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Dissipative Mayer's waves in fluid-filled viscoelastic tubes

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ABSTRACT

Wave propagation in a viscoelastic tube filled with viscous fluid is addressed. We show that the dissipative Navier–Stokes equations can asymptotically be reduced to a pair of nonlinearly coupled complex Ginzburg–Landau equations. Modulational instability is then investigated analytically and numerically. The instability domain, using the growth rate, is shown to be importantly dependent on the vessel relative stiffness and fluid viscosity. A comprehensive analysis is proposed to that effect, which is confirmed by direct numerical simulations. Dissipative trains of impulses are found as the main manifestation of modulational instability and results are recorded for some hemodynamic factors such as the pressure, velocity and vessel cross-section.

1. Introduction

Nonlinear waves and solitons are found in any system where there is a competition between nonlinearity and dispersion. The physical systems where they appear span from biophysics to hydrodynamics, through optical communication and material sciences. One of the most effective systems where solitons play a mayor role is blood vessels. In fact, from the seminal works of Yomosa [1] and Hashimuze [2], it has been accepted and experimentally demonstrated that blood pressure waves are not different from solitary waves. This has been applied to large vessels by several authors, with emphasis on the nonlinear elasticity of the vessel wall, using some weakly nonlinear theories [3–7]. In the same vein, it was shown by several investigators that the appropriate equation that gives a wave profile similar to experimental blood pulses was the Korteveg–de Vries (KdV) one [3,4,8]. In recent years, attention has also been paid to Mayer waves [9–12], known as periodic fluctuations in blood pressure (BP), mainly in vasodepressor carotid sinus hypersensitivity [13]. Abnormal conditions, lack of oxygen, severe hemorrhage, and many other sudden changes in blood circulation, in organs and tissues, may be responsible for the appearance of Mayer waves [14,15]. The emergence of Mayer waves was recently predicted using a discrete nonlinear Schrödinger (NLS) equation, where it was considered that the amplitude of the wave may be considered small-but-finite, and may vary both in space and time variables in presence of nonlinearity and dispersion [16]. Nonlinear self-modulation in fluid-filled elastic tubes was already addressed by Ravindran and Prasad [17], where the NLS equation was derived. Erbay and Erbay [18] also derived the dissipative NLS equation from nonlinear equations of fluid flow in viscoelastic thin tubes. Bakirtaş and Demiray [19], adopting a viscous fluid flow in prestressed elastic tubes, showed the dynamics of modulated amplitude blood waves to be modeled by the dissipative NLS equation.

Blood is a concentrated suspension of blood cells in plasma, and may display non-Newtonian behaviors [20]. These characteristics originate from the deformability and aggregation of red blood cells, and have strong impact on blood viscosity (BV). BV is a key factor for the normal operation of the circulatory system, and can change due to several factors such as the Hematocrit. In general, the variations in BV are detected by the endothelium, leading to the activation of vasoactive materials which includes nitric oxide, prostacyclin and endothelin, capable of controlling BV, and the wall shear stress [21]. When the aforementioned compensation mechanism fails, blood exhibits hyperviscosity features, thrombosis can appear, with negative effects, and even damage, on the endothelium [22]. Many diseases are associated with BV, mainly because blood cell velocities drop, especially in diabetic patients [23,24]. The occlusions of the oxygen-transporting vessels cause tissue ischemia and necrosis, bringing in other factors like high blood pressure (BP). Indubitably, that may affect arterial distensibility/stiffness. However, the correlation between arterial stiffness and blood viscosity is not yet well understood, although the measurement of arterial pulse wave velocity was used recently to that effect, with interest in isolated systolic hypertension, responsible for blood reflexion and pulse pressure [25]. Nevertheless, there are gathered evi-

