Nonlinear problems in arterial flows

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Abstract

A differential model for a viscous fluid flowing in a deformable tube fixed at the two ends is presented as a fundamental study. The main application is in arterial mechanics where the fluid-solid interaction is of primary importance. A nonlinear viscoelastic constitutive equation for the wall is coupled with the 1D averaged fluid momentum equation. The equations are solved numerically by a finite difference method on a staggered grid and the dependence on the relevant physical parameters is discussed.

Key words: Wave propagation, wall-fluid interaction, viscoelasticity, nonlinear models.
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1 Introduction

Model studies of flows in liquid filled distensible tubes are motivated from the desire to understand the many aspects of the cardiovascular system in physiological and pathological states. Blood flow in arteries is dominated by unsteadiness and by wave propagation phenomena generated by the interaction of the blood with the arterial wall.
The importance of the arterial mechanics is widely recognized in modelling hemodynamical problems. Some work has been carried out with the simplistic assumption that the vessel wall is linearly elastic and isotropic [1],[2]. Actually, the complex nature of biological tissues needs the development of nonlinear theories. Nonlinearities are not much relevant for predictions of wave speed,

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but influence the pressure and flow waveforms. This type of nonlinearity is a consequence of the curvature of the stress-strain function which shows that an artery becomes stiffer as the distending pressure is raised. Some authors have shown that elasticity dominates the nonlinear mechanical properties of arterial tissues, whereas the vessel viscosity can be considered as a second order effect [3]. On the other hand, experimental studies indicate that the arterial material is viscoelastic and anisotropic [4]. In principle, viscoelastic dissipation of the vascular wall proves to be more important than viscous dissipation of the fluid. Actually, the latter can be neglected in a number of applications involving large blood vessels [1]. A review on the theoretical developments and new trends in arterial mechanics is given in [5].

Many theoretical and experimental formulations have been developed to describe the finite deformation and the nonlinear viscolasticity of arteries in time dependent flows. A nonlinear constitutive relation for the vascular wall that depends on the Green strains has been introduced in [6] and a stability analysis on the saccular aneurysm evolution is presented in [7]. The model is here extended to include the effect of the viscoelasticity of the solid tube and, when necessary, the viscosity of the blood. According to the experimental results, the wall stress is a function of both the strain and strain rate. The inertia of the wall mass, even including the effective mass from the surrounding soft tissues, is negligible compared with the elastic force because of low wall velocities [8].

Being interested in the pulse propagation phenomena, the assumption of a quasi-1D flow is a valid approach under the hypothesis that the wave amplitude is small and the wavelength is long compared with the tube radius, so that the slope of the deformed wall remains small at all times [9]. We consider a homogeneous nonlinear viscoelastic tube filled with an incompressible fluid and all the quantities are assumed to vary in the axial direction only because the equations have been averaged over the cross section.

Our aim is to get a satisfactory understanding of the mechanism of propagation of the pressure pulse, and of the changes in the pressure waveform which occur as it travels along the arteries through the nonlinear wall-fluid interaction. The effect of the elasticity parameter is related to the frequency of oscillations in the transient, while the influence of viscosity parameter is to attenuate the oscillations, to reduce the tendency of shock formation as in a purely elastic wall model, and to counterbalance possible instability phenomena. Despite the nonlinearity of the elastic part, the results are qualitatively similar to those obtained with a linear elastic relation studied in [10], because of the small arterial deformations. However, the numerical value of the elastic and viscous coefficients appearing in the constitutive equation are critical and need to be carefully identified by comparing numerical results with measurements.

2 The viscoelasticity of the vessel wall

The adequate mechanical characterization of blood vessels is an important prerequisite for a quantitative description of blood flow, as well as for the study of the wave propagation phenomena.
For an incompressible hyperelastic material it is possible to define a strain-energy function W as a function of the stretch-ratio invariants I_1, I_2, I_3: it represents the elastically stored energy per unit volume in terms of the strain variables and is a potential for determining the stress [5]. The problem of determining the form of the strain-energy function for biological material has been examined from theoretical and experimental points of view. A variety of mathematical expressions for W has been proposed in biomechanics, according to different materials and organic tissues, and their efficiency is tested in the ability to fit experimental data over a wide range of strains. As pointed out by Fung [4] and other authors [5], the properties of vascular tissues are highly nonlinear. Some attempts to define a non-linear strain-energy density function for the arterial tissue are based on the static relationship between strains and elastic energy (see for example [4],[6],[11], and references therein).
Let us now consider the vessel wall modelled as an elastic axisymmetric membrane. This is a 2D thin shell with a mass negligible compared with that of the fluid contained in it. The membrane is capable to deform under the forces exerted by the fluid, is subject only to stresses in the tangential plane and has no bending stiffness. Let $(x_{_P}(s), r_{_P}(s))$ be the lagrangian coordinates of a particle P having a parametric coordinate s along the membrane in its symmetry plane. The strain-energy density function per unit area can be formulated as:

$$w = w(\lambda_1, \lambda_2)$$

where

$$\lambda_1 = \sqrt{\left(\frac{dr_{_P}}{ds}\right)^2 + \left(\frac{dx_{_P}}{ds}\right)^2} \qquad \lambda_2 = \frac{r_{_P}}{R_u} \tag{2.1}$$

are the principal deformation ratios in the meridional and circumferential directions and R_u is the undeformed radius. In this context, a constitutive strain-energy function modelling the mechanical properties of the arterial wall has been recently proposed [6],[7] as:

$$w = c\,(e^Q - 1) \qquad Q = c_1(E_1^2 + E_2^2) + 2c_3E_1E_2$$

where c is a material parameter, c_1, c_3 are nondimensional constants and $E_k = \frac{1}{2}(\lambda_k^2 - 1) \quad k = 1, 2$ are the principal Green strains. Once the form of w

is specified, the mechanical properties are completely determined, being the stress components (averaged across the thickness) along the longitudinal and circumferential directions given by differentiation of w:

$$T_1(\lambda_1,\lambda_2)=\frac{\lambda_1}{\lambda_2}\frac{\partial w}{\partial E_1}=\frac{1}{\lambda_2}\frac{\partial w}{\partial \lambda_1} \qquad T_2(\lambda_1,\lambda_2)=\frac{\lambda_2}{\lambda_1}\frac{\partial w}{\partial E_2}=\frac{1}{\lambda_1}\frac{\partial w}{\partial \lambda_2} \tag{2.2}$$

The former relations hold in the case of an incompressible and isotropic material, wherein principal directions of strain and stress coincide and express the property that the instantaneous Young's modulus increases with the strain, but with a different amount in the two directions [5].
On the other hand, many authors have pointed out that the vessel walls are viscoelastic. Patel and Vaishnav verified the existence of the arterial viscoelasticity through a dynamical experiment [9]. Reuderink found that a neglect of the viscoelasticity generates an underestimation of both phase velocity and damping [2]. Generally, a viscoelastic wall model yields results closer to physiological measurement than an elastic one, and a dissipative wall is more effective than a dissipative fluid in eliminating the high frequency oscillations. The damping resulting from viscoelasticity inhibits the sharp peaks of the pressure and flow pulses and leads to more realistic results when a comparison with experimental data is carried out [12].
The simplest generalization of (2.2), including a viscoelastic effect, is given by the following strain-stress relationship:

$$\begin{aligned} T_1(\lambda_1,\lambda_2,\dot\lambda_1,\dot\lambda_2)&=\frac{1}{\lambda_2}\frac{\partial w}{\partial \lambda_1}+\gamma\left(\dot\lambda_1+\frac{\dot\lambda_2}{2}\right)\\ T_2(\lambda_1,\lambda_2,\dot\lambda_1,\dot\lambda_2)&=\frac{1}{\lambda_1}\frac{\partial w}{\partial \lambda_2}+\gamma\left(\dot\lambda_2+\frac{\dot\lambda_1}{2}\right) \end{aligned} \tag{2.3}$$

where $\gamma>0$ is a wall viscosity coefficient and the dot denotes time derivative [13].
Although the inertia of the membrane is neglected and a general theoretical framework is still lacking, in the model case studied here the simple functional dependence strain-stress in equations (2.3) takes into account the viscous effects of a material in time dependent motions and models the response of the arterial wall to the deformation and to the rate of deformation. In other words, equations (2.3) mean that the membrane does not respond instantaneously to forces, as a purely elastic body, but with a dissipative mechanism as a viscoelastic material.

3 The wall-fluid coupling

Being interested in pulse propagation in arteries and because of the small deformations of the vascular wall, a quasi-one dimensional model is assumed. Let us consider the 1D cross averaged momentum equation of a homogeneous, incompressible fluid in an axisymmetrical distensible tube of circular cross section of radius R and length L, with the two ends at fixed radius R_0 (all variables are assumed to be nondimensional):

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} = -\frac{\partial p}{\partial x} + f \tag{3.1}$$

where x is the axial coordinate, u is the mean axial velocity, p denotes the transmural pressure, t the time and f a friction term. The latter is locally approximated by the friction term of the Poiseuille steady flow in a tube of radius R given by:

$$f \simeq -\frac{8u}{Re\, R^2} \tag{3.2}$$

with $Re = \dfrac{U_0 R_0}{\nu}$ the Reynolds number and U_0 a characteristic velocity. As a consequence, the wall shear stress is approximated by:

$$\tau = \left.\frac{du}{dr}\right|_R \simeq -\frac{4u}{Re\, R} \tag{3.3}$$

In principle, the expressions (3.2) and (3.3) hold for a steady flow in a rigid tube, but they are considered acceptable for quasi-steady regimes and for small deformations ($R \approx R_0$) [2].

In a distensible tube the continuity equation is:

$$\frac{\partial R}{\partial t} + \frac{R}{2}\frac{\partial u}{\partial x} + u\frac{\partial R}{\partial x} = 0 \tag{3.4}$$

[1]. Because of its small inertia, the vessel wall is modelled as a membrane which deforms under the fluid forces and reaches an equilibrium state. Let us indicate by $R(x,t)$ and $S(x,t)$ the Eulerian counterparts of the Lagrangian coordinates of a particle of the a membrane (see previous section). The fluid–membrane equilibrium equations in x and r directions are provided [14]:

$$R'(T_1 - T_2) + RT_1' = \tau R R'$$

$$\frac{-R''}{(1+R'^2)^{\frac{3}{2}}}T_1 + \frac{1}{R(1+R'^2)^{\frac{1}{2}}}T_2 = p \tag{3.5}$$

where τ is the shear stress exerted by the viscous fluid on the wall (cfr. (3.3)), nondimensional T_1 and T_2 are defined as in (2.3) with:

$$\lambda_1 = \sqrt{\frac{1+R'^2}{S'^2}} \qquad \lambda_2 = \frac{R}{R_u}$$

the principal strains (the prime denotes x-derivative).
The following boundary conditions are provided at the ends of the tube:

$$\begin{array}{ll} R\,(0,t) = 1 & S\,(0,t) = 0 \\ R\,(L,t) = 1 & S\,(L,t) = L_u \end{array}$$

where L_u is the length of the undeformed membrane. Following the experimental set up, only the values of $p(0,t)$ and $p(L,t)$ are given at both boundaries. In the inviscid case, this leads to some indeterminacy in the value of u, which depends on the initial data. This approach is different from that in [13], where the value of flow rate is assigned at one of the boundaries together with the value of the pressure. In particular, in order to study the transient to the equilibrium configuration, the same constant value of the pressure is assigned at both ends:

$$p\,(0,t) = p_{ref} \qquad p\,(L,t) = p_{ref} \tag{3.6}$$

As a second test case, an oscillating forcing pressure is given at the right end:

$$p(L,t) = p_{ref} + \epsilon sin(2\pi\ S_t\ t) \tag{3.7}$$

where $\epsilon < p_{ref}$ and S_t are the nondimensional amplitude and the Strouhal number of the excitation, respectively.
The initial condition is chosen by considering a finite perturbed configuration of the steady Poiseuille flow, corresponding to a viscous fluid ($Re = 1$), a purely elastic wall and a non zero pressure gradient. Then the system is left evolving towards its equilibrium configuration (see (3.6)) or forced by an oscillating pressure (see (3.7)). For further details, see [15].

4 Numerical method and results

The equations of evolution of the fluid (3.1) and (3.4), the equations of the equilibrium of membrane (3.5) with the constitutive equations (2.3) model the

