A mathematical model of flow in a liquid-filled visco-elastic tube

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Abstract—*In biofluid mechanics the fluid–solid interaction is important. To this aim the propagation of waves in a distensible tube filled with a viscous fluid was studied numerically. Based on the assumption of long wavelength and small amplitude of pressure waves, a quasi-1D differential model was adopted. The model accounted for vessel wall visco-elasticity and included the wall deformations in both radial and axial directions. The non-linear problem was solved in non-dimensional form by a finite difference method on a staggered grid. The boundary conditions were for two relevant cases: natural oscillations in a deformable tube fixed at the ends and persistent oscillations due to a periodical forcing pressure. The natural frequency St* was found to vary as the square root of the elasticity coefficient K, with $0 \leq K \leq 6000$, and was not affected by the viscosity. These results highlight a strong influence of both wall visco-elasticity and blood viscosity. The natural oscillations are damped in a few time units and the damping time was found to be inversely proportional to the wall viscosity coefficient and the fluid viscosity provided an even larger damping factor.*

Keywords—*Wave propagation, Wall–fluid interaction, Visco-elasticity, Haemodynamics*

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1 Introduction

FLOWS OF viscous fluids in deformable tubes are quite common in many applications, for example, in modelling blood flow problems. Experimental evidence shows that, when an unsteady forcing perturbs a steady flow in a distensible tube, damped waves are formed and propagated downstream. In spite of many theories having been developed to explain this phenomenon, the propagation mechanism is not yet fully understood, because of the complexity of the system and of the non-linear fluid–structure interaction.

Although physiological flows are better described in multi-dimensional models, simpler 1D models give some useful hints on wave propagation and offer an easy tool to understand the basic features of the damping effect, allowing a systematic analysis of a wide range of parameters. As a matter of fact, the unidirectional nature of blood flow justifies the attempt to apply the 1D approximation to long arterial conduits. Several studies of pulse propagation in arteries have been carried out to provide some insight into the mechanical interaction between blood and vessel wall. Many of these models are concerned with an incompressible Newtonian fluid contained in a compliant tube. Moreover, most of them are based on linearised equations of motion and linearised relationships between stress and strains (LIGHTHILL, 1978; PEDLEY, 1980).

Under the simplistic assumption that the vessel wall is purely elastic, such linear models are known to produce shocklike flow patterns in the propagating pulses that are not observed under physiological conditions (ANLIKER *et al.*, 1971; OLSEN and SHAPIRO, 1967).

On the other hand, many authors have pointed out that the blood vessel walls are non-linear, visco-elastic and anisotropic (ROCKWELL *et al.*, 1974). By including an appropriate mathematical model for the visco-elastic properties of the wall, the applicability of the one-dimensional theory can be extended. The damping resulting from visco-elasticity inhibits the sharp peaks of the pressure and flow pulses and flattens the abrupt rise of wave fronts. Thus, such models lead to more realistic results when a comparison with experimental data is carried out (HORSTEN *et al.*, 1989).

In most of these papers, however, the distensibility of the arterial wall is modelled by non-linear algebraic relationships between the cross-section of the tube and the transmural pressure (MORGAN and PARKER, 1989) and sometimes is a function of the frequency (REUDERINK *et al.*, 1989): in some cases, the integration is possible along the characteristics, and the wave celerity is expressed in explicit form (ROCKWELL *et al.*, 1974).

In contrast with this approach, which lacks a reliable mechanical justification, a constitutive strain–stress equation modelling the mechanical properties of the arterial wall has been proposed (HUMPHREY, 1995). A recent study has been carried out on a two-dimensional flow in a rigid channel, where a part of the wall was replaced by a visco-elastic membrane subject to deformation (PEDRIZZETTI, 1998). In this paper, a similar but simpler one-dimensional (1D) model is demonstrated to be sufficient to describe propagative phenomena. The wall–fluid interaction in arterial flow problems and the role played by the constitutive equation of the vessel, which includes a visco-elastic term, are examined.

The motivation for this study lies in the possibility of understanding the evolution of small flow disturbances induced by a local vessel insertion or generated by a pathological state.

