Proceedings of the ASME 2021 International Mechanical Engineering Congress & Exposition
IMECE2021
November 8-14, 2021, Salt Lake City, Utah, USA

IMECE2021-69412

PULSE WAVE VELOCITY AND TRANSMISSION AT THE CAROTID ARTERY AND THE ASCENDING AORTA

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ABSTRACT

With consideration of a full set of mechanical properties: elasticity, viscosity, and axial and circumferential initial tensions, and radial and axial motion of the arterial wall, this paper presents a theoretical study of pulse wave propagation in arteries and evaluates pulse wave velocity and transmission at the carotid artery (CA) and the ascending aorta (AA). The arterial wall is treated as an initially-tensioned, isotropic, thin-walled membrane, and the flowing blood in the artery is treated as an incompressible Newtonian fluid. Pulse wave propagation in arteries is formulated as a combination of the governing equations of radial and axial motion of the arterial wall, the governing equations of flowing blood in the artery, and the interface conditions that relate the arterial wall variables to the flowing blood variables. We conduct a free wave propagation analysis of the problem and derive a frequency equation. The solution to the frequency equation indicates two waves: Young wave and Lamb wave, propagating in the arterial tree. With the related values at the CA and the AA, we evaluate the influence of arterial wall properties on their wave velocity and transmission, and find the opposite effects of axial and circumferential initial tensions on transmission of both waves. Physiological implications of such influence are discussed.

Keywords: Arterial wall, elasticity, viscosity, axial initial tension, circumferential initial tension, radial motion, axial motion, pulse wave propagation, wave velocity, wave transmission, atherosclerosis

1. INTRODUCTION

From the physical perspective, blood circulation in the cardiovascular (CV) system is pulse wave propagation in the arterial tree. The arterial wall plays a critical role in determining the characteristics of pulse wave propagation in arteries [1]. Pathological changes in the arterial wall alter physical parameters of the arterial wall and cause changes in pulse wave propagation, which may consequently cause damage to the heart. This may explain why the dominant cause of CV disease is atherosclerosis [1].

Radial motion of the arterial wall has been extensively studied, due to its clinical applications in detection and diagnosis of atherosclerosis [1-3]. Arterial elasticity and viscosity have been evaluated from radial motion of the arterial wall for their clinical values. Particularly, Pulse Wave Velocity (PWV) based on arterial elasticity has become a well-established index in the clinical field [3]:

$$PWV = \sqrt{\frac{Eh}{2\rho_b a}} \tag{1}$$

where E, h, and a denote the elasticity, thickness, and the inner radius of the arterial wall at diastolic blood pressure (DBP), respectively, and ρ_b denotes blood density. Arterial viscosity has not been studied as extensively as arterial elasticity, due to technical complexity involved in its measurement [4].

In recent years, the advancement of imaging technologies has allowed measurement of axial motion of the arterial wall. Clinical studies have established clinical values of axial motion of the arterial wall for serving as a more sensitive and possibly earlier measure of subclinical atherosclerosis and providing a comprehensive assessment of arterial health, together with radial motion of the arterial wall [5, 6].

Due to its anatomy, the arterial wall contains significant inherent axial pre-stretch, which greatly affects the remodeling and growth of the arterial wall and also decreases with aging [7]. Axial pre-stretch translates to axial initial tension in the arterial wall. Meanwhile, DBP in the artery causes circumferential initial tension and also varies with arterial health condition [8]. As the arterial wall properties, axial and circumferential initial tensions are expected to affect pulse wave propagation in the artery.

To date, numerous studies of pulse wave propagation in arteries have been conducted for radial motion of the arterial wall, with solely arterial elasticity and viscosity being considered [1-3]. Despite their identified clinical values, axial motion, axial and circumferential initial tensions of the arterial wall have been mostly neglected in the related theoretical studies [9]. This work is aimed to investigate the influence of arterial wall properties on pulse wave propagation in arteries for a better understanding of CV physiology and improved

applications of arterial wall properties in detection and diagnosis of atherosclerosis. In this work, we conduct a theoretical study of pulse wave propagation in arteries, which includes both radial motion and axial motion of the arterial wall and a full set of arterial wall properties: elasticity, viscosity, and axial and circumferential initial tensions. The problem is formulated as a combination of the governing equations of radial and axial motion of the arterial wall, the governing equations of flowing blood in the artery, and the interface conditions that relate the arterial wall variables to the flowing blood variables. We conduct a free wave propagation analysis of the problem and derive a frequency equation, which is a quadratic equation of the squared wave velocity with arterial wall properties and geometries as coefficients. The solution to the frequency equation is two complex wave velocities and translates to the wave velocity and transmission of two waves: Young wave and Lamb wave, propagating in the arterial tree. With the related values at the carotid artery (CA) and the ascending aorta (AA), we evaluate the wave velocity and transmission of each wave at the two locations in the arterial tree and examine the influence of arterial wall properties on their wave velocity and transmission for physiological implications.

