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Nonlinear Model of Blood Flow Through Stenosed Coronary Arteries

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Abstract
Blood flow and pulse wave propagation in the blood vessels are usually studied on the incompressible linearized axsymmetric Navier-Stokes equations (Womersley model) coupled with linear and nonlinear viscoelastic equations for the vessel walls. A brief review of the nonlinear models is given. Blood flow and wave propagation through the stenosed artery terminated by a viscoelastic chamber with different resistivity and compliance has been considered. The material parameters of the wall corresponded to healthy elastic and degenerated compliant human arteries. Both 2d linearized and 1d nonlinear models have been applied for numerical solution of the problem. The computational results for the pressure P(t), flow U(t) and diameter d(t) oscillations have been compared to the measurement data. It is shown that the dynamics of the fluid-filled vessels with healthy elastic walls is satisfactory described by the linearized equations while for the age-related degenerative walls, stenosed or diluted vessels the nonlinear models are preferable.

Keywords
Biomechanics, mitral valve, chordae rupture, mitral insufficiency, surgery planning

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Introduction

Development of a detailed mathematical model of human blood circulation system is a challenging problem of the Virtual Cardiovascular Human Project that allows *in silico* medical diagnostics, planning of therapy and surgery, estimation of the results of the treatment and rehabilitation procedures basing on the patient-specific geometrical model built on the measurement data (ultrasound or computed thomography) [1,2]. The problem is also important for different technical systems dealing with suspensions, polymer solutions and other complex fluids exhibiting Newtonian behaviour at the larger scales and non-Newtonian properties at smaller scales. Solutions of the linear and linearized models could be obtained as expansions that allow detailed analysis of their behaviour at different model parameters. Nonlinear models could be solved numerically, still that produces the time-consuming numerical procedures for the detailed models of the circulatory system composed by $>10^8$ larger, medium and smaller arteries and veins [1,2]. Discussion on application of linear vs nonlinear models is not finished yet. In this paper the computation results obtained from the different models are compared to the measurement data.

1. Review of the linear and nonlinear mathematical models

Flows of the multiphase and multicomponent fluids in the systems of tubes, ducts, and reservoirs have been studied on different mathematical models. Biological fluids are the most complex in nature as well as many technical fluids exhibiting viscoelasticity, anisotropic viscosity, shear thinning and thickening, and related phenomena. The space scale determines importance of the non-Newtonian properties while the time scale defines the influence of the local flow characteristics on the long time behavior and properties, like sediment accumulation, crystallization, and phase transitions. In the

biofluids the most important unsolved phenomena is blood clot or atherosclerotic plaque formation which lasts from several months to years, while the direct negative influence of the chemical components and hydrodynamic factors (blood pressure, local pressure oscillations and wall shear stress) are at the heart beat scale (t<1s).

The detailed 3D modelling of the circulatory system is still impossible due to incredible computer power needed and the number of the model parameters to be specified. The reasonable simplification is incorporation of the more detailed model of a part of the system into a simplified model of the general circulation model. The 3D models of the aortic arch, carotid and cardiac vessels [1] combined with 0D model of the heart, larger and smaller vessels are developed. The boundary condition problem at the interfaces of the 3D and 0D models is discussed in [2]. Some coupling schemas for the flows of suspensions in the complex multiscale systems of tubes are presented in fig.1.

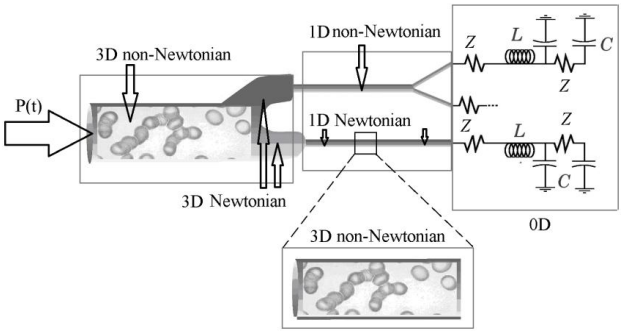

Figure 1. A schema of coupled 3D, 1D and 0D models.

1.1 0D models of elastic/viscoelastic chamber

0D models are based on the ordinary differential equations of the mechanic-electric analogy (Windkessel model) [3]

$$K_j \frac{dP_j}{dt} = Q_j - Q_{j+1}$$
$$L_j \frac{dQ_j}{dt} = P_j - P_{j-1} - Z_j Q_j \tag{1}$$

where P_j, Q_j and Q_{j+1} are pressure, inflow and outflow rates in the j-th tube, K_j is the wall compliance, L_j is the fluid inertia, Z_j is the tube resistivity to the flow.

When a single non-inertial elastic chamber terminated by a resistivity element (j=1) is considered, the solution of (1) can be written as

$$P(t) = e^{-t/ZK}\left(P_0 + \int_0^t Q_{in}(\tau) e^{-\tau/ZK} d\tau\right) \tag{2}$$

where $P_0 = P(0)$. Generally speaking $Z = Z(\mu_0)$, μ_0 is the blood viscosity due to the non-Newtonian properties of blood in the smaller arteries.

A nonlinear viscoelastic 0D model for the bioactive wall has been proposed in [4] in the form

$$\tau_\upsilon \frac{dV}{dt} + V = F(p) + \tau_p \frac{dP}{dt} + \Lambda(C_i) \tag{3}$$

where F(P) is the experimental dependence for the passive wall, $\Lambda(C_i)$ is the function describing the active changes of the chamber depending on the regulating factors C_i, i=1,2,3… , $\tau_{v,p}$ are coefficients. In this case the model can be reduced to the nonlinear ordinary differential equation

$$\frac{d^2P}{dt^2} + f_1\left(\frac{dP}{dt}\right)^2 + f_2\frac{dP}{dt} + f_3 P = \tilde{Q}(t) \tag{4}$$

where $f_{1,2,3}$ are known functions.

0D model does not describe the wave propagation at a constant speed which makes a problem of coupling with other models.

1.2 Nonlinear 1D models

1D Euler model was generalized for the viscous fluid and nonlinear elastic wall in the form [5,6]

$$\frac{\partial Q}{\partial t}+\frac{\partial}{\partial x}\left(\frac{\alpha Q^2}{A}\right)+\frac{A}{\rho_f}\frac{\partial P}{\partial x}=-k(\mu)\frac{Q}{A}$$
$$\frac{\partial A}{\partial t}+\frac{\partial Q}{\partial x}=0, P(A)=P_0+\beta\left(\sqrt{A}-\sqrt{A_0}\right)/A_0 \quad (5)$$

where A and Q are the lumen area and volumetric flow rate, ρ_f and μ are fluid density and viscosity, P_0 is the external pressure, A_0 is the tube lumen area at $P=P_0$, β is the wall rigidity. Equations (2) can be applied to a complex system of connected tubes. The corresponding boundary condition problem is discussed in [7].

1.3 2D models

2D theory of the blood flow and wave propagation in the fluid-filled tubes is based on the linearized Navier-Stokes equations for the fluid and elastic or viscoelastic wall motion [1,2]:

$$div\vec{v}=0, \qquad \rho\left(\frac{\partial v}{\partial t}+(\vec{v}\nabla)\vec{v}\right)=-\nabla p+\mu\Delta\vec{v} \quad (6)$$

$$div\vec{u}=0, \qquad \rho_s\frac{\partial^2\vec{u}}{\partial t^2}=-\nabla p_s+div\,\hat{\sigma} \quad (7)$$

$$r=0: \qquad v_r=0, \quad |v_x|<\infty \quad (8)$$

$$r=R': \quad \frac{\partial\vec{u}}{\partial t}=\vec{v}, \quad -p+\mu\frac{\partial v_r}{\partial r}=-p_s+\sigma_{rr}, \quad \mu\left(\frac{\partial v_x}{\partial r}+\frac{\partial v_r}{\partial x}\right)=\sigma_{rx} \quad (9)$$

$$r=R_0+H: \qquad \vec{u}=0 \quad (10)$$

where $\vec{v}=(v_r,0,v_x)$, $\vec{u}=(u_r,0,u_x)$ are the fluid velocity and wall displacement in the cylindrical coordinate system connected to the tube, ρ_s is the wall density, p, p_s are the hydrostatic pressures in the fluid and wall, $R'(t,x)=R_0(x)+u_r(t,r,x)\big|_{r=R(x)}$ is the actual tube radius, $R_0(x)=\sqrt{S_0(x)/\pi}$ is undisturbed radius, $\hat{\sigma}$ is the stress tensor in the wall:

$$\tau_s\frac{d\hat{\sigma}}{dt}+\hat{\sigma}=\hat{E}\hat{\varepsilon}+\mu_s\frac{d\hat{\varepsilon}}{dt} \quad (11)$$

where $\hat{\varepsilon}$ is the strain rate tensor, $\hat{E}$ is the tensor of elastic coefficients, τ_s is the stress relaxation time, μ_s is the wall viscosity coefficient.