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The analysis of 1D models has been shown to provide much insight into the mechanism and the alteration of pulse propagation (REUDERINK *et al.*, 1989). The model used is based on quasi-1D flow equations, coupled with the massless membrane equilibrium equation. A strain–stress constitutive equation for the wall is also provided, allowing for wall deformations in both longitudinal and circumferential directions. Although blood flow in large arteries is inertia dominated, the limiting cases of very small and very large Reynolds numbers are also considered, so that we can understand the important role of the dissipative mechanism induced by viscous friction and giving rise to damped waves when the system evolves to the steady state. The critical parameters involved in the mechanics and responsible for possible numerical instability are pointed out.

2 Mathematical formulation

Let us consider the flow of a homogeneous, incompressible Newtonian fluid in a cylindrical distensible tube, of circular cross-section and of radius R_0 and length L. As we are interested in the flow pulse propagation, the assumption of one-dimensional flow is a valid approximation 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.

2.1 *Fluid equations*

Assuming that the flow is axisymmetric and indicating with x and r the longitudinal and radial co-ordinates, respectively, let us consider the x-component of the momentum equation

$$\frac{\partial w}{\partial t}+w\frac{\partial w}{\partial x}=-\frac{1}{\rho}\frac{\partial p}{\partial x}+\frac{\nu}{r}\frac{\partial}{\partial r}\left(r\frac{\partial w}{\partial r}\right) \tag{1}$$

where w is the axial component of the velocity, ν is the kinematic viscosity, and p is the transmural pressure. The diffusive term $\partial^2 w/\partial x^2$ and the other velocity components are comparatively small and have been neglected (PEDLEY, 1980).

Let us define the flow rate and the averaged velocity over a cross-section $A=\pi R^2$, respectively, as

$$Q=2\pi\int_0^R wr\,dr \qquad u=\frac{Q}{A}$$

and let us introduce a set of non-dimensional variables

$$\begin{aligned} x&\rightarrow\frac{x}{R_0} \qquad r\rightarrow\frac{r}{R_0} \qquad t\rightarrow\frac{tU_0}{R_0}\\ u&\rightarrow\frac{u}{U_0} \qquad p\rightarrow\frac{p}{\rho U_0} \end{aligned} \tag{2}$$

where U_0 is a characteristic velocity, and R_0 is a reference radius.

By integrating (1) over the cross-section A, we obtain the quasi-1D non-dimensional form of the momentum equation

$$\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial x}=-\frac{\partial p}{\partial x}+f \tag{3}$$

where f is the friction term.

If we locally approximate the friction term in (3) by that corresponding to the steady Poiseuille flow in a tube of radius R, then

$$f\simeq-\frac{8u}{\mathrm{Re}\,R^2} \tag{4}$$

where $\mathrm{Re}=U_0R_0/\nu$ is the Reynolds number. Consequently, the wall shear stress is approximated by

$$\tau=\mu\left.\frac{du}{dr}\right|_R\simeq-\frac{4u}{\mathrm{Re}\,R} \tag{5}$$

Strictly speaking, (4) and (5) hold for a steady flow in a rigid tube, but, as we are interested in the average effect of pulse propagation, they are reasonable when $\partial R/\partial x\ll 1$ and for quasi-steady flows. Actually, (4) and (5) are considered acceptable by many authors in one-dimensional averaged flows (ANLIKER *et al.*, 1971; MORGAN and PARKER, 1989; PORENTA *et al.*, 1986). Moreover, as, in larger arteries, Re is quite large, τ and f are both extremely small and are negligible in most applications.

In a distensible tube, the continuity equation reads

$$\frac{\partial A}{\partial t}+\frac{\partial(Au)}{\partial x}=0 \tag{6}$$

or, alternatively,

$$\frac{\partial R}{\partial t}+\frac{R}{2}\frac{\partial u}{\partial x}+u\frac{\partial R}{\partial x}=0 \tag{7}$$

2.2 *Wall equations*

To close the system in (3)–(7), an algebraic relationship relating the local transmural pressure and the radius under static conditions

$$p=p(R,x)$$

(known as the *local tube law*) is required. Many tube laws have been proposed in arterial mechanics to model the deformation as a consequence of a given pressure (*compliance*) (see PORENTA *et al.* (1986)). In most of them, the pressure acts as a loading term for the radial displacement, but shear stress along the wall surface is neglected (*independent ring models*).