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dences that since the wall shear stress is highly sensitive to high BV, the risk for aneurysm formation, progression and rupture is high [26,27]. This may induce sudden changes in both heart rate and arterial pressure fluctuations, similar to Mayer wave, for example [9,10,14]. The aim of the present contribution is then to characterize such waves in the presence of BV and arterial stiffness [28]. As said so far, high arterial stiffness induces reflected blood waves. We therefore show that the suitable description of their amplitude and phase modulation may lead to a pair of nonlinearly coupled complex Ginzburg-Landau (CGL) equations. The CGL equation is, in fact, well known in the dynamics of nonlinear dissipative patterns, with application to nonlinear optics [29], reaction-diffusion systems [30], neural networks [31,32] and molecular structures such as DNA and proteins [33–35]. Indeed, long-time envelope soliton dynamics may be satisfactorily described by both the NLS and CGL equations, which is not the case for modulated periodic stokes waves that may be explained exclusively using the CGL equation [29,36]. Modulated periodic waves, such as Mayer waves may arise in such system under the activation of modulational instability (MI), a consequence of the concomitant effects of nonlinearity and dispersion. MI is one of the direct mechanisms that lead to the formation of nonlinear waves, where a specific range of wavenumbers of plane wave profiles of the form $\varphi(x,t) \sim \exp[i(kx-\omega t)]$ becomes unstable to modulations. Unstable modes exponentially emerge, resulting to the formation in space and time of coherent solitary structures that appear in a broad range of physical systems [29,31,32,37–44]. The MI technique is adopted in this work, both analytically and numerically, to address the emergence of Mayer waves and, more importantly, their response to high BV and arterial stiffness variations. To proceed, we introduce the dissipative Navier–Stokes model that describes both the blood and arterial wall dynamics in Section 2. Thereafter, the reductive perturbation expansion is used to show that amplitude equations are nonlinearly coupled CGL equations. In Section 3, a comprehensive scheme of the MI analysis is presented, along with numerical confirmation of analytical predictions. Importance is given to the impact of BV and and arterial stiffness parameters on the waveforms of some hemodynamic factors such as blood pressure, blood velocity and arterial cross-section. Some concluding remarks are finally given in Section 4.

2. Governing equations and mathematical background

The system under our study is made of simultaneous equations that couple the dynamics of the blood and the deformation of the arterial wall. Blood is in fact considered as an inviscid fluid flowing in a cylindrical elastic tube of reference radius A_0, subjected to a uniform inner pressure P_0. In the cylindrical coordinate formulation, i.e., (r^*, θ^*, z^*), where r and z are respectively the radial and longitudinal coordinates, we consider a one-dimensional field of longitudinal flow velocity $W(z^*, t^*)$, fluid pressure $P(z^*, t^*)$ and the radial displacement of the arterial wall $A(z^*, t^*)$. Based on the formulation proposed by Yomosa [1,45], the equation of mass conservation of the fluid may be written as

$$\frac{\partial W}{\partial t^*} + W\frac{\partial W}{\partial z^*} + \frac{1}{\rho}\frac{\partial P}{\partial z^*} = \nu\left(-8\frac{W}{A^2} + \frac{\partial^2 W}{\partial z^{*2}}\right), \tag{1}$$

where ρ and ν are the fluid density and kinematic viscosity, respectively. The above equation has been obviously modified to include viscous effects that are not considered in the simple Yomosa's model [1]. In the course of blood flow in arteries, the red blood cells migrate to the central region of the artery and, thus, the hematocrit ratio drops near the arterial wall, where the shear rate is quite high, as usually obvious in Poiseuille flows. In order to derive the above equation and the ones that follow, Demiray [3] made use of the averaging method and additionally included variable cross-section. However, in this work, that last aspect is not considered. Moreover, the following equation of continuity stands for the incompressibility of blood:

$$\frac{\partial A^2}{\partial t^*} + \frac{\partial}{\partial z^*}(A^2 W) = 0. \tag{2}$$