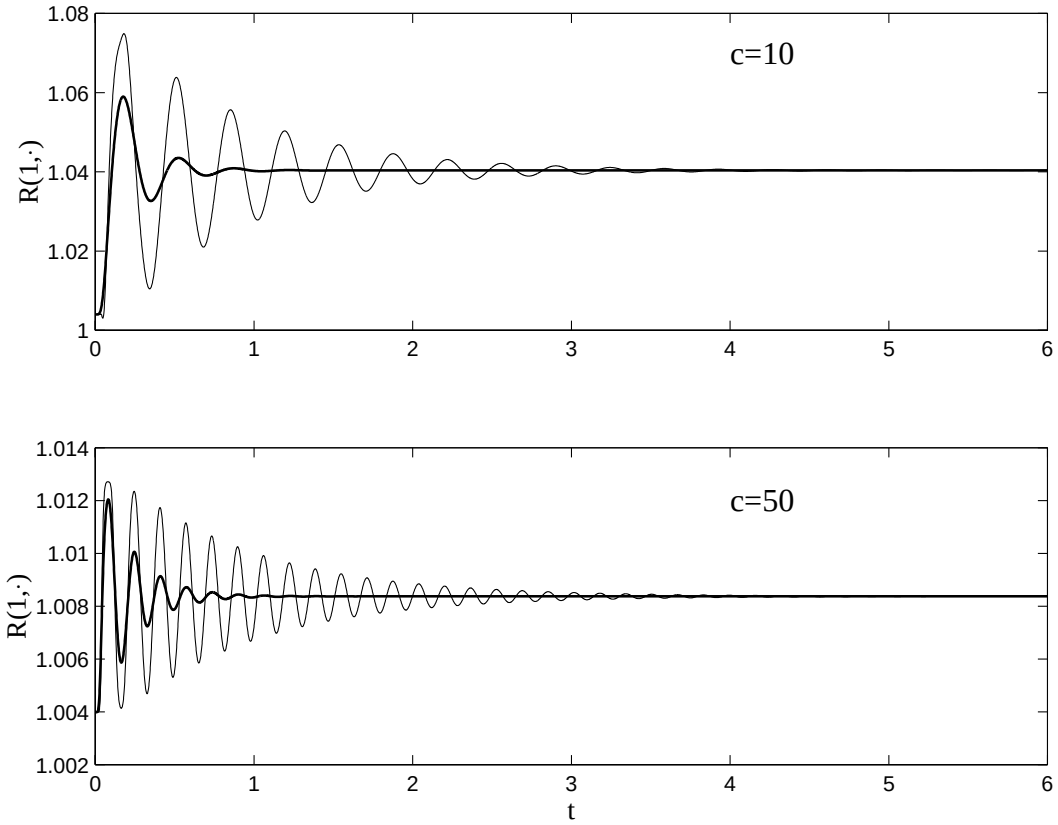

Fig. 1. Evolution of the arterial deformation $R(1,\cdot)$ for 2 values of the elasticity parameter c with the same initial data. Thin line corresponds to $\gamma = 2$, thick line to $\gamma = 8$. Note the different scale.

nonlinear interaction between the blood and the arterial wall. They are solved numerically by a second order finite difference method centered in space. Let us consider a sequence of $n+1$ equispaced grid points with $x_0 = 0$ and $x_n = L$. The spatial discretization is obtained by evaluating membrane stresses, strains, and their time derivatives (see eqns. (2.3)) at n inner points $\xi_i = \dfrac{x_i + x_{i+1}}{2}$ of a staggered grid by considering averaged neighbouring quantities. On the other hand, equilibrium equations (3.5) and fluid equations (3.1), (3.4) are computed at the $n-1$ inner points x_i. The time discretization is based on the trapezoidal formula, in such a way the global scheme is of second order in space and time. The resulting nonlinear system is solved by a globally convergent Newton type method.

Nonlinear models turn out to be very sensitive to the many material parameters which characterize the specific flow problem. The value of the reference pressure is fixed as $p_{ref} = 10$, $c_1 = 11.82$, $c_3 = 1.18$ [7], and the other parameters have been chosen around some typical values to obtain results of physiological interest and varied in a typical range to test the sensitivity of the system to their perturbation.

The problem is studied in the interval: $5 \leq c \leq 1000$: it turns out that for a larger value of c the wall increases its stiffness and the numerical problem