Here, we follow another approach that relies on mechanical arguments and is particularly suited for time-dependent flows. Despite its thickness, the vessel wall is modelled using membrane theory (TIMOSHENKO, 1940). This is a mathematical model of a 2D shell, with a negligible mass compared with that of the fluid and without bending forces. It is subject to stresses in the tangential plane that are assumed to be averaged across the thickness (HUMPHREY, 1995). Thus, when forces act on the membrane, it deforms and reaches an equilibrium state. Let us indicate by $R(x,t)$ and $S(x,t)$ the Eulerian counterparts of the Lagrangian co-ordinates of a particle of the membrane, as in PEDRIZZETTI (1998). The fluid-membrane equilibrium equations in tangential and normal directions are provided in PEDRIZZETTI (1998) and are rewritten here for completeness

$$\begin{aligned} &R'(T_1-T_2)+RT_1'=\tau R(1+R'^2)^{1/2}\\ &\frac{-R''}{(1+R'^2)^{3/2}}T_1+\frac{1}{R(1+R'^2)^{1/2}}T_2=p \end{aligned} \tag{8}$$

where T_1 and T_2 are the non-dimensional membrane stresses in the meridional and circumferential directions, respectively, and τ is the shear stress exerted by the viscous fluid on the wall (see (5)). The static membrane equations (8) are used to model the equilibrium state of the wall each time. To define the constitutive equation for the wall, let us consider the principal deformation ratios in the tangential plane, defined as

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

where R_u is the undeformed radius.

The adequate mechanical characterisation of blood vessels is an important prerequisite for a quantitative description of blood flow, mostly in wave propagation phenomena. The properties of

vascular tissues are highly non-linear, and many models have been developed for modelling the arterial wall dynamics in physiological and pathological conditions (HUMPHREY, 1995). However, in a normally stressed vessel, the radial deformation around the equilibrium configuration is quite small (typically, it does not exceed 10%), and a linear strain–stress law around it is likely to be used.

To characterise the stresses T_1 and T_2 in (8), in the limit of small deformations, we adopted a linear elastic 2D model. This constitutes a basic choice that characterises an elastic behaviour, although other possible non-linear expressions for the constitutive equation are possible (KYRIACOU and HUMPHREY, 1996). On the other hand, many authors have pointed out the importance of visco-elasticity in modelling arterial walls. REUDERINK *et al.* (1989) found that neglecting visco-elasticity generates an underestimation of both phase velocity and damping. Generally, a visco-elastic wall model yields numerical results closer to measurements than an elastic one, and a dissipative wall is more effective than a viscous fluid in eliminating the high-frequency oscillations. The damping resulting from visco-elasticity inhibits sharp peaks of the pressure and of flow pulses and leads to more realistic results when a comparison with experimental data is carried out (HORSTEN *et al.*, 1989).

As a preliminary study, we consider a linearly varying viscous term and we add it to the elastic part, obtaining the following strain–stress relationship:

$$T_1(\lambda_1, \lambda_2, \dot{\lambda}_1, \dot{\lambda}_2) = K\left(\lambda_1 + \frac{\lambda_2}{2} - \frac{3}{2}\right) + C\left(\dot{\lambda}_1 + \frac{\dot{\lambda}_2}{2}\right)$$

$$T_2(\lambda_1, \lambda_2, \dot{\lambda}_1, \dot{\lambda}_2) = K\left(\lambda_2 + \frac{\lambda_1}{2} - \frac{3}{2}\right) + C\left(\dot{\lambda}_2 + \frac{\dot{\lambda}_1}{2}\right) \quad (9)$$

In (9), $K = Eh/\rho R_0 U_0 > 0$ is a non-dimensional elasticity coefficient, where E is the Young's modulus, and h is the undeformed arterial thickness, $C > 0$ is a wall viscosity coefficient, and the dot denotes a time derivative (PEDRIZZETTI, 1998).

The relationships of (9) 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, although by a different amount in each of the two directions.

Although the inertia of the membrane is neglected and a general theoretical framework is still lacking, in the 1D case studied here, the simple functional dependence strain–stress in (9) 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, (9) means that the membrane does not respond instantaneously to forces, as would a purely elastic body, but with a dissipative mechanism, as would a visco-elastic material.

Note that (3) and (7) depend explicitly on time, whereas the membrane equilibrium equations of (8) depend on time through the dependence of the stresses T_1 and T_2 on the strain rates in (9).

The boundary conditions are imposed by considering the wall fixed at the two ends; that is

$$R(0, t) = 1 \quad S(0, t) = 0$$
$$R(L, t) = 1 \quad S(L, t) = L_u \quad (10)$$

where L_u is the length of the undeformed membrane.

Following the experimental set-up of a distensible tube fixed at the two extrema, only the values of $p(0, t)$ and $p(L, t)$ are assigned at both boundaries. This approach is different from that in PEDRIZZETTI (1998), where the flow rate value is assigned at one of the boundaries, together with the value of the pressure. In particular, to study the transient to the equilibrium configuration, the same value of the pressure is assigned at both ends

$$p(0, t) = p_{ref} \quad p(L, t) = p_{ref} \quad (11)$$

As a second test case, an oscillating forcing pressure is given at the outlet

$$p(L, t) = p_{ref} + \varepsilon \sin(2\pi St t) \quad (12)$$

where $\varepsilon < p_{ref}$ and $St = R_0/U_0 T$ are the non-dimensional amplitude and the Strouhal number (with T the period) of the excitation, respectively. The use of boundary conditions based on such physiological considerations is being developed (PONTRELLI, 2002).

The initial condition is chosen by considering a finite perturbed configuration of the steady flow, corresponding to a purely elastic wall and a constant pressure gradient. Then the system is left to evolve towards its equilibrium configuration (see (11)) or forced by an oscillating pressure (see (12)).

3 Numerical method and parameters

The equations of evolution of the fluid, (3) and (7), the equation of the equilibrium of the membrane (8) and the constitutive equations (9) are discretised by centred second-order finite differences in space.

Let us consider a sequence of $n + 1$ equispaced grid points, with $x_0 = 0$ and $x_n = L$. The spatial discretisation is obtained by evaluating membrane stresses, strains and their time derivatives (see (9)) at n inner points $\xi_i = (x_i + x_{i+1})/2$ of a staggered grid by considering averaged neighbouring quantities.

On the other hand, the equilibrium equations (8) and fluid equations (3)–(7) are computed at the $n - 1$ inner points x_i. The time discretisation is based on the trapezoidal formula, in such a way that the global scheme is of second order in space and time (FLETCHER, 1988). The resulting non-linear system is solved by a globally convergent Newton-type method.

Non-linear models turn out to be very sensitive to the choice of the material parameters that characterise the specific flow problem. The reference values used in (2) are $U_0 = 50\,\mathrm{cm\,s^{-1}}$ and $R_0 = 0.5$ cm. The non-dimensional p_{ref} has been fixed as $p_{ref} = 10$, and the other parameters have been chosen around some typical values to obtain results that are of physiological interest and varied in a typical range to test the sensitivity of the system to their perturbation.

The value of the elasticity parameter K must be larger than p and p/K is approximately proportional to the radial deformation of the membrane. In a physiological context, $h/R_0 \approx 0.1$ (NICHOLS and ROURKE, 1990), and consequently $K \approx 200$. Nevertheless, to investigate better the capabilities of the model and the functional dependence of the solution, the problem is studied for K ranging in the wider interval $200 \leq K \leq 6000$: it turns out that, for a larger value of K, the stiffness of the wall becomes extremely high. On the other hand, for $K < 200$, the system undergoes an unrealistically large deformation, and the present model is not representative of the physics (see Section 4).

In all 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 was validated by considering a finer grid that did not reveal any different solution structure or unresolved patterns. For all the experiments, the same initial data set was chosen.

Although the Strouhal number of the forcing in the physiological flow is extremely small ($St \approx 0.01$), in this model study we consider it to be comparable with the natural frequency St^* of the membrane (see following Section). This is computed by spectral analysis when 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.