Finally, the radial motion of the arterial wall is describes by the equation

$$\rho_0 H\frac{\partial^2 \delta A}{\partial t^{*2}} = P - \overline{P} + \gamma\frac{\partial \delta A}{\partial t^*} + \beta\frac{\partial^2 \delta A}{\partial z^{*2}} + \varphi\frac{\partial^3 \delta A}{\partial t^* \partial z^{*2}} - \frac{h}{A}\sigma_t, \tag{3}$$

obtained through the second law of Newton [1,16,46]. $\overline{P}$ is the pressure outside the vessel, δA is the vessel deformation, ρ_0 represents the wall density. H and h stand for the effective inertial thickness and the thickness of the wall, respectively. γ is the viscoelasticity coefficient, β is the shear modulus, and φ is another coefficient of viscoelasticity. σ_t is the approximated relation function for the stress-strain relationship proposed in [47,48] as

$$\sigma_t = E\frac{\delta A}{\overline{A}}\left(\frac{1+a\delta A}{\overline{A}}\right), \tag{4}$$

where $\overline{A}(z)$ is the stationary radius of the vessel, E is the Young modulus and a represents the nonlinear coefficient of elasticity. In the context of weak wall displacements, the approximations

$$A - \overline{A} = \delta A = \frac{1}{2}(u-1) \quad \text{and} \quad A = \frac{1}{2}(u+1) \tag{5}$$

can be used, where $u = \frac{s}{s_0}$ and $s_0 = \pi\overline{A}^2$ are the normalized cross sectional area of the tube and the cross sectional area at the entrance. A_0, ω_0 and E_0 are, respectively the reference values of radius, velocity and Young modulus at the entrance of the vessel. We also introduce dimensionless parameters $W \to \omega_0 v$, $P - \overline{P} \to p_0 p$, $t \to t^* T$, and $z \to z^* A_0$ so that after substituting Eqs. (4) and (5) into (2) and (3), we obtain the following set of dimensionless equations both for the fluid and the tube:

$$s_t\frac{\partial u}{\partial t} + v\frac{\partial u}{\partial z} + \frac{(u+1)}{2}\frac{\partial v}{\partial z} = 0 \tag{6a}$$

$$s_t\frac{\partial v}{\partial t} + v\frac{\partial v}{\partial z} + \frac{\partial p}{\partial z} + \eta_1 v(1-2u) - \eta_2\frac{\partial^2 v}{\partial z^2} = 0 \tag{6b}$$

$$a_1(1-u)\frac{\partial^2 u}{\partial t^2} - \frac{a_2}{2}\frac{\partial u}{\partial t} - \frac{b_1}{2}\frac{\partial^2 u}{\partial z^2} - \frac{b_2}{2}\frac{\partial^3 u}{\partial t \partial z^2} + \Xi^{-1}(u-1)^2[2 + a_1(u-1)] - p(z,t) = 0, \tag{6c}$$

where $\eta_1 = \frac{32A_0\nu}{\omega_0}$ and $\eta_2 = \frac{\nu}{A_0\omega_0}$ are the viscosity coefficients, $a_1 = \frac{s_t^2}{\rho\omega_0}$, $a_2 = \frac{\gamma s_t}{\rho\omega_0}$, $b_1 = \frac{\beta}{\rho\omega_0^2 A_0}$, $b_2 = \frac{\varphi s_t}{\rho\omega_0 A_0^2}$, $s_t^{-1} = T\omega_0 A_0^{-1}$, with $\Xi = \rho\omega_0^2 A_0 (E_0 h_0)^{-1}$ and $s_t = A_0/T\omega_0$ being the relative stiffness of the vessel and the Strouhal number, respectively. For the description of modulated pressure waves using Eqs. (6), we consider the multiple-scaling scheme, where spatial and temporal variables take the form $z_n = \epsilon^n z$ and $t_n = \epsilon^n t$, with $n = 0, 1, 2, 3, \ldots$, where ϵ is a small parameter. Moreover, after assuming the viscosity coefficients η_1 and η_2 to be of order ϵ^2, solutions u, v and p for (6) are expanded in the form

$$\begin{aligned} u &= \sum_{n=1}^{\infty}\epsilon^n u_n(z_0, z_1, z_2, \ldots; t_0, t_1, t_2; \ldots), \\ v &= \sum_{n=1}^{\infty}\epsilon^n v_n(z_0, z_1, z_2, \ldots; t_0, t_1, t_2; \ldots) \\ p &= \sum_{n=1}^{\infty}\epsilon^n p_n(z_0, z_1, z_2, \ldots; t_0, t_1, t_2; \ldots). \end{aligned} \tag{7}$$