2. RELATED THEORIES

2.1 Five Variables in the Artery

The anatomy of the arterial wall is rather complex [1]. For the purpose of gaining insights on the role of arterial wall properties in pulse wave propagation, the arterial wall is treated as an initially-tensioned, elastic, isotropic, thin-walled circular membrane. As shown in Fig. 1(a), the arterial wall has two geometrical parameters: a as the inner radius of the arterial wall at DBP and h as the thickness of the arterial wall. Both geometrical parameters remain unchanged during arterial wall motion. Axial initial tension per unit length and circumferential initial tension per unit length in the arterial wall are denoted by T_{x0} and $T_{\theta 0}$, respectively. The arterial wall undergoes radial motion and axial motion and thus has two associated variables: $\eta(t)$ as the radial displacement and $\xi(t)$ as the axial displacement. Flowing blood in the artery is assumed to be an incompressible Newtonian fluid. There are three variables related to the flowing blood: w(t) and u(t) as the radial velocity and the axial velocity, respectively, and $\Delta p(t)$ as the pulsatile pressure.

2.2 Problem Formulation

Two fundamental assumptions in this subsection are that 1) the five variables are axisymmetric and small perturbations; and 2) the inner radius of the arterial wall is much smaller than the wavelength λ of the pulse wave ($a \ll \lambda$) [3, 9, 10].

Governing equations of flowing blood in the artery

The governing equations of flowing blood in the artery include the continuity equation and the two Navier-Stokes equations along the radial (r-axis) and the axial (x-axis) directions [9]:

$$\frac{\partial w}{\partial r} + \frac{w}{r} + \frac{\partial}{\partial x} u = 0 \qquad (2a)$$

$$\rho_b \frac{\partial w}{\partial t} = -\frac{\partial \Delta p}{\partial r} + \mu\left(\frac{\partial^2 w}{\partial r^2} + \frac{1}{r}\frac{\partial w}{\partial r} - \frac{w}{r^2} + \frac{\partial^2 w}{\partial x^2}\right) \qquad (2b)$$

$$\rho_b \frac{\partial u}{\partial t} = -\frac{\partial \Delta p}{\partial x} + \mu\left(\frac{\partial^2 u}{\partial r^2} + \frac{1}{r}\frac{\partial u}{\partial r} + \frac{\partial^2 u}{\partial x^2}\right) \qquad (2c)$$

where ρ_b and μ denote the density and viscosity of flowing blood in the artery, respectively.

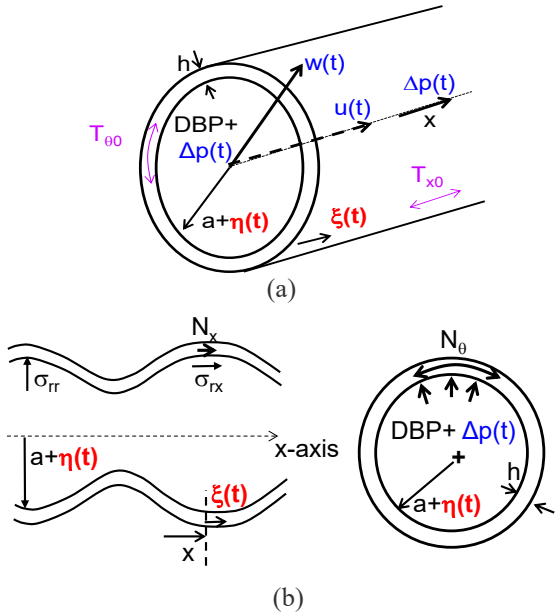

FIGURE 1: Schematic views of an artery (a) 3D view (b) 2D views in the axial direction and the circumferential direction, with the arterial wall geometries (a, h) and two initial tensions (T_{x0} and $T_{\theta 0}$); and three variables (u, w, Δp) of flowing blood in the artery and two variables (η, ξ) of the arterial wall.

Governing equations of the arterial wall

As shown in Fig. 1(b), the stresses acting on the inner surface of the arterial wall are due to pulsatile pressure and shear forces of flowing blood. These stresses are:

$$\sigma_{rr} = \Delta p - 2\mu \frac{\partial w}{\partial r} \text{ (3a)} \qquad \sigma_{rx} = \tau_w = \mu \cdot \left(\frac{\partial u}{\partial r} + \frac{\partial w}{\partial x}\right) \qquad (3b)$$

where σ_{rx} is commonly referred to as wall shear stress and is commonly denoted by τ_w. Strains in the circumferential direction and the axial direction are expressed as:

$$\varepsilon_{\theta\theta} = \partial\eta / \partial r = \eta / a \text{ (4a)} \qquad \varepsilon_{xx} = \partial\xi / \partial x \qquad (4b)$$

As shown in Fig. 1(b), during its motion, the arterial wall experiences the axial tension per unit length N_x and the circumferential tension per unit length N_θ:

$$N_x = \frac{Eh}{1-\upsilon^2}\cdot\left\{\frac{\partial\xi}{\partial x}+\upsilon\frac{\eta}{a}\right\}+T_{x0} \quad (5a)$$

$$N_\theta = \frac{Eh}{1-\upsilon^2}\cdot\left\{\frac{\eta}{a}+\upsilon\frac{\partial\xi}{\partial x}\right\}+T_{\theta 0} \quad (5b)$$

where E and ν denote the elasticity and Poisson's ratio of the arterial wall, respectively.