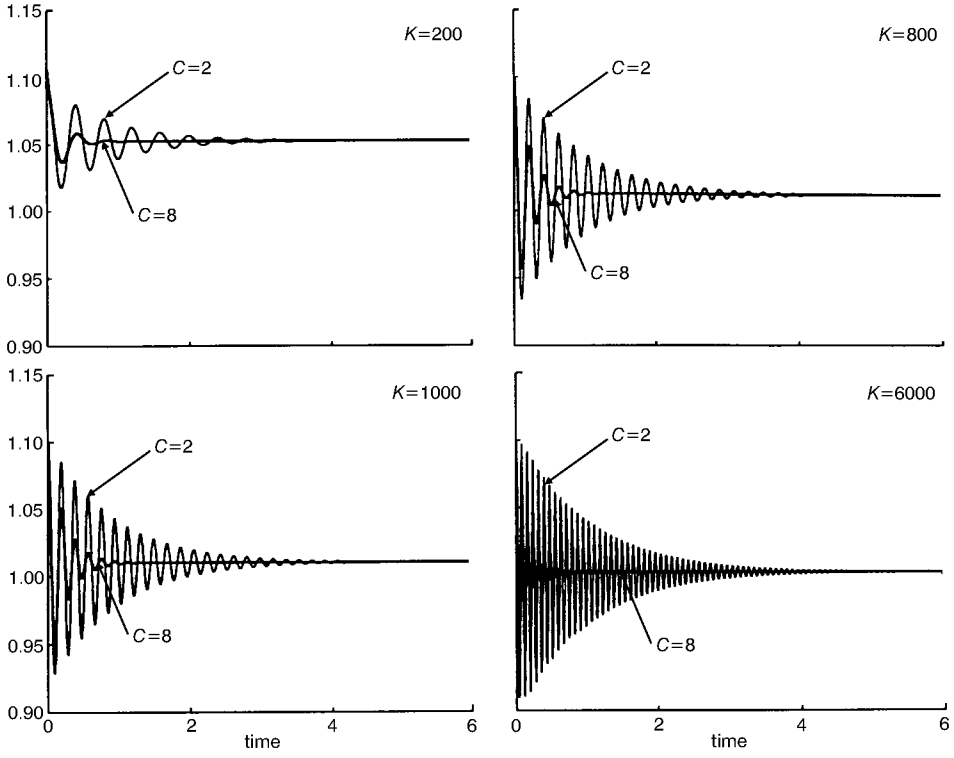

Fig. 1 *Evolution of R(1, ·) for values of K.*

4 Results and discussion

In this Section, the results of several numerical tests are reported to reproduce realistic flow fields close to the biomechanical applications. When the system is left evolving from an initial perturbation towards equilibrium, all the variables, after an initial transient, asymptotically reach a steady-state value with damped oscillations having an exponential decay and with a K-dependent frequency St^* (natural frequency). Owing to the elasticity of the wall, a positive deformation and a positive flow rate at the final state are found to be equal at both ends. If not otherwise stated, a convection-dominated fluid is considered ($f \equiv 0$ in (3) and $\tau = 0$ in (8)).

As no quantitative data for the dissipation of the wall in dynamic experiments are available, the influence of the membrane viscosity is investigated by varying the value of C. It turns out that the attenuation factor increases with C but is independent of K (see Fig. 1). For $C \to 0$, the solution is not damped and tends to that of a system with a purely elastic wall.

To understand better the effect of the reference pressure, the value of p_{ref} is varied in the range [0.5, 200], for two assigned values of K and C. Instability develops for larger pressures as a consequence of deformations that are too large. In such a case, the 1D model is not adequate to describe the phenomenon. The value of u does not change with p_{ref}, whereas the deformation is proportional to it, and a slight increase in the natural frequency is observed (see Fig. 2). However, the convergence of the system in the transient case critically depends on the initial data.

On the other hand, the steady-state values of the deformation decrease with K and are independent of C and of the initial data. Similarly, the natural frequency St^* increases with K (at the first instance, a functional dependence $St^* \propto \sqrt{K}$ is assumed (LIGHTHILL, 1978)), but stays unchanged with C (see Fig. 3)

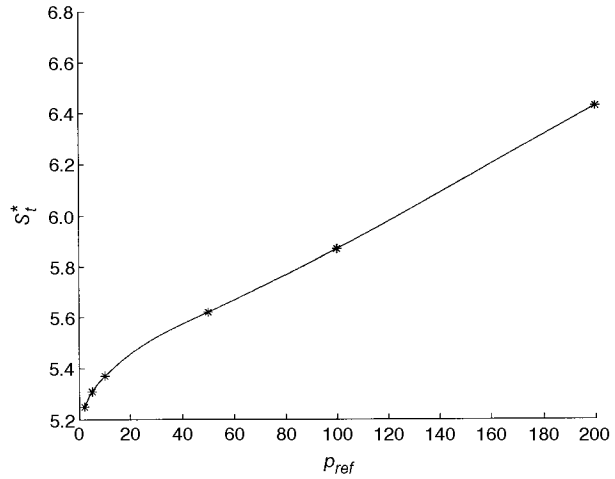

Fig. 2 *Dependence of St* on p_{ref} in case where $K = 1000$, $C = 2$. (*) results from simulations; continuous curve is obtained by cubic spline interpolation*

