Relations of Radial Vibration of the Arterial Wall to Pulsatile Parameters in Blood Flow for Extraction of Arterial Indices

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Given the wide utility of radial vibration of the arterial wall for clinical values, this paper presents a theoretical study on the relations of radial vibration of the arterial wall to pulsatile parameters in blood flow. Pulse wave propagation in an artery is formulated as a combination of the governing equations of blood flow and the arterial wall and no-slip conditions at the blood-wall interface and is analyzed to obtain the wave velocity and the theoretical expressions for blood flow rate and radial wall displacement in terms of pulsatile pressure. With the harmonics of a pulse signal, theoretical relations of radial vibration of the arterial wall to pulsatile parameters in blood flow are derived under two conditions: without and with wave reflection. These theoretical relations identify the assumptions for the simplified relations employed in the utility of radial vibration of the arterial wall for clinical values. With the arterial wall treated as a unit-mass vibration system, these simplified relations are utilized for extraction of arterial indices from radial vibration of the arterial wall. Other applications of such relations for clinical values are discussed, and the interaction between the arterial wall and blood flow is further revealed from the perspective of energy and one-dimensional wave equations. With harmonics and wave reflection considered, the derived theoretical expressions for radial wall vibration, pulsatile parameters in blood flow, and the relations between them provide theoretical guidance for improving their interpretation of clinical values with clearly defined physiological implications and assumptions. [DOI: 10.1115/1.4055390]

Keywords: arterial pulse signal, time derivatives, radial vibration, blood flow rate, pulsatile pressure, wave reflection, harmonics, arterial indices

1 Introduction

Arterial pulse signals carry physiological and pathological information about the cardiovascular (CV) system and are interpreