



Residual stresses reduce pulse wave velocity in arteries

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Abstract

In this note, we develop simple analytical formulas to estimate the effect of residual stresses on the pulse wave velocity in blood vessels. We combine these formulas with three constitutive models of the arterial wall. Particularly, we consider the Fung model and two models accounting for the dispersion of collagen fibers via 8 and 16 structure tensors accordingly. The residual stresses come into play with a description of the initial kinematics—the opening angle. Our numerical examples reveal that residual stresses reduce the pulse wave velocity. The latter effect becomes especially pronounced at high values of blood pressure.

Keywords Residual stresses · Pulse wave velocity · Constitutive models

1 Introduction

Blood pressure is one of the significant health indicators. Many factors such as physical exercise, emotional, and physiological states affect blood pressure. Excessive fall and rise in blood pressure leads to accompanying disorders, hypotension and hypertension, respectively [1–3]. Conventionally, blood pressure is measured by releasing the external pressure that is imparted to the arm by an inflating cuff, which brings the blood flow to halt temporarily [4]. This cannot be used for continuous monitoring of blood pressure. Continuous monitoring is possible only through invasive procedures that rely on the measurements of intra-arterial pressure [5]. These techniques put patients at a disadvantage of exposure to risk of complications and, hence, call for intensive care [6]. Blood pressure measurement alone cannot provide any conclusive prediction of the health status of heart.

Pulse wave velocity (PWV) is described by the velocity with which the blood pressure pulse propagates in arteries. Pulse wave velocity measurement is suitable for continuous and noninvasive monitoring of blood pressure [7–10]. The Moens-Korteweg equation [11] was used together with Hughes equation [12] to correlate the blood pressure to the measured pulse wave velocity (PWV): $PWV = \sqrt{Eh_0/(2\rho R_0)}$ and $E = E_0 \exp(\zeta p)$, where p is the blood pressure, E_0 is the elastic modulus at zero blood pressure, ζ is a material coefficient of artery, ρ is the blood mass density, and h_0 and R_0 are the initial wall thickness and radius of artery.

The Moens-Korteweg equation was developed based on the assumptions that the artery wall was thin and its thickness and radius were constant. In reality for human arteries, the thickness to radius ratio varies with blood pressure [13] and the thickness to radius ratio is much higher than the range which can be used for thin shell [14]. Recently, it was proposed [15], instead of the Moens-Korteweg equation, to use formula $PWV = \sqrt{(s/\rho)dp/ds}$, where s is the inner cross-sectional area of the artery, together with Fung's hyperelastic constitutive model [16] for the arterial wall. This new formula is valid for arteries with thick walls and with thickness to radius ratio varying with blood pressure.

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In the present work, we make the next step and develop simple analytical formulas to estimate the effect of residual stresses—prestresses—on the pulse wave velocity in blood vessels. We combine these formulas with three constitutive models of the arterial wall. Particularly, we consider the Fung model and two models accounting for the dispersion of collagen fibers via 8 and 16 structure tensors accordingly. The residual stresses come into play with a description of the initial kinematics—the opening angle.

We note in passing that residual stresses can affect mechanical behavior of materials in various and unexpected ways. We refer to [20] and [21] for the effect of residual stresses on surface wave propagation and bifurcation in soft materials and structures accordingly.

2 Pulse wave velocity

We refer to [17] for the general theoretical background and notation.

With account of axial symmetry, nontrivial equilibrium equations for the arterial wall in cylindrical coordinates reduce to

$$r \frac{\partial \sigma_{rr}}{\partial r} = \lambda_\varphi \frac{\partial \psi}{\partial \lambda_\varphi} - \lambda_r \frac{\partial \psi}{\partial \lambda_r}, \quad (1)$$

where σ_{rr} is the principal Cauchy stress in the radial direction and $\psi(\lambda_r, \lambda_\varphi, \lambda_z)$ is the strain energy density depending on the principal stretches in directions of cylindrical coordinates r, φ, z .