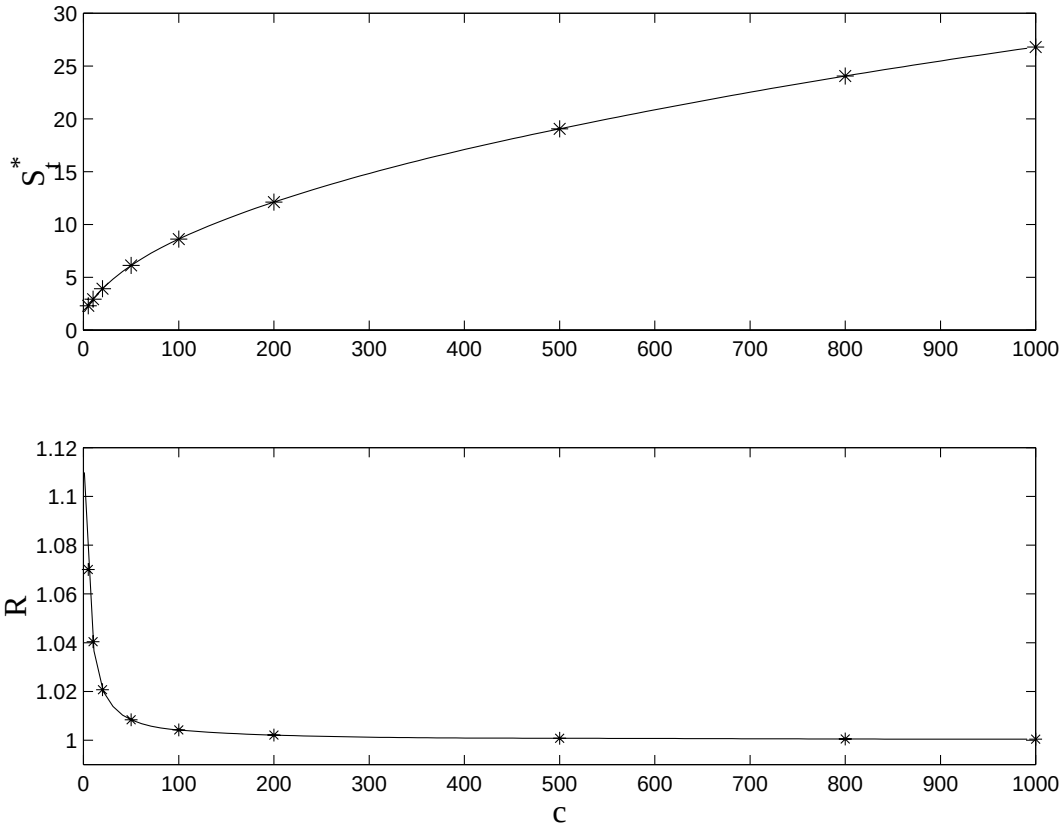

Fig. 2. Dependence of the frequency S_t^* and of the maximum arterial deformation $R(1, \nearrow)$ on the elasticity parameter c. Both are independent of the viscosity coefficient γ. Starred points are results from simulations, continuous curve is obtained by a cubic spline interpolation.

becomes harder. On the other hand, for $c < 5$ the system undergoes an unrealistic large deformation and the present model is not physically admissible. In all the experiments we selected $L = 2$, $\Delta x = 10^{-2}$ and $\Delta t = 10^{-3}$. These values guarantee the numerical stability of the system for the set of parameters considered. The accuracy of the solution is controlled since the solution corresponding to a finer grid does not reveal a different structure or unresolved patterns. Since in wave propagation phenomena the dissipative effect of the blood is a minor effect [4], in the following simulations, an inviscid fluid is considered ($f \equiv 0$ in (3.1) and $\tau = 0$ in (3.5.1)).
Firstly the system, subject to an initial deformation, is left evolving towards the equilibrium configuration obtained by imposing the same value of the pressure at the two extrema (see boundary conditions (3.6)). After an initial transient, all the variables reach asymptotically a steady state value with damped oscillations (*natural oscillations*) having exponential decay and with a c-dependent frequency S_t^* (*natural frequency*), computed by spectral analysis. Due to the elasticity of the wall, a positive deformation R and a positive flow rate u at the final state at both ends is found equal. The influence of the membrane viscosity is investigated by varying the value of γ. It turns out that the

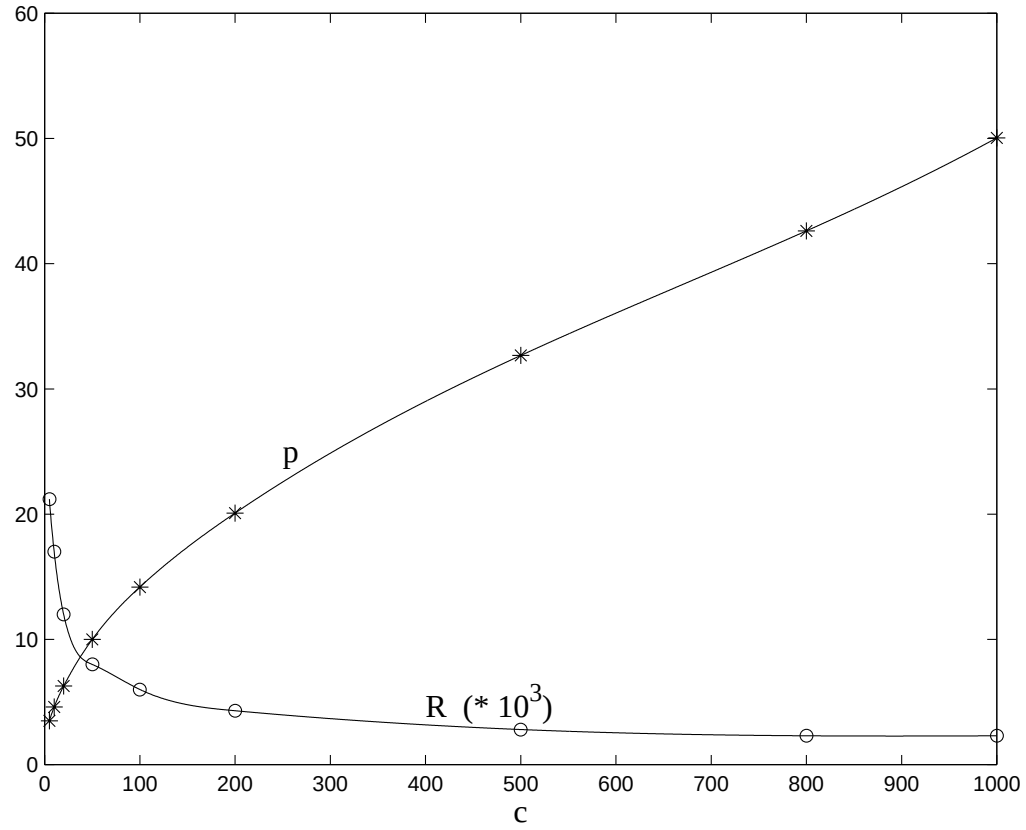

Fig. 3. Amplitude of the pressure $p(1,\cdot)$ and the arterial deformation $R(1,\cdot)$ when a forcing oscillatory pressure is assigned at the right end ($\epsilon = 1, \quad S_t = S_t^*$) as a function of the elasticity coefficient c.

attenuation factor increases with γ but is independent of c (see fig. 1). Results show, for any c, the existence of a critical value for γ, below which difficulties in convergence arise. In this case the low viscosity of the wall is unable to attenuate the natural oscillations and the steady state is never reached [15]. As a matter of fact, no stable steady solution has been found as $\gamma \to 0$. This behaviour persists either by refining the computational mesh and by changing the initial data.

On the other hand, the steady state values of the deformation decrease with c, and are independent of γ and of the initial data. Similarly, the natural frequency S_t^* increases with c but stays unchanged with γ (fig. 2) and with the initial data. The effect of the viscosity coefficient γ is to dissipate energy, to damp the oscillations (but not to modify their frequency) and is observed only in the transient regime. The longitudinal deformation S exhibits very small changes compared to the radial one and is not discussed.

By considering the importance of a pulsatile forcing, an oscillating pressure having Strouhal number S_t and amplitude $\epsilon = 1$ (10% of $p_{ref} = 10$) has been imposed at the right boundary - see boundary conditions (3.7). Even if the Strouhal number of the forcing oscillation in the physiological flow is extremely small, in this work it has been selected to be comparable with the

natural frequency S_t^* of the membrane. In this case, the flow is not damped anymore, but the persistence of steady oscillations of sinusoidal type occurs with the same input frequency. The oscillations are about the same value as that corresponding to steady state with $p_{ref} = 10$ and their amplitudes attain a maximum when $S_t = S_t^*$ (*resonance* phenomenon). The dependence of the oscillations amplitude on c is shown in fig. 3. The analysis of the radial velocity of the wall proves that the propagation features correspond to transverse waves which do not propagate along the tube and are due to the boundary conditions that generate reflections. The phenomenon is similar to that of a stretched string of a finite length with one extreme fixed and the other oscillating. Results do not differ if the excitation at the right boundary is replaced by one at the left boundary. Results agree qualitatively with those presented in [10] and show that the nonlinear character of the strain-stress function is responsible for minor changes with respect to the linear case. This is because of the small strains of the arterial motion (in the range of parameter considered: $\max_{x,t} \lambda_1 = 1.02$ and $\max_{x,t} \lambda_2 = 1.13$).
A wider analysis of the problem has been developed and more results can be found in [15].

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