Eq. (10) corresponds to the fastening of the outer wall of the tube to the rigid tissues which is valid for the deep arteries. For the superficial arteries the zero stress boundary conditions $\sigma_{rr},\sigma_{xx}=0$ should be used instead of (10). Solution of (6)-(11) can be found as superposition of the small excitations (Fourier expansion)

$$x=0: \qquad P=\sum_{k=0}^{\infty}P_k^0(r)e^{i\omega_k t} \quad (12)$$

At the outlet of the tube the conditions of the wall reflections must be posed, for instance in the form

$$x=L: \qquad \int_0^{R'}rpdr=\pi Z\left(R'\right)^2\int_0^{R'}rv_x dr \quad (13)$$

1.4 3D models

3D flow of Newtonian fluids in viscoelastic tubes is governed by mass and momentum balance equations

$$\frac{\partial \rho_f}{\partial t} + div(\rho_f \vec{v}) = 0$$
$$\rho_f \left(\frac{\partial \vec{v}}{\partial t} + (\vec{v}\nabla)\vec{v} \right) = -\nabla p + div(\hat{T}) + \zeta div(\vec{v})\hat{I} \qquad (14)$$

$$div\vec{u} = 0\,, \qquad \rho_w \frac{\partial^2 \vec{u}}{\partial t^2} = -\nabla p_w + div\,\hat{\sigma} \qquad (15)$$

where $\vec{u}$ and $\vec{v}$ are wall displacement and fluid velocity, ρ_f and ρ_w are fluid and wall densities, ζ is the second viscosity for the compressible fluid, p_w is hydrostatic pressure in the incompressible wall.

The rheological model for the wall is

$$\left[I + \sum_j \tau_j \frac{D^j}{Dt^j} \right] \hat{\sigma} = \left[\hat{E} + \sum_j k_j \frac{D^j}{Dt^j} \right] \hat{\varepsilon} \qquad (16)$$

where $\hat{\sigma}$ and $\hat{\varepsilon}$ are stress and strain tensors, $\hat{E}$ is tensor of elastic coefficients, τ_j and k_j are the j-th order relaxation and retardation times, I and D/Dt are unit operator and invariant time derivative.

For Newtonian fluids the boundary conditions for (3)-(5) include the stress and flow continuity at the fluid-wall interface and no displacement (I), no stress (II) or attachment to the surrounding viscoelastic media (III) at the outer surface

$$\partial\Omega: \quad \frac{\partial \vec{u}}{\partial t} = \vec{v}\,, \quad -p + \mu \frac{\partial v_r}{\partial r} = -p_s + \sigma_n\,, \quad \mu\left(\frac{\partial v_x}{\partial r} + \frac{\partial v_r}{\partial x} \right) = \sigma_\tau$$

$$\partial\Omega': \quad \vec{u} = 0 \ \text{(I)} \quad \text{or} \quad \sigma_n = 0 \ \text{(II)} \quad \text{or} \quad p_m \frac{\partial^2 \vec{u}}{\partial t^2} + \mu_m \frac{\partial \vec{u}}{\partial t} + E_m \vec{u} = 0 \ \text{(III)} \qquad (17)$$

where $\partial\Omega$ and $\partial\Omega'$ are undisturbed and actual outer surface of the tube; ρ_m, μ_m and E_m are density, viscosity and elasticity of the surrounding medium.

Substitution of (16) into the second equation (15) gives equations with 3-th order time derivatives which needs additional initial condition and careful estimation of possible changes in the equation type.

2. Wave propagation in a tube terminated by Windkessel element

The studied model is presented in fig.2. The arterial segment is modeled as viscoelastic tube terminated by a branching system of smaller arteries. The system is modeled as a single Windkessel element with resistivity Z.

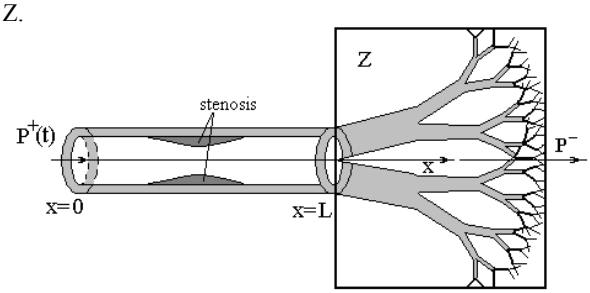

Figure 2. A model of arterial system as a combination of the tube with terminal Windkessel element Z

The model (6)-(11) with the boundary conditions (12), (13) has been considered as a linear model. The solution of the problem has been found as Fourier expansions [8]. The input data (12) has been measured by ultrasound sensors on a group of healthy volunteers and a group of elderly patients with stenosed arteries.

