with $l = 1$. Then, by means of Eq. (24), the first term disappears, and the compatibility condition Eq. (23) eliminates $\phi^{(2)}$ in the second term. Consequently, it reduces to an equation for $\psi = \phi(x, t)$, that is, the complex Ginzburg-Landau equation (1),

$$i\frac{\partial}{\partial t}\psi(x, t) + (p_r + ip_i)\frac{\partial^2}{\partial x^2}\psi(x, t) + (q_r + iq_i)|\psi(x, t)|^2\psi(x, t) = i\gamma\psi(x, t). \quad (33)$$

5. Conclusions

We have heuristically derived the Complex Ginzburg-Landau equation from the nonlinear viscoelastic model by means of the modified reductive perturbation method [12, 22]. By the novel ultrasonic method[8], we have obtained the significant data of phase-defects in the excited waves due to the mechanical motions of the aortic-valve in a time of aortic-valve closure at end-systole. Although it is difficult to estimate all the coefficients of CGLE from the observed data, we found consistently all the corresponding coefficients of CGLE. We have already shown that the dynamics of nonlinearly excited waves on the septum wall in vivo can be described by BN hole solution in the direct comparison between the data of the noninvasive direct measurements and BN hole solution with the corresponding coefficients. We have succeeded in obtaining the data related to the BN hole and in confirming physically that these data of excited waves on the septum wall in vivo can be explained by BN hole solution in CGLE [10]. We have found the first evidence of the BN hole generated by the nonlinear modulation of the excited waves on the septum wall. It should be noted that the coefficient $\Im(p) = p_i$ based on the present model plays an important role in estimating the cardiac viscoelasticity of human heart. We can apply our viscoelastic model to the cardiac systems. These results will be published elsewhere.

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