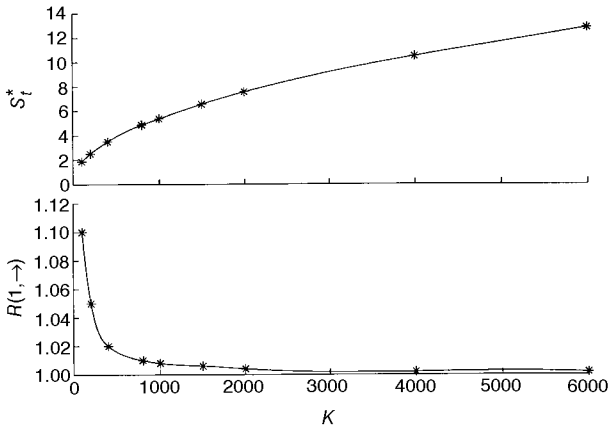

Fig. 3 *Dependence of St* and R(1, ↗) on parameter K. Both are independent of C. (*) results from simulations; continuous curve is obtained by cubic spline interpolation*

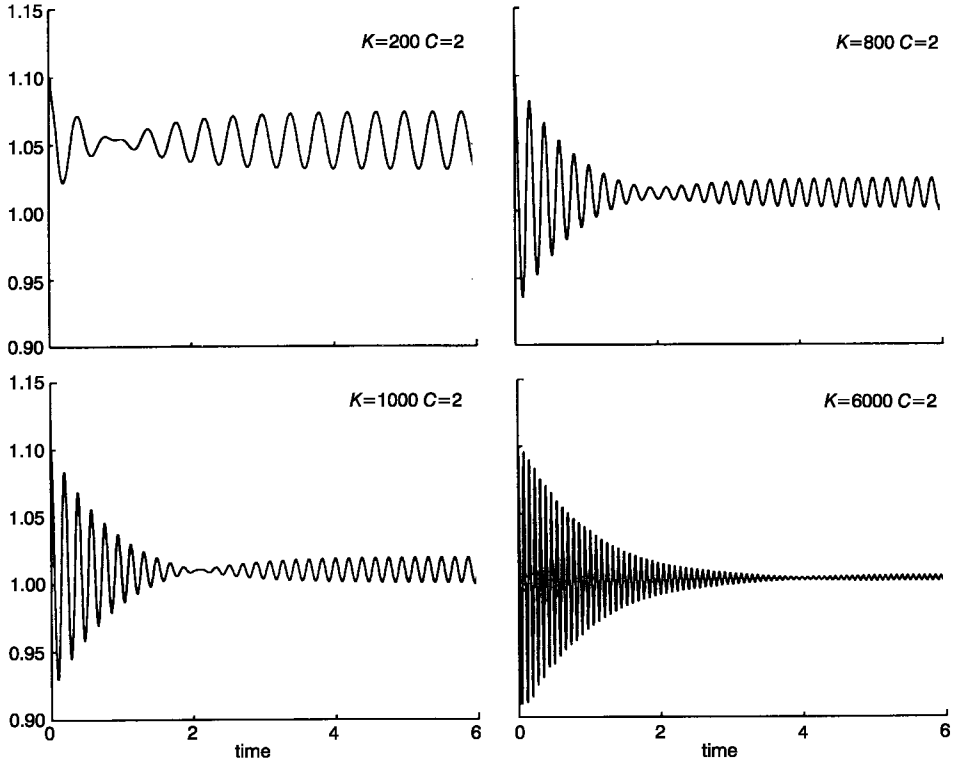

Fig. 4 *Persistence of oscillations of R(1, ·) at centre of tube when forcing oscillatory pressure is applied at right end (ε = 1, St = St* in (12)) (resonance case)*

and with the initial data. The effect of the viscosity coefficient C is to dissipate energy and to damp the high-frequency oscillations (but not to modify their frequency) and is observed only in the transient regime. The longitudinal displacement S turns out to be very small (three orders of magnitude lower) compared with R and is not displayed. However, the influence of such a variable becomes more relevant when the fluid friction at the wall is considered.