For the comparative analysis the nonlinear model (5) for the tube and the 0D model (2) for the terminal element has been used. Solution of (5), (2) has been computed using the method of characteristics [9].

3. Results and discussions

Oscillations of the pressure $P(t)$, fluid flow $\vec{u}(t)$ and wall displacement $\vec{v}(t)$ has been computed for different input boundary conditions [11], wall rheology and resistivity Z of the terminal element. For the healthy wall $E=10^5-10^6$ Pa has been taken, while for the stenosed wall with calcification and atherosclerotic plaques the elastic modulus has been taken as $E=10^7-10^8$ Pa. It is convenient to analyze the computational results as $P(u)$ curves which are repeatable at the long-time measurements. The measured and computed curves $P(u)$ for the healthy coronary artery of a volunteer are presented in fig.3a-d; the systolic parts of the curves are marked by solid lines. The typical results for the patients with coronary stenosis are depicted in fig.3c,d. It is clear, the linear and

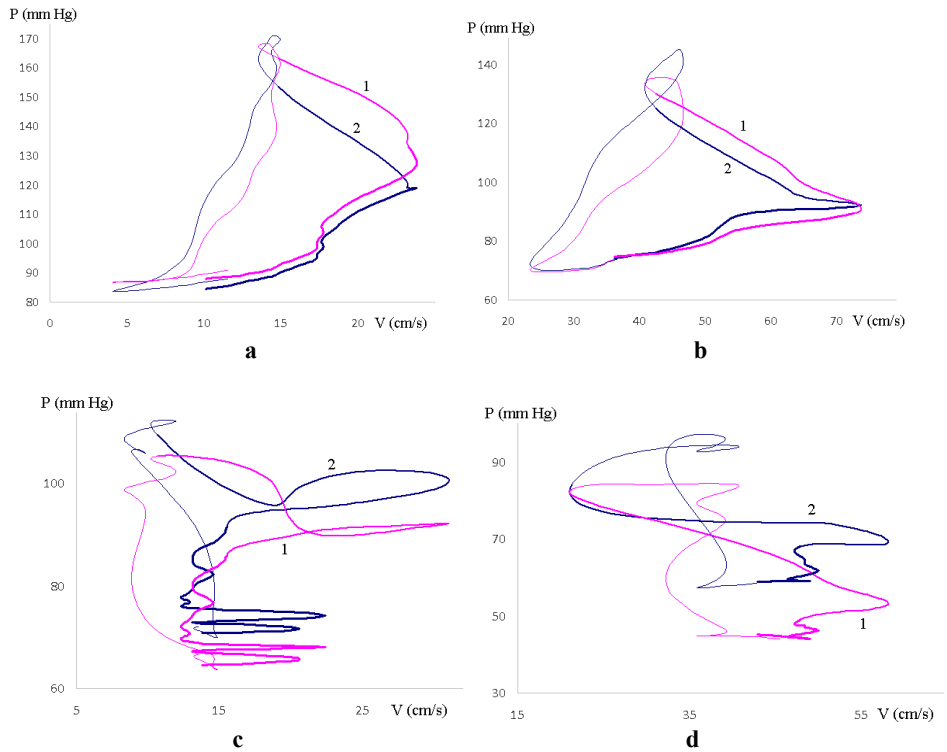

Figure 3. The $P(u)$ curves computed on the nonlinear (a,c) and linear (b,d) models (curves 1) in comparison to the measurement data (curves 2)

nonlinear models demonstrate equal exactness in comparison with the measured curves, while for the degenerated pathological wall is better described by the nonlinear models, though the exactness is much lower than for the young healthy wall.

Conclusions

Blood flow and wave propagation through the stenosed artery terminated by a viscoelastic chamber with different resistivity and compliance has been considered. The material parameters of the wall corresponded to healthy elastic and degenerated compliant human arteries. Both 2d linearized and 1d nonlinear models have been applied for numerical solution of the problem. The computational results for the pressure P(t), flow U(t) and diameter d(t) oscillations have been compared to the measurement data. It is shown that the dynamics of the fluid-filled vessels with healthy elastic walls is satisfactory described by the linearized equations while for the age-related degenerative walls, stenosed or diluted vessels the nonlinear models are preferable.

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