With account of incompressibility, $\lambda_r \lambda_\varphi \lambda_z = 1$, we can introduce a reduced strain energy function

$$w(\lambda, \lambda_z) = \psi(\lambda_r, \lambda_\varphi, \lambda_z) = \psi(\lambda^{-1} \lambda_z^{-1}, \lambda, \lambda_z). \quad (2)$$

Then, we calculate

$$\begin{aligned} \frac{\partial w}{\partial \lambda} &= \frac{\partial \psi}{\partial \lambda_r} \frac{\partial \lambda_r}{\partial \lambda} + \frac{\partial \psi}{\partial \lambda_\varphi} \frac{\partial \lambda_\varphi}{\partial \lambda} + \frac{\partial \psi}{\partial \lambda_z} \frac{\partial \lambda_z}{\partial \lambda} \\ &= \frac{\partial \psi}{\partial \lambda_\varphi} - \frac{1}{\lambda^2 \lambda_z} \frac{\partial \psi}{\partial \lambda_r}, \end{aligned} \quad (3)$$

or

$$\lambda_\varphi \frac{\partial \psi}{\partial \lambda_\varphi} - \lambda_r \frac{\partial \psi}{\partial \lambda_r} = \lambda \frac{\partial w}{\partial \lambda}. \quad (4)$$

Now, Eq. 1 can be rewritten as follows

$$r \frac{\partial \lambda}{\partial r} \frac{\partial \sigma_{rr}}{\partial \lambda} = \lambda \frac{\partial w}{\partial \lambda}. \quad (5)$$

Further transformation of the equilibrium equation requires calculation of $\partial \lambda / \partial r$. For this purpose, we use the incompressibility condition in the form

$$\gamma \lambda_z (r^2 - a^2) = R^2 - A^2, \quad (6)$$

where $\gamma = 2\pi / (2\pi - \omega)$ and ω is the artery opening angle after the cut; r, a and R, A are the radial coordinate and internal radius of the artery in the current and referential configurations accordingly.

Consequently, we have

$$\lambda = \gamma r R^{-1} = \gamma r (\gamma \lambda_z (r^2 - a^2) + A^2)^{-1/2}, \quad (7)$$

and, by the direct calculation,

$$r \frac{\partial \lambda}{\partial r} = \lambda (1 - \lambda_z \gamma^{-1} \lambda^2). \quad (8)$$

Substitution of Eq. 8 in Eq. 5 yields

$$\frac{\partial \sigma_{rr}}{\partial \lambda} = \frac{1}{1 - \lambda_z \gamma^{-1} \lambda^2} \frac{\partial w}{\partial \lambda}. \quad (9)$$

Integrating Eq. 9 we get for the blood pressure on the arterial wall

$$p(\lambda_a) = \int_{\lambda_b(\lambda_a)}^{\lambda_a} \frac{w' d\lambda}{\lambda_z \gamma^{-1} \lambda^2 - 1}, \quad (10)$$

Table 1 Material constants for Fung’s model [16]

| c | c_1 | c_2 | c_3 | c_4 | c_5 | c_6 |
|-----------|--------|--------|-------|--------|--------|--------|
| 26.95 kPa | 0.0089 | 0.9925 | 0.418 | 0.0193 | 0.0749 | 0.0295 |

where $w' \equiv \partial w / \partial \lambda$; $\lambda_a = \gamma a / A$; $\lambda_b = \gamma b / B$; and

$$\lambda_b = \sqrt{\lambda_a^2 A^2 B^{-2} + (1 - A^2 B^{-2}) \gamma \lambda_z^{-1}} \tag{11}$$

by virtue of the incompressibility condition.

According to Fung [13], the pulse wave velocity is calculated as follows

$$PWV = \sqrt{\frac{s}{\rho} \frac{\partial p}{\partial s}}, \tag{12}$$

where $s = \pi a^2$ is the artery internal cross-section area and ρ is the blood mass density.

In terms of the boundary stretch λ_a , we can rewrite Eq. 12 as follows

$$PWV = \sqrt{\frac{\lambda_a}{2\rho} \frac{\partial p}{\partial \lambda_a}}, \tag{13}$$

where the pressure derivative is calculated from Eq. 10 analytically

$$\frac{\partial p}{\partial \lambda_a} = \frac{w'(\lambda_a)}{\lambda_z \gamma^{-1} \lambda_a^2 - 1} - \frac{w'(\lambda_b)}{\lambda_z \gamma^{-1} \lambda_b^2 - 1} \frac{\lambda_a A^2}{\lambda_b B^2}. \tag{14}$$

Thus, we finally have (compare also with [18])

$$PWV = \sqrt{\frac{\lambda_a}{2\rho} \left\{ \frac{w'(\lambda_a)}{\lambda_z \gamma^{-1} \lambda_a^2 - 1} - \frac{w'(\lambda_b)}{\lambda_z \gamma^{-1} \lambda_b^2 - 1} \frac{\lambda_a A^2}{\lambda_b B^2} \right\}}. \tag{15}$$

3 Constitutive models of arterial wall

We consider the following three models of the arterial wall.

The Fung constitutive model [16] is defined by the following strain energy density function

$$w(\lambda, \lambda_z) = \frac{c}{2} \left\{ \exp \left[\frac{c_1}{4} (\lambda^{-2} \lambda_z^{-2} - 1)^2 + \frac{c_2}{4} (\lambda^2 - 1)^2 + \frac{c_3}{4} (\lambda_z^2 - 1)^2 + \frac{c_4}{2} (\lambda^{-2} \lambda_z^{-2} - 1) (\lambda^2 - 1) + \frac{c_5}{2} (\lambda^2 - 1) (\lambda_z^2 - 1) + \frac{c_6}{2} (\lambda^{-2} \lambda_z^{-2} - 1) (\lambda_z^2 - 1) \right] - 1 \right\}, \tag{16}$$

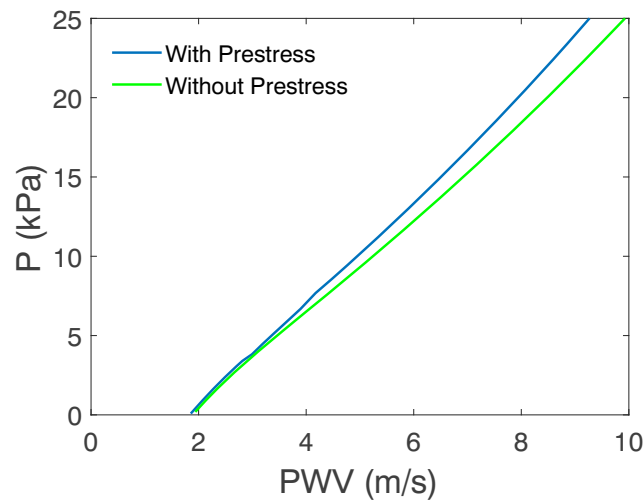
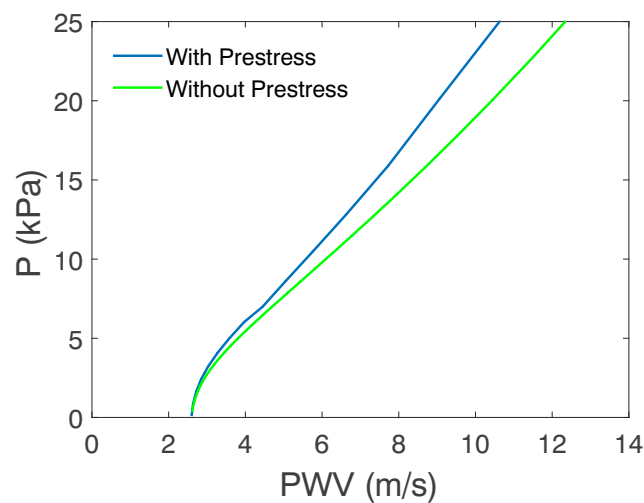
where material constants c and c_i , fitted in tests on rabbit carotid arteries, are given in Table 1.

Table 2 Material constants for model with 8 (left) and 16 (right) structure tensors [19]

| c | k_1 | k_2 | c | k_1 | k_2 |
|-------|----------|-------|-------|----------|-------|
| 5 kPa | 2.04 kPa | 1.58 | 5 kPa | 2.23 kPa | 1.63 |