4.1 *Oscillating forcing*

The boundary condition (12) reflects the basic physiological waveform at outlet. Results from simulations with several Strouhal numbers and amplitude $\varepsilon = 1$ (10% of $p_{ref} = 10$) show that the flow no longer decays to a steady solution, but there persist steady oscillations of sinusoidal type occuring at the same frequency as the input forcing (a blow up of the solution is obtained with larger values of ε depending on the value of K). The oscillations are of about the same value as that corresponding to steady state with $p_{ref} = 10$, and their amplitudes attain a maximum when $St = St^*$ (*resonance* phenomenon) (Figs 4 and 5). The analysis of the radial velocity of the wall proves that the propagation features correspond to transverse waves that do not propagate along the tube and are due to the boundary conditions that generate reflections and spurious effects (Fig. 6). The boundary conditions (10) of Dirichlet type may be unrealistic in physiological flows, but reproduce a physical experiment. The phenomenon is similar to that of waves propagating in a stretched string of a finite length. The results do not differ if the excitation at the right boundary is replaced by one at the left boundary.

4.2 *Influence of fluid viscosity*

To understand the role of viscous and inertial terms in the fluid motion and its interaction with the wall, results of an inertia-dominated flow (i.e. with $\mathrm{Re} \to \infty$) and of a very viscous flow (i.e. with $\mathrm{Re} = 1$ in (4) and (5)) (and with all the other parameters fixed) have been compared.

Such a comparison shows that the mean velocities first exhibit a stronger damping to zero in the transient and then a rise to the steady-state values (see Fig. 7, where a non-monotonic envelope is visible). The steady-state deformation value and the oscillation frequency remain the same in both cases, even under oscillating pressure, and the wave celerity changes with Re only when K is small. Therefore it appears that the frequency of oscillations is an intrinsic characteristic of the wall and is not greatly influenced by the fluid properties. Nevertheless, the presence of fluid viscosity allows for a larger value of the amplitude ε of the forcing pressure, without the vessel blowing up. As reported elsewhere, the attenuation of the wall viscoelasticity turns out to be a dominant effect in larger vessels, whereas the damping induced by the fluid viscosity becomes more important in small vessels (PONTRELLI, 1998).

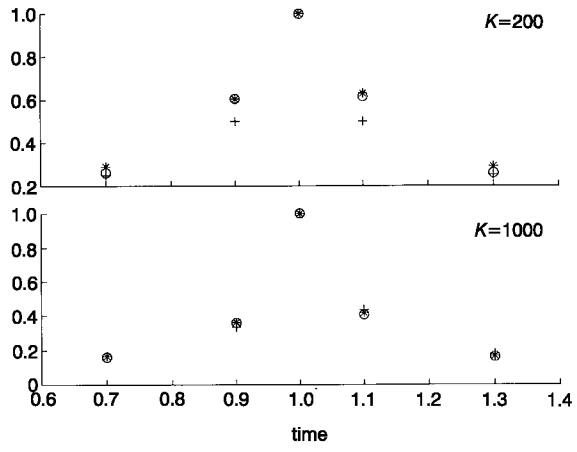

Fig. 5 *Ratio of forcing and natural amplitudes against St/St*. (⋆) upstream mean velocity; (○) pressure at centre of tube; (+) deformation at centre of tube. Ratio is maximum when St = St* (resonance case)*

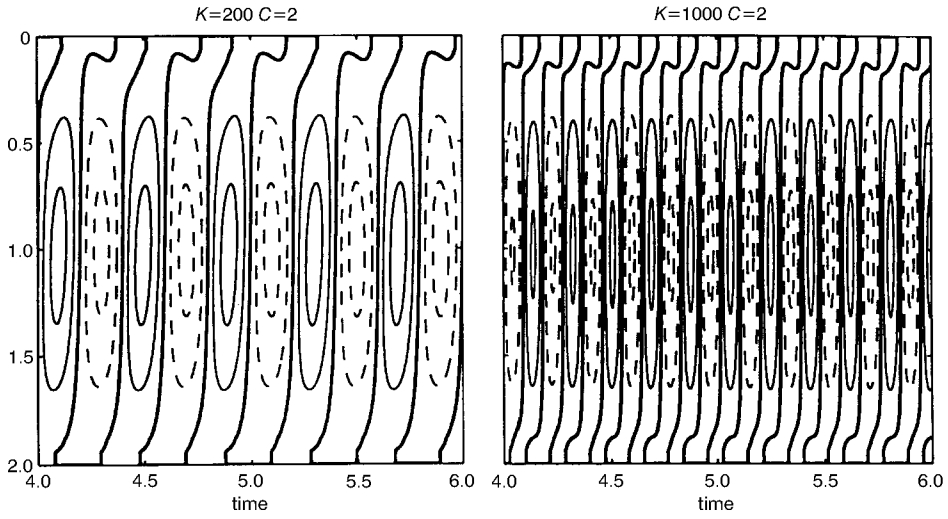

Fig. 6 *Space–time evolution of membrane radial velocity in case of resonance ($\varepsilon = 1$, $St = St^*$). (—) Zero level; (—) positive levels; (---) negative levels. Level step is 0.01 in both plots*

