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Pressure pulse wave velocity and axial prestretch in arteries

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Abstract. The velocity of the propagation of a pressure pulse wave is considered to be a useful marker of the state of health of the cardiovascular system. Many clinical measurements, laboratory experiments and computational simulations have proved that the pressure pulse velocity correlates with age-related changes in the mechanical properties of arteries. Age-related stiffening of arteries, referred to as arteriosclerosis, leads to the increase in the pressure pulse velocity. However, the stress and strain state of an artery is not given solely by its loading and mechanical properties. Arteries are residually stressed that can be seen when cylindrical segment of an artery is excised from the body. The segment retracts because the axial prestretch is released. This prestretch declines with age, as a consequence of the damage accumulated to elastic lamellae during aging. Previous studies have paid little attention to the effect of axial prestretch on the velocity of the pressure pulse wave. The study presented here is based on a combination of a linearized 1D model of the fluid dynamics and the nonlinear anisotropic response of the human abdominal aorta. The model predicts that the application of axial prestretch can significantly change the velocity of the pressure pulse. To be more specific, preliminary results suggest that within the range of physiological pressures, the model that considers initial axial stretch of the aorta predicts lower pressure pulse velocity in comparison with the model that neglects axial prestrain of the tube.

Keywords: Aorta, Hyperelasticity, Pulse Wave Velocity.

1 Introduction

The blood ejected into the aorta during heart systole induces a pressure pulse wave, which is transmitted by the arterial wall towards the periphery [1]. As this is a mechanical wave, the pressure pulse travels with finite velocity (PWV), which depends on the mechanical properties of the wall and the blood, and on the geometry of the artery. Due to its direct relation to arterial stiffness, PWV has been established as a useful marker

of the state of health of the cardiovascular system [2-3]. Aged, therefore stiffened, arteries transmit a pressure pulse wave with higher velocity. This has been proven in many clinical measurements, laboratory experiments and computational simulations [4-5].

The mechanical state of an artery, however, is not dictated merely by its material properties and by the external loading caused by blood pressure. Arteries exhibit stresses and strains induced by their growth and remodeling [6-7]. When a tubular segment of an artery is excised in an autopsy, the segment retracts because arteries grow axially prestretched [8-10]. This way induced axial residual stress plays an important mechanical role. Under physiologically optimal conditions, axial prestress reduces the variation of axial stress and strain during the pressure cycle, and also contributes to the circumferential distensibility of an artery [11-12].

The axial prestretch declines with age, and this goes hand-in-hand with the damage accumulated to elastic membranes [8,10-11]. Our study aims to elucidate the effect of axial prestretch on the pressure pulse wave velocity in the human abdominal aorta. Our computational model for predicting PWV will be based on a 1D linearized model of the fluid dynamics, which will be combined with the nonlinear solid mechanical response of the aorta. It will be shown that axially prestretched artery transmits a pressure wave with a lower velocity than its non-prestretched counterpart.

2 Methods

2.1 Inflation and extension of the aortic wall

The abdominal aorta is modeled as a thin-walled tube with middle radius R, thickness H and length L, which, due to a change in internal pressure or axial force, changes its radius to r, its length to l, and its thickness to h. Thus, the deformation gradient $\mathbf{F}$ expressed in polar cylindrical coordinates takes the form as follows, $\mathbf{F} = diag(\lambda_R,\lambda_\Theta,\lambda_Z) = diag(h/H,r/R,l/L)$. The material of the wall is assumed to be incompressible, $det\mathbf{F} = \lambda_R\lambda_\Theta\lambda_Z = 1$.