Table 3 Material parameters for models with 8 (left) and 16 (right) structure tensors [19]

| $\phi^{(i)}$ | $\vartheta^{(i)}$ | $\alpha^{(i)}$ | $\phi^{(i)}$ | $\vartheta^{(i)}$ | $\alpha^{(i)}$ |
|--------------|-------------------|----------------|--------------|-------------------|----------------|
| 10° | 90° | 0.440527 | 10° | 82.5° | 0.211123 |
| 34° | 90° | 2.6916 | 34° | 82.5° | 1.28996 |
| 59° | 90° | 2.36997 | 59° | 82.5° | 1.13581 |
| 80° | 90° | 0.724365 | 80° | 82.5° | 0.347153 |
| 100° | 90° | 0.724365 | 100° | 82.5° | 0.347153 |
| 121° | 90° | 2.36997 | 121° | 82.5° | 1.13581 |
| 146° | 90° | 2.6916 | 146° | 82.5° | 1.28996 |
| 170° | 90° | 0.440527 | 170° | 82.5° | 0.211123 |
| | | | 10° | 97.5° | 0.211123 |
| | | | 34° | 97.5° | 1.28996 |
| | | | 59° | 97.5° | 1.13581 |
| | | | 80° | 97.5° | 0.347153 |
| | | | 100° | 97.5° | 0.347153 |
| | | | 121° | 97.5° | 1.13581 |
| | | | 146° | 97.5° | 1.28996 |
| | | | 170° | 97.5° | 0.211123 |

**Fig. 1** Pressure versus pulse wave velocity with and without residual stress for Fung's model**Fig. 2** Pressure versus pulse wave velocity with and without residual stress for the model with 8 structure tensors

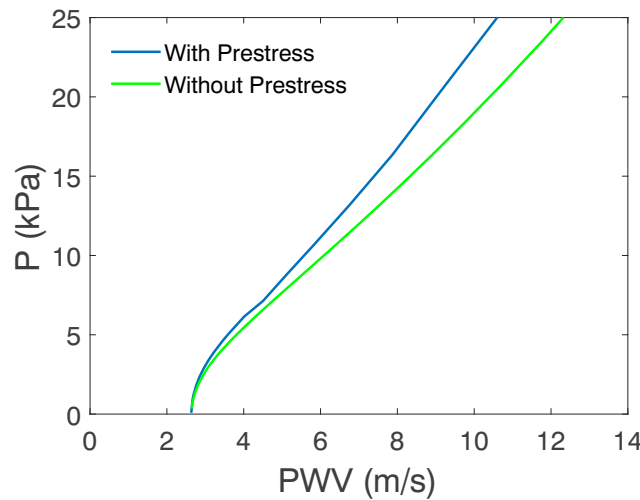


Fig. 3 Pressure versus pulse wave velocity with and without residual stress for the model with 16 structure tensors

Two constitutive models with fiber dispersion [19] include $N = 8$ and $N = 16$ structure tensors and the strain energy density function is defined as follows

$$\begin{aligned}
 w(\lambda, \lambda_z) = & \frac{c}{2}(\lambda\lambda_z^{-2} + \lambda^2 + \lambda_z^2 - 3) \\
 & + \frac{k_1}{2k_2} \sum_{i=1}^N \alpha^{(i)} \{ \exp[k_2((\lambda \cos \Phi^{(i)} \sin \Theta^{(i)})^2 \\
 & + (\lambda_z \sin \Phi^{(i)} \sin \Theta^{(i)})^2 + (\lambda^{-1}\lambda_z^{-1} \cos \Theta^{(i)})^2 \\
 & - 1)^2] - 1 \}, \tag{17}
 \end{aligned}$$

where material constants c, k_1, k_2 and parameters $\alpha^{(i)}, \Phi^{(i)}, \Theta^{(i)}$, fitted in [19] to the experimental data on human arteries, are given in Tables 2 and 3 accordingly.

4 Results

We calculate the blood pressure and the pulse wave velocity from Eqs. 10 and 15 accordingly as functions of the current internal radius of the artery. This radius can be interpreted as a parameter, which is excluded from the pressure-velocity plots. Such plots are shown in Figs. 1, 2, and 3 for arterial models described above. These plots were generated for $\gamma = 1.8$ ($\omega = 160^\circ$), $\lambda_z = 1$, $A = 1.43$ mm, and $B = 1.82$ mm with account of residual stresses and $\gamma = 1$ ($\omega = 0^\circ$), $\lambda_z = 1$, $A = 0.71$ mm, and $B = 1.1$ mm without residual stresses. The blood mass density was $\rho = 1060$ kg/m³.

All plots show monotonic decrease of the pulse wave velocity with the increase of residual stresses in the physiological range. The difference can reach 20% for high pressures.

5 Conclusion

In this note, we presented a simple analytical study of the effect of residual stresses—prestresses—on the pulse wave velocity of blood in arteries. We found that the residual stresses generally reduced the pulse wave velocity.

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