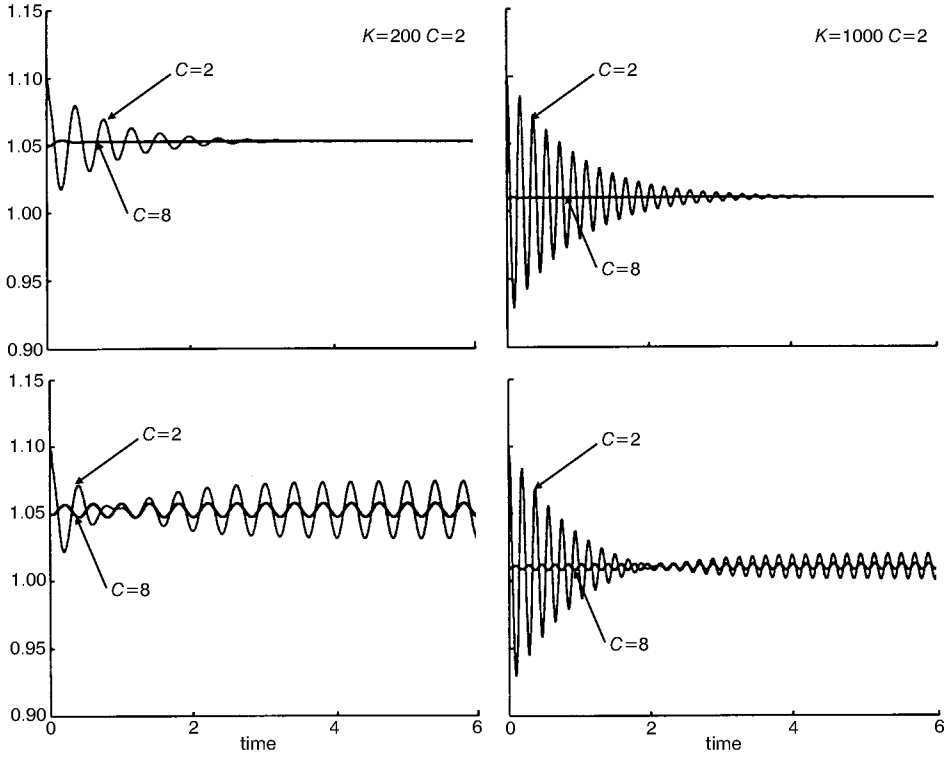

Fig. 7 *Evolution of $R(1, \cdot)$ in cases of (a) viscous fluid ($Re = 1$) and of (b) inertia dominated fluid ($Re \to \infty$). Steady-state case (above) and oscillating forcing case (below) are compared*

5 Conclusions

A coupled wall–fluid model for studying the unsteady flow of a viscous fluid in a visco-elastic tube has been presented, with application in arterial flows. The unidirectional nature of blood flow suggests modelling the propagating flow pulses with a quasi-1D mathematical approximation, which is well supported by measurements. However, this formulation yields realistic results provided that the wavelength is long and the wave amplitude small compared with the mean radius of the tube.

A linear constitutive relationship for the vessel wall that depends on both the strain and the strain rate is presented. The model includes the combined effects of the visco-elasticity of the solid tube and the viscosity of the blood. The effect of the elasticity parameter is related to the frequency of oscillations in the transient, and the influence of viscosity parameter is to attenuate the high-frequency oscillations, to reduce the tendency of shock formation, as present in a purely elastic wall model, and to counterbalance possible instability phenomena.

Although some simplifying hypotheses are necessary to assess the theory, the model study presented here is able to describe the basic physical mechanism of 1D pulse propagation through the non-linear interaction between the fluid and the wall. However, the numerical values of the elastic and viscous coefficients appearing in the constitutive equation, as well the boundary conditions, are critical and need to be carefully identified by comparing numerical results with experimental measurements.

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