A numerical study of the arterial fluid-wall interaction

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Introduction

Models of viscous fluids in compliant tubes are often used for simulating blood flow problems. However, in spite of many theories having been developed to explain the pressure waves in the vascular system, the propagation mechanism is not yet fully understood, because of the complexity of the system and of the nonlinear fluid-structure interaction. Although physiological flows are better described in multi-dimensional cases, simpler 1D models give some useful hints on the wave propagation and offer an easy tool for understanding the basic features of the damping effect, allowing a systematic analysis on a wide range of parameters.

The purpose of this work is to examine the wall-fluid interaction in arterial flow and the role played by the constitutive equation of the vessel. Another goal is the possibility to analyze the evolution of small flow disturbances induced by a local vessel insertion or generated by a pathological state.

Methods

The fluid model is based on the nondimensional quasi-1D momentum equation:

\[ \frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} = -\frac{\partial p}{\partial x} + f \]

with \( u \) the cross section averaged velocity, \( p \) the transmural pressure, \( f \) a friction term, and the continuity equation:

\[ \frac{\partial R}{\partial t} + R \frac{\partial u}{\partial x} + u \frac{\partial R}{\partial x} = 0 \]

where \( R \) is the radius of the vessel (Fung, 1984).

Despite its thickness, the vessel wall is modelled with the theory of the membranes. This is a mathematical model of a shell with a mass negligible compared with that of fluid and without
bending forces. When a shear stress \( \tau \) and a pressure \( p \) act on the membrane, the latter deforms and reaches an equilibrium state expressed by the following balance equations:

\[
R(T_1 - T_2) + RT_1' = \tau R(1 + R^2)^{1/2} \\
\frac{-R'}{(1 + R^2)^{1/2}}T_1 + \frac{1}{R(1 + R^2)^{1/2}}T_2 = p
\]

where \( T_1 \) and \( T_2 \) are the membrane stresses along the longitudinal and circumferential directions respectively and the prime denotes \( x \)-derivative.

To define a constitutive equation for the vessel wall, we used the strain-energy density function modelling the biomechanical properties of the arterial wall recently proposed by Shah and Humphrey (1999):

\[
w = c_0 (e^0 - 1) \quad Q = c_1 (E_1^2 + E_2^2) + 2c_3 E_1 E_2
\]

where \( c_0, c_1, c_2 \) are material parameters and \( E_k = \frac{1}{2} (\lambda_k^2 - 1) \quad k = 1, 2 \) are the principal Green strains. In an incompressible and isotropic elastic material, the stress components are given by differentiation of \( w \):

\[
T_1(\lambda_1, \lambda_2) = \frac{\lambda_1}{\lambda_2} \frac{\partial w}{\partial E_1} = \frac{1}{\lambda_2} \frac{\partial w}{\partial \lambda_1} \quad T_2(\lambda_1, \lambda_2) = \frac{\lambda_2}{\lambda_1} \frac{\partial w}{\partial E_2} = \frac{1}{\lambda_1} \frac{\partial w}{\partial \lambda_2}
\]

with \( \lambda_1 \) and \( \lambda_2 \) are the principal deformation ratios.

On the other hand, many authors have pointed out that the blood vessel walls are viscoelastic and anisotropic. Thus, such models lead to more realistic results when a comparison with experimental data is carried out (Horsten and al., 1989). By generalizing the strain-stress relationship of a pure elastic material, the arterial viscoelastic behaviour is modelled by:

\[
T_1 = \frac{1}{\lambda_2} \frac{\partial w}{\partial \lambda_1} + \gamma \left( \dot{\lambda}_1 + \frac{\dot{\lambda}_2}{2} \right) \\
T_2 = \frac{1}{\lambda_1} \frac{\partial w}{\partial \lambda_2} + \gamma \left( \dot{\lambda}_2 + \frac{\dot{\lambda}_1}{2} \right)
\]

where \( \gamma \) is an arterial viscosity coefficient, and dot stands for time derivative (Pedrizzetti, 1998).

The system of the above differential equations models the fluid-wall interaction and describes the dynamics of an arterial segment: it is supplemented by a simple sinusoidal boundary condition downstream and coupled with a Windkessel type model downstream (Segers and Verdonck, 1998). The full problem is solved by a second order finite difference method on a staggered grid (Fletcher, 1988).
Results & Discussion

The dependence on the many parameters and their influence on the wave propagation has been analyzed by some numerical simulations. The persistence of pulsatile oscillations with the same characteristics of the incoming pulse has been found. The analysis of the radial velocity proves that the propagation features correspond to travelling waves which propagate along the vessel.

The limiting cases of very small and very large Reynolds number are considered, in order to understand the important role of the dissipative mechanism induced by viscous friction. The effect of the wall viscosity is found to damp the amplitude of oscillations, to reduce the tendency of shock formation, and to counterbalance possible instability phenomena. The wall viscosity does not affect the wave frequency in the typical physiological range.

To simulate the influence of a stent insertion or of a local wall stiffening on the wave propagation, the effect of a limited rising of the elasticity coefficient along the vessel is also studied.

The method can be useful to get simple information and estimates on some relevant averaged quantities of clinical interest. The geometrical, physical and biomechanical parameters need to be carefully identified according to a specific flow problem.

References


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