It is assumed that the thin-walled tube modeling the abdominal aorta satisfies the balance equations expressed in (1). The radial stress σ_{rr} is considered to be zero, thanks to the thin-walled assumption. The circumferential stress $\sigma_{\theta\theta}$ and the axial stress σ_{zz} correspond to the Laplace law for a closed circular cylindrical tube loaded by internal pressure P and axially prestretched by force F_{red}. The inertial force is neglected due to the small volume of the wall in comparison to the blood.

$$\sigma_{rr} = 0 \qquad \sigma_{\theta\theta} = \frac{rP}{h} \qquad \sigma_{zz} = \frac{rP}{2h} + \frac{F_{red}}{2\pi rh} \tag{1}$$

2.2 Constitutive behavior of the aortic wall

The abdominal aorta is considered to be an anisotropic and nonlinearly elastic continuum. Its mechanical response is governed by the strain energy density function W, which is expressed in (2). The stored energy is here decoupled into the part related to

the isotropic matrix (elastic fibers, passive smooth muscle cells, and other connective proteins), which is represented in (2) by a linear neo-Hookean term, and into the part expressing the contribution of the collagen fibers, which is represented by the sum of two exponential expressions. These two exponential expressions reflect the large strain stiffening of an artery wall due to nonlinear recruitment of collagen fibers into the load-carrying process. I_1 denotes the first principal invariant of the right Cauchy-Green strain tensor $\mathbf{C} = \mathbf{F}^{\mathrm{T}}\mathbf{F}$. K_j (j = 4, 6) denotes structural invariants arising from the anisotropy of the material, and is expressed in (3). The anisotropy of an artery wall is a consequence of the collagen fibers that reinforce the wall. The collagen fibers are assumed to be arranged into two families of helices, around which a small portion of the fibers is dispersed. The predominant directions of fiber families are inclined from the tangential axis of the tube with angles β and $-\beta$. Under this situation, $I_4 = I_6 = \lambda_\Theta^2 cos^2(\beta) + \lambda_Z^2 sin^2(\beta)$ holds.

$$W = \frac{\mu}{2}\left(I_1 - 3\right) + \sum_{j=4,6} \frac{k_1}{2k_2}\left(e^{k_2\left(K_j - 1\right)^2} - 1\right) \tag{2}$$

$$K_j = \kappa I_1 + \left(1 - 3\kappa\right) I_j \quad j = 4,6 \tag{3}$$

Strain energy density function W expressed in (2) has been introduced by T. C. Gasser et al. in [13] and has become very popular in the last decade in cardiovascular biomechanics community. Constitutive equations for the hyperelastic incompressible wall of the abdominal aorta are expressed in (4). The Cauchy stress tensor is assumed to be of the form $\boldsymbol{\sigma} = diag(\sigma_{rr}, \sigma_{\theta\theta}, \sigma_{zz})$. In (4), p denotes a Lagrangean multiplier enforcing a hydrostatic stress component, which has to be determined by means of boundary conditions.

$$\sigma_{rr} = \lambda_R \frac{\partial W}{\partial \lambda_R} - p \qquad \sigma_{\theta\theta} = \lambda_\Theta \frac{\partial W}{\partial \lambda_\Theta} - p \qquad \sigma_{zz} = \lambda_Z \frac{\partial W}{\partial \lambda_Z} - p \tag{4}$$

2.3 Fluid dynamics equations

The blood flow in the abdominal aorta is considered to be 1D. Under this condition, the balance of the momentum can be written in the form of (5) and the conservation of mass can be written in the form of (6). Here v is the axial velocity of the flowing blood, A is the cross-section of the pressurized artery, ρ is blood density, and t denotes time.

$$\frac{\partial v}{\partial t} + v\frac{\partial v}{\partial z} + \frac{1}{\rho}\frac{\partial P}{\partial z} = 0 \tag{5}$$

$$\frac{\partial vA}{\partial z} + \frac{\partial A}{\partial t} = 0 \tag{6}$$

Following standard arguments and assumptions (long wave and insignificant $r = r(z)$, insignificant contribution of the convective acceleration), explained in detail for instance in Fung's monograph [14] pp. 140 – 146, one can arrive to (7). Pulse wave velocity c is in (7) expressed in one of possible forms of the famous Moens-Korteweg equation. Here, in contrast to ordinary linear form, where Young elastic modulus appears in (7), stiffness is introduced via term $\lambda_\Theta(\partial P/\partial\lambda_\Theta)$ which opens a possibility to combine linear fluid dynamics inside the elastic tube with nonlinear solid mechanics of the tube's wall.

$$c = \sqrt{\frac{\lambda_\Theta}{2\rho}\frac{\partial P}{\partial \lambda_\Theta}} \tag{7}$$

2.4 Differentiation of the pressure

Equation (7) gives us a simple instrument for estimating how the axial prestretch induced by axial force F_{red} influences the velocity of the propagation of the pressure pulse in the abdominal aorta. What we need here is $P = P(\lambda_\Theta)$ to be able to evaluate $\partial P/\partial\lambda_\Theta$, which appears in (7). This can be done with the help of balance equations (1). The substitution of σ_{rr}, $\sigma_{\theta\theta}$, and σ_{zz} from constitutive equations (4) into (1) and the replacement of the unknown deformed dimensions by means of kinematic equations of the inflated thin-walled cylindrical tube $h = \lambda_R H$, $r = \lambda_\Theta R$, leads to the system of equations (8-10).

$$\lambda_R \frac{\partial W}{\partial \lambda_R} - p = 0 \tag{8}$$

$$\lambda_\Theta \frac{\partial W}{\partial \lambda_\Theta} - p = P\frac{\lambda_\Theta}{\lambda_R}\frac{R}{H} \tag{9}$$

$$\lambda_Z \frac{\partial W}{\partial \lambda_Z} - p = \frac{P}{2}\frac{\lambda_\Theta}{\lambda_R}\frac{R}{H} + \frac{F_{red}}{2\pi\lambda_\Theta\lambda_R RH} \tag{10}$$

Equation (8) determines p. It is substituted to (9) and (10). Simultaneously, the incompressibility condition $\lambda_R\lambda_\Theta\lambda_Z = 1$ is used to replace λ_R. Equation (9) takes the form of (11), and equation (10) takes the form of (12).

$$\left[\lambda_\Theta \frac{\partial W}{\partial \lambda_\Theta}\right]_{\lambda_R = \lambda_\Theta^{-1}\lambda_Z^{-1}} - \left[\lambda_R \frac{\partial W}{\partial \lambda_R}\right]_{\lambda_R = \lambda_\Theta^{-1}\lambda_Z^{-1}} = P\lambda_\Theta^2\lambda_Z\frac{R}{H} \tag{11}$$

$$\left[\lambda_Z \frac{\partial W}{\partial \lambda_Z}\right]_{\lambda_R = \lambda_\Theta^{-1}\lambda_Z^{-1}} - \left[\lambda_R \frac{\partial W}{\partial \lambda_R}\right]_{\lambda_R = \lambda_\Theta^{-1}\lambda_Z^{-1}} = \frac{P}{2}\lambda_\Theta^2\lambda_Z\frac{R}{H} + \frac{F_{red}}{2\pi RH}\lambda_Z \tag{12}$$

3 Simulation of the effect of axial prestretch on PWV

Computational model based on the system of equations (11-12) and (7) will be used to estimate how axial prestretch affects pressure pulse wave velocity in the human abdominal aorta. The material parameters and the reference dimensions were adopted from [15].

In the first step of the simulation, neglecting inertial forces for the aortic wall, quasi-static predictions of the simultaneous inflation and extension of the aorta were conducted on the basis of equations (11) and (12). With the parameters adopted form [15], the initial axial stretches were given as λ_Z^{ini} = 1, 1.05, 1.1, 1.15, 1.2, 1.25, 1.3, 1.35, and 1.4. (11) and (12) were used to compute λ_Θ and λ_Z for pressures $P = 0.1(i - 1)$ kPa, where i = 1..181. For i = 1, $\lambda_Z = \lambda_Z^{ini}$ and P = 0 hold and λ_Θ^{ini} and F_{red} are computed.

When $i > 1$, P and F_{red} are assigned and λ_Θ and λ_Z are obtained from the simulation. This means that when the pressure rises, the tube axially prestressed by F_{red} inflates and, as a consequence, the tube deforms both in circumferential direction (λ_Θ) and in axial direction (λ_Z). Although F_{red} is constant during pressurization, $\sigma_{\theta\theta}$ and σ_{zz} change to satisfy the constitutive equations (4). In the second step of the simulation, $\partial P/\partial\lambda_\Theta$ was computed for specific P, F_{red}, λ_Θ and λ_Z, and all these quantities were substituted into (7) to obtain PWV. For the sake of completeness, blood density ρ was considered to be 1060 kg/m3 throughout the study. All the computations were performed with the help of Maple 2015.

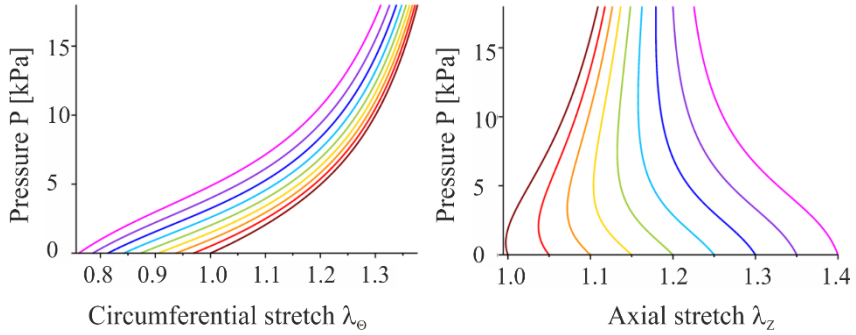

Fig. 1. Quasi-static predictions of the inflation-extension behavior for abdominal aorta obtained from (11-12). Axial prestretch λ_Z^{ini} is the value at which P–λ_Z curves begin for P = 0. Prestretches are distinguished by the color of the curves.

4 Results and Discussion

The computed results are presented in Figures 1 and 2. Figure 1 depicts the quasi-static inflation-extension response of 38 years old male individual characterized with the following constitutive parameters μ = 15.90 kPa, k_1 = 78.49 kPa, k_2 = 4.991, β = 41.41°, κ = 0.1875, and referential geometric dimensions R_i = 5.3 mm, H = 1.22 mm. Figure 2

shows the dependence of the pressure pulse wave velocity on the axial prestretch determined in the simulation.

Figure 1 is easy to interpret, when the reader realizes that the $P–\lambda_\Theta$ curves shift to the left for increasing prestretch, and the $P–\lambda_Z$ curves shift monotonically to the right. It is clear that the mechanical response depends substantially on the prestretch. From previous studies [11,12], it is known that axial prestretch makes the aorta more distensible at in vivo pressures (approx. 10 – 16 kPa). Detailed exploration of $P–\lambda_\Theta$ dependences in Figure 1 confirms this statement. One reveals that highly axially prestretched artery (violet curve, $\lambda_Z^{ini} = 1.4$) is more deformed when the pressure rises from 10 kPa to 16 kPa than a non-prestretched artery (brown curve, $\lambda_Z^{ini} = 1.0$).

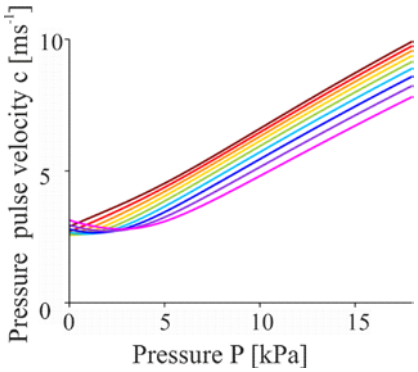

Fig. 2. Dependence of the pressure pulse velocity on the pressure. Prestretches are distinguished by the color of the curves. Color scheme is the same as in Figure 1. The highest prestretch $\lambda_Z^{ini} = 1.4$ is indicated by the violet color (lowest curve).

Figure 2 shows that axial prestretch also affects pressure pulse velocity. The lowest velocities were computed for the highest axial prestretchces. It suggests that axial prestretch not only makes arteries more distensible, it simultaneously decreases the velocity at which pressure pulse propagates along the aorta. Although this study is based on simplified model of the fluid-structure interaction, it again indicates that initial axial stretch plays very important role in the cardiovascular physiology. Limitations of the study consists in assumptions adopted in the model. The most important limitations are neglected nonlinearity in flow equations and the assumption of inviscid flow.

Conflict of Interest

Authors declare that they have no conflict of interest.

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