The force balance analysis of the arterial wall gives rise to the governing equations of the arterial wall undergoing the radial and axial motion [10]:

$$\rho h\cdot\frac{\partial^2\eta}{\partial t^2} = \left\{\Delta p - 2\mu\cdot\frac{\partial w}{\partial r}\right\}_{r=a} + T_{\theta 0}\cdot\frac{\eta}{a^2} + T_{x0}\cdot\frac{\partial^2\eta}{\partial x^2} - \frac{Eh}{1-\upsilon^2}\cdot\left\{\frac{\eta}{a^2}+\frac{\upsilon}{a}\cdot\frac{\partial\xi}{\partial x}\right\} \quad (6a)$$

$$\rho h\frac{\partial^2\xi}{\partial t^2} = -\mu\left\{\frac{\partial u}{\partial r}+\frac{\partial w}{\partial x}\right\}_{r=a} + \frac{Eh}{1-\upsilon^2}\cdot\left\{\frac{\partial^2\xi}{\partial x^2}+\frac{\upsilon}{a}\cdot\frac{\partial\eta}{\partial x}\right\} + \frac{T_{x0}-T_{\theta 0}}{a}\cdot\frac{\partial\eta}{\partial x} \quad (6b)$$

where ρ denotes the arterial wall density. Since the arterial wall displacements are small, the actual location of the wall inner surface differs very little from its initial location during arterial wall motion. Thus, the values of stresses from flowing blood in the brackets in Eq. (6) are calculated at r=a.

Blood-wall interface conditions

Given that flowing blood in the artery needs to adhere to the inner surface of the arterial wall, the velocities of flowing blood must be equal to the velocities of the arterial wall in the radial and axial directions at r=a:

$$u_{r=a} = \frac{\partial\eta}{\partial t} \quad (7a) \qquad w_{r=a} = \frac{\partial\xi}{\partial t} \quad (7b)$$

Note that Eq. (7) relates the two variables of the arterial wall to the two velocity variables of flowing blood in the artery.

2.3 Solution of the Problem

Under the condition that finite axial velocity at the center (r=0) of the artery and a<<λ, the solution to the governing equations of flowing blood, Eq. (2), has been well established [9]. Here, we assume that each variable propagates along the positive x-axis. Then, the wave expressions for the three variables of flowing blood in the artery become [9]:

$$w = \left[-A\frac{\beta_0^2 r}{2\mu\alpha_0^2} + B\frac{\beta_0}{\alpha_0 J_0(\alpha_0)}J_1(\alpha_0 r/a)\right]\cdot e^{i(\omega t-kx)} \quad (8a)$$

$$u = \left[-A\frac{\beta_0 a}{\mu\alpha_0^2} + B\frac{J_0(\alpha_0 r/a)}{J_0(\alpha_0)}\right]\cdot e^{i(\omega t-kx)} \quad (8b)$$

$$\Delta p = A\cdot e^{i(\omega t-kx)} \quad (8c)$$

where $\alpha_0^2 = i^3\alpha^2$, $\alpha^2 = \rho_b a^2\omega/\mu$ and $\beta_0 = i\frac{a\omega}{c} = i\beta$; and A and B are two constants associated with the amplitudes of these three variables. Similarly, the wave expressions for the two variables of the arterial wall are:

$$\eta = C\cdot e^{i(\omega t-kx)} \quad (8d)$$

$$\xi = D\cdot e^{i(\omega t-kx)} \quad (8e)$$

where C and D are the amplitudes of the radial and axial variables of the arterial wall, respectively.

There are four unknown constants: A, B, C, and D, in the five variables in Eq. (8). Now, we substitute these wave expressions into the governing equations of the arterial wall, Eq. (6), and the two blood-wall interface conditions, Eq. (7), yielding the following four equations:

$$-\frac{\beta_0^2 a}{2\mu\alpha_0^2}A + \frac{1}{2}\beta_0 F_{10}B - i\omega C = 0 \quad (9a)$$

$$-\frac{\beta_0 a}{\mu\alpha_0^2}A + B - i\omega D = 0 \quad (9b)$$

$$A + \left[(T_{x0}\beta_0^2 + T_{\theta 0} - \frac{Eh}{1-\upsilon^2})\frac{1}{a^2} + \rho h\omega^2\right]C + \frac{Eh}{1-\sigma^2}\frac{\upsilon\beta_0}{a^2}D = 0 \quad (9c)$$

$$-\frac{\beta_0^3}{\alpha_0^2}A + \frac{\mu\alpha_0^2}{2a}F_{10}B - (\frac{Eh}{1-\upsilon^2}\upsilon + T_{x0} - T_{\theta 0})\frac{\beta_0}{a^2}C + (\frac{Eh}{1-\upsilon^2}\frac{\beta_0^2}{a^2} + \rho h\omega^2)D = 0 \quad (9d)$$

where $F_{10} = 2J_1(\alpha_0)/\left[\alpha_0 J_0(\alpha_0)\right]$. We further re-organize the above four equations into a 4×4 matrix equation with a vector of the four unknowns, Eq. (10), where the axial and circumferential initial tensions per unit length are both normalized as below:

$$\begin{bmatrix} -\frac{\beta_0 a}{\mu\alpha_0^2} & 1 & 0 & -i\omega \\ -\frac{\beta_0^2 a}{2\mu\alpha_0^2} & \frac{1}{2}\beta_0 F_{10} & -i\omega & 0 \\ 1 & 0 & (T_{x0}\beta_0^2 + T_{\theta 0} - \frac{Eh}{1-\upsilon^2})\frac{1}{a^2} + \rho h\omega^2 & \frac{Eh\upsilon}{1-\upsilon^2}\frac{\beta_0}{a^2} \\ -\frac{\beta_0^3}{\alpha_0^2} & \frac{\mu\alpha_0^2}{2a}F_{10} & -(\frac{Eh\upsilon}{1-\upsilon^2} + T_{x0} - T_{\theta 0})\frac{\beta_0}{a^2} & \frac{Eh}{1-\upsilon^2}\frac{\beta_0^2}{a^2} + \rho h\omega^2 \end{bmatrix} \begin{Bmatrix} A \\ B \\ C \\ D \end{Bmatrix} = \begin{Bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{Bmatrix} \quad (10)$$

$$(1-F_{10})\frac{4}{(1-\upsilon^2)^2}\cdot\left\{1-\upsilon^2-\tau_{\theta 0}-(\tau_{x0}-\tau_{\theta 0})\upsilon\right\}\frac{c_0^4}{c^4} + \left\{-K(1-F_{10})(1-\tau_{\theta 0}) + F_{10}(2\upsilon+\tau_{x0}-1/2\tau_{\theta 0}-1/2) - 2\right\}\frac{2}{1-\upsilon^2}\frac{c_0^2}{c^2} + F_{10} + 2K = 0 \quad (12)$$

$$\frac{1-(k_L a)^2-\upsilon^2}{k_f^2}\frac{\omega^4}{c^4}+\left\{(k_L a)^2-1+\upsilon^2-\beta\right\}\frac{\omega^2}{c^2}+k_L^2\left\{1-(k_L a)^2+\beta\right\}=0 \quad \text{with} \quad k_f=\omega/\sqrt{B_b/\rho_b}\,,\ \beta=\frac{2B_b a}{Eh}(1-\upsilon^2),\ k_L=\omega/c_L \quad (14)$$

$$\tau_{x0}=T_{x0}\Big/\frac{Eh}{1-\upsilon^2} \quad (11a) \qquad \tau_{\theta 0}=T_{\theta 0}\Big/\frac{Eh}{1-\upsilon^2} \quad (11b)$$

Evidently, if a nontrivial solution is pursued for the four unknowns, the determinant of the 4×4 matrix in Eq. (10) must be equal to zero. With $a<<\lambda$, we can obtain a frequency equation, Eq. (12), where

$$K=\rho h/(\rho_b a) \ \text{ and } \ c_0=PWV=\sqrt{\frac{Eh}{2\rho_b a}} \quad (13)$$

Getting rid of axial and circumferential initial tensions in Eq. (12) leads to the same frequency equation derived in the well-cited Womersley's 1955 paper [9].

Here, we provide the frequency equation, Eq. (14), for pulse wave propagation in arteries without blood viscosity and the two initial tensions. In Eq. (14), B_b denotes the bulk modulus of blood and c_L denotes the elastic in-plane wave velocity [11]:

$$c_L=\sqrt{\frac{E}{\rho(1-\upsilon^2)}} \quad (15)$$

2.4 Wave Velocity and Transmission

Eq. (12) is essentially a quadratic equation of $(c_0/c)^2$, with arterial wall properties and geometries as coefficients. Note that F_{10} in Eq. (12) results from blood viscosity and is complex. Thus, the solution to Eq. (12) is two complex roots of c: c_1 and c_2. While the first root, c_1, represents the Young wave, the second root, c_2, represents the Lamb wave. The complex root c is further expressed in terms of its real and imaginary values:

$$c_0/c=X+Yi \quad (16)$$

where X and Y take real values. While the real value, real(c)=c_0/X, represents the wave velocity (or phase velocity) and wave transmission per wavelength is then calculated as exp(2πY/X). The wavelength is related to the wave velocity by:

$$\lambda=2\pi\frac{real(c)}{\omega} \quad (17)$$

Eq. (14) is a quadratic equation of $(\omega/c)^2$. Since blood viscosity is not considered, no complex values are involved in the coefficients of the equation, and thus the solution to Eq. (14) are two real roots of c, indicating co-existence of the two waves and also no transmission loss (or 100% transmission) for both waves.

It should be noted that when radial motion and axial motion of the arterial wall are separately considered and blood viscosity is not considered, the wave velocities associated with the Young wave and the Lamb wave are c_0 and c_L, respectively.

3. RESULTS AND DISCUSSION

In this section, based on the frequency equations, Eq. (12) and (14), we evaluate pulse wave velocity and transmission at the CA and the AA, analyze the difference between them, as well as examine the influence of blood viscosity and arterial wall properties on their wave velocity and transmission. Table 1 summarizes the physical properties and geometrical parameters of the CA and the AA and the physical properties of flowing blood. With a heart rate of 70 beats per minute (bpm), all of the calculations are conducted in MATLAB.

3.1 Influence of Blood Viscosity

Table 2 summarizes the calculated values of the wave velocity, wave transmission per unit length, and wavelength at the AA and the CA with and without blood viscosity. Although the AA and the CA are initially tensioned in the axial and circumferential directions, the values of these initial tensions are unknown and thus are assumed to be zero here. At the AA, while the influence of blood viscosity on the Young wave velocity is negligible, blood viscosity moderately reduces the Lamb wave velocity. Blood viscosity reduces the Young wave transmission by roughly 10%, and cuts the Lamb wave transmission from 100% to about 50%, indicating that blood viscosity plays a more important role in the Lamb wave transmission. At the CA, blood viscosity moderately reduces the Young wave velocity, but causes a large reduction in the Lamb wave velocity. Blood viscosity reduces the Young and Lamb wave transmissions to about 50% and about 25%, respectively. When blood viscosity is not considered, the Young wave velocity and the Lamb velocity at the AA and the CA are very close to c_0 and c_L, respectively, indicating the important role of wall shear stress in pulse wave propagation.

Table 1 Physical properties and geometrical parameters of the carotid artery (CA) and the ascending aorta (AA) and physical properties of flowing blood [12, 13]

	Parameter	Symbol	Carotid artery (CA)	Ascending aorta (AA)
Arterial wall	Radius	a	3.3mm	14.7mm
	Thickness	h	0.62mm	1.63mm
	Elasticity	E	771kPa	400kPa
	Poisson's ratio	ν	0.5	
	Density	ρ	1055kg/m^3	
Blood	Density	ρ_b	1055kg/m^3	
	Viscosity	μ	0.0032Pa·s	
	Bulk modulus	B_b	2.2GPa	

Table 2 Influence of blood viscosity on wave velocity, wave transmission, and wavelength at the CA and the AA

	CA ($\tau_{\theta 0}$=0 and τ_{x0}=0)		AA ($\tau_{\theta 0}$=0 and τ_{x0}=0)	
Parameter	With blood viscosity	Without blood viscosity	With blood viscosity	Without blood viscosity
real(c_1) (m/s)	7.68	8.18	4.48	4.5844
real(c_2) (m/s)	22.30	31.61	19.66	22.65
$\exp(2\pi Y_1/X_1)$	0.52	1	0.89	1
$\exp(2\pi Y_2/X_2)$	0.23	1	0.48	1
λ_1 (m)	6.59	7.01	3.84	3.90
λ_2 (m)	19.11	27.09	16.85	19.41
c_0	8.29		4.58	
c_L	31.20		22.48	

Blood viscosity manifests in wall shear stress acting on the arterial wall along the axial direction. While the Young wave is a fluid-dominant wave, the Lamb wave is a wall-dominant wave. These might explain why blood viscosity affects the lamb wave velocity and transmission to a much larger extent than the Young wave velocity and transmission. Yet, given that the AA has a much larger size than the CA, the influence of wall shear stress on pulse wave velocity and transmission is much less prominent at the AA than at the CA.

With blood viscosity, the Young wave transmission is more efficient than the Lamb wave transmission at both the AA and the CA. Although the value of each wave transmission is much less than 100%, the overall transmission loss is expected to be low, given that the wavelength of each wave is well above the arterial tree length. The transmission of each wave is much lower at the CA than at the AA, but the wavelength of each wave is much higher at the CA than at the AA. Consequently, the overall transmission loss of the two waves might be comparable between the CA and the AA.

3.2 Influence of Arterial Elasticity and Viscosity

As shown in Fig. 2, the velocities of the two waves both increase with arterial elasticity. The elasticities of the AA and the CA start from their normal values in Table 1 and are increased by 200kPa. Overall, the Young wave velocity at the AA is lower than at the CA, and the Lamb wave velocity at the AA is comparable with that at the CA. The transmission of the two waves is not affected by arterial elasticity. With blood viscosity, the transmission of each wave is much higher at the AA than at the CA. Note that the transmission of both waves at the AA and the CA without blood viscosity is also included in Fig. 2(b) for the influence of blood viscosity on the wave transmission at the AA and the CA.

Fig. 3 compares the difference in the influence of arterial elasticity on the wave velocities between with and without blood viscosity at the AA and the CA. At the AA and the CA, the influence of blood viscosity on the two wave velocities remains the same, as arterial elasticity goes up.

To consider the influence of arterial viscosity on wave velocity and transmission, a complex arterial elasticity E′ is defined as below:

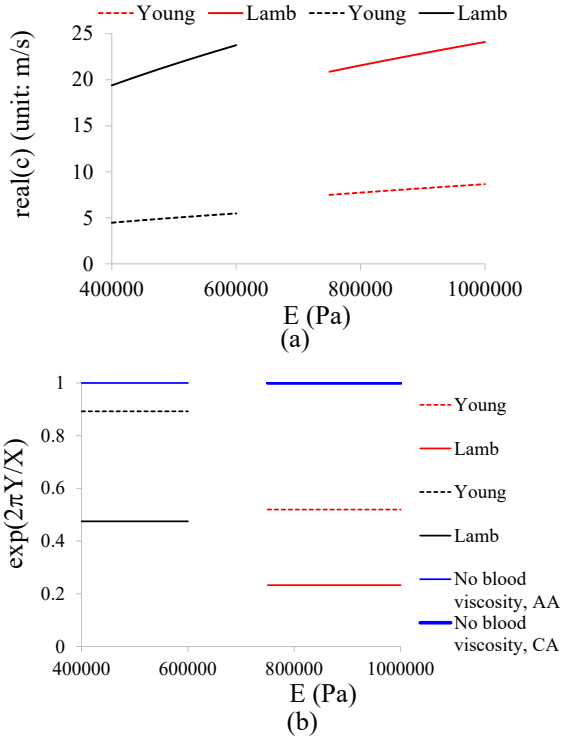

FIGURE 2: The influence of arterial elasticity on (a) wave velocity and (b) transmission per wavelength at the CA and the AA with blood viscosity (blacklines: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave; blue lines: without blood viscosity)

$$E' = E + i\omega\chi E \tag{18}$$

By keeping arterial elasticity in Table 1 for the AA and the CA as constant, the influence of arterial viscosity is plotted in Fig. 4. The influence of arterial viscosity on the Young wave velocity is slight but is moderate on the Lamb wave velocity at the AA and the CA. However, transmission of both waves decreases significantly with arterial viscosity, with the Young wave transmission decreasing even faster. Without blood viscosity, transmission of the two waves at the CA and the AA is the same and is also plotted in Fig. 4(b) for comparison. Note that blood viscosity dramatically decreases the Lamb wave transmission while moderately reducing the Young wave transmission at the AA and the CA. As arterial viscosity

increases, the influence of blood viscosity on each wave transmission becomes less prominent.

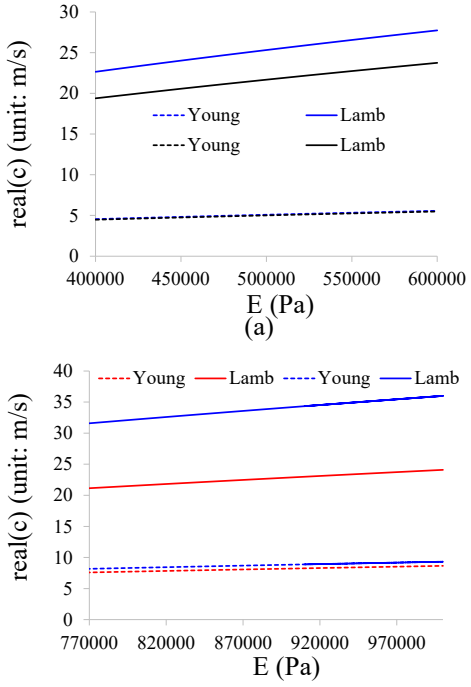

FIGURE 3: The influence of arterial elasticity on wave velocities (a) at the AA and (b) at the CA with/without blood visocity (blacklines: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave; blue lines: without blood viscosity)

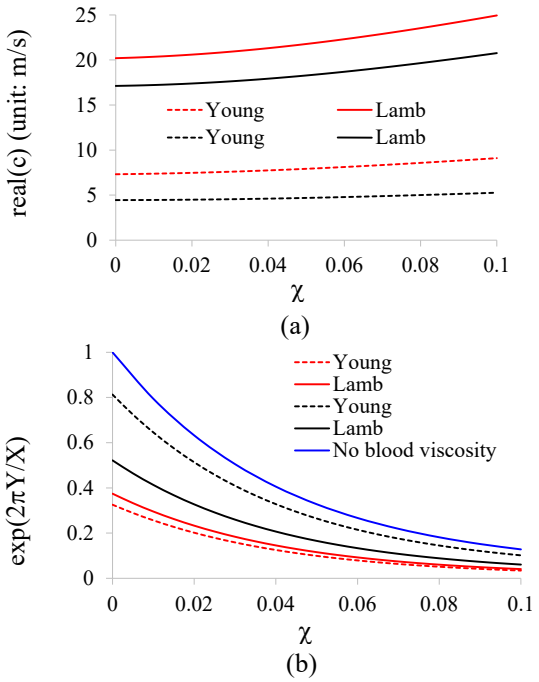

FIGURE 4: The influence of arterial viscosity on (a) wave velocity and (b) transmission per wavelength with blood viscosity at the CA and the AA (blackline: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave; blue line: without blood viscosity)

Fig. 5 compares the difference in the influence of arterial viscosity on the wave velocities at the AA and the CA between with and without blood viscosity. At both the AA and the CA, arterial viscosity does not affect the Young wave velocity, regardless of whether blood viscosity is considered. Yet, with blood viscosity, arterial viscosity greatly increases the Lamb wave velocity at the AA and the CA.

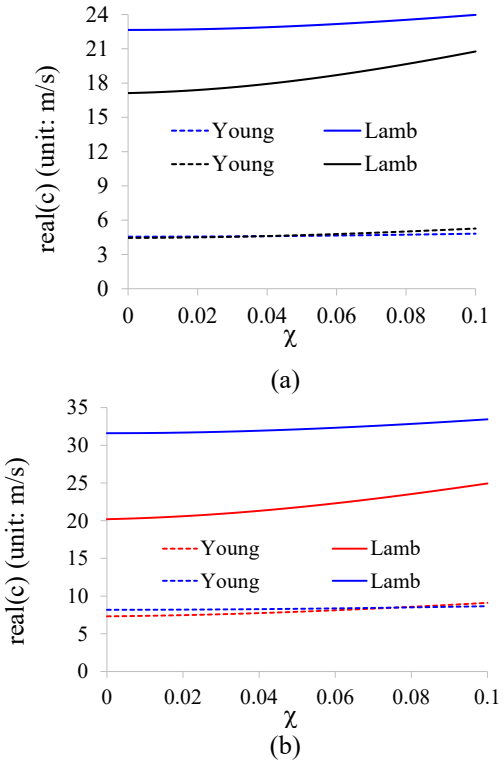

FIGURE 5: The influence of arterial viscosity on wave velocities (a) at the AA and (b) at the CA with/without blood viscosity (blackline: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave; blue lines: without blood viscosity)

3.3 Influence of Axial and Circumferential Initial Tensions

As shown in Fig. 6, the influence of axial initial tension on the Lamb wave velocity is negligible at the AA and the CA. However, axial initial tension reduces the Young wave velocity at the CA to a larger extent than that at the AA. Axial initial tension moderately reduces the Lamb wave transmission at the AA and the CA to the same extent, but increases the Young wave transmission to a much larger extent at the CA than at the AA.

As shown in Fig. 7, similar to axial initial tension, the influence of circumferential initial tension on the Lamb wave velocity is negligible at the CA and the AA. However, circumferential initial tension reduces the Young wave velocity to a larger extent at the CA than at the AA. The Lamb wave transmission is moderately increased by circumferential initial tension and such increase is very similar at the CA and the AA.

Circumferential initial tension reduces the Young wave transmission to a larger extent at the CA than at the AA.

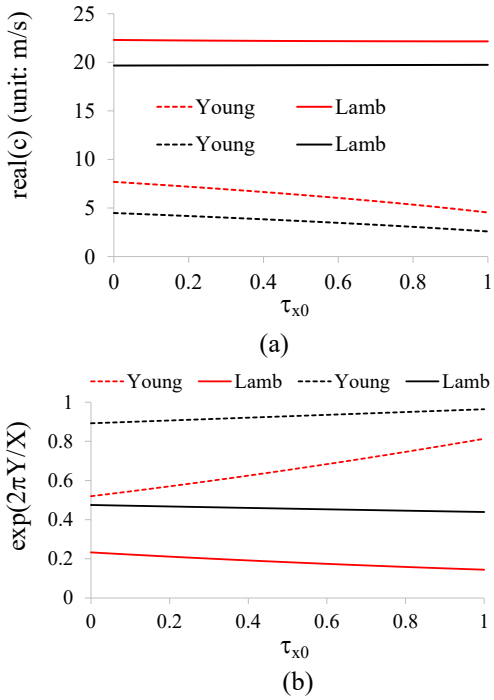

FIGURE 6: The influence of axial initial tension on (a) wave velocity and (b) transmission per wavelength with blood viscosity at the CA and the AA (blacklines: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave)

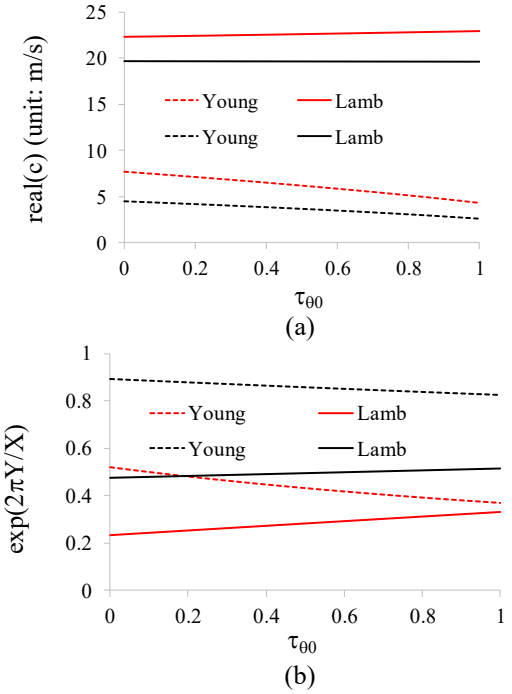

FIGURE 7 The influence of circumferential initial tension on (a) wave velocity and (b) transmission per wavelength with blood viscosity at the CA and the AA (blacklines: AA; red lines: CA; dashed lines: the Young wave; solid lines: the Lamb wave)

3.4 Physiological Implications

The physiological implication of arterial elasticity has been well established. Increase in arterial elasticity (or arterial stiffening) causes an increase in PWV, which undermines blood circulation and eventually increases burden to the heart. Therefore, a low Young wave velocity is favorable for facilitating blood circulation and alleviating the burden to the heart. With this as the guidance, both axial and circumferential initial tensions decrease the Young wave velocity and thus are both favorable for facilitating blood circulation. Yet, axial initial tension increases the Young wave transmission, while the circumferential initial tension reduces the Young wave transmission. Given that high wave transmission indicates high efficiency in blood circulation and low burden to the heart. Thus, circumferential initial tension undermines blood circulation and increases the workload of the heart. Circumferent initial tension results from DBP and is positively related to DBP. Recently, diastolic pressure hypertension was linked to cardiovascular risks in young adults [8]. Meanwhile, axial initial tension is inherent and decreases with aging. As such, it might be concluded that while axial initial tension is inherent in facilitating the Young wave propagation, DBP hypertension is a passive reaction of the artery to facilitate the Young wave propagation.

Although axial motion of the arterial wall has been studied [5, 6], the Lamb wave velocity has not been measured and studied for its clinical values. Nonetheless, axial and circumferential initial tensions have very slight influence on the Lamb wave velocity. However, axial initial tension reduces the Lamb wave transmission, circumferential initial tension has just the opposite effect. The dominant wave in blood circulation is the Young wave. Thus, although the reduced Lamb wave transmission is not preferred, it does not cause much burden to the heart. In contrast, although circumferential initial tension improves the Lamb wave transmission, its influence on the Young wave transmission is more prominent.

To date, there are no direct measures on arterial viscosity and indirect measures of arterial viscosity found that arterial viscosity increases with aging [4]. As predicted by this study, although the influence of arterial viscosity on the two wave velocities is negligible, arterial viscosity has a large influence on the transmissions of the two waves. As such, increased arterial viscosity translates to reduced efficiency in blood circulation and causes increased burden to the heart.

3.5 Study Limitations

There are two major study limitations in this work. First, the arterial wall is treated as an isotropic, thin-walled membrane. In reality, the arterial wall has a thin-layered anatomical structure and thus is orthotropic in nature. The radius/thickness ratio (a/h) of the AA and the CA are 9.0 and 5.3, respectively. According to the literature [14], when the radius/thickness ratio is above 10, there is no difference in the calculated values between a thin-walled model and a thick-

walled model. Although the calculated values are off, the influence of arterial wall properties on pulse wave propagation in arteries is illustrated. Certainly, a thick-walled model with orthotropic properties will improve the accuracy in the calculated values, but also greatly increase the mathemetical complexity and may prevent a better illustration of such influence.

Second, this study does not consider the excitation source at the heart and also wave reflection. As mentioned above, the goal of this work is to gain insights in the influence of arterial wall properties on pulse wave propagation and thus improve their clinical applications in detection and diagnosis of arterial abnormalities. Neglect of the excitation source and wave reflection may affect the calculated values, but are not expected to change the nature of the influence.

4. CONCLUSION

We have presented a free wave propagation analysis of pulse wave propagation in arteries, with a full set of mechanical properties: elasticity, viscosity, and axial and circumferential initial tensions, and radial and axial motion of the arterial wall. The frequency equation resulting from the analysis indicates the co-existence of two waves: the Young wave and the Lamb wave, propagating in the arterial tree. The obtained two complex roots of the frequency equation relate the wave velocity and transmission of both waves to arterial wall properties. The wave velocities and transmissions at the CA and the AA show a large difference, due to their significant difference in size and mechanical properties.

Based on the frequency equation, the influence of arterial wall properties on pulse wave propagation is revealed. The physiological implications of such influence are then discussed. In particular, axial and circumferential initial tensions are found to make their own non-negligible contributions to the efficiency of blood circulation and the heart.

ACKNOWLEDGEMENTS

This work is partially supported by the National Science Foundation (NSF) under Grant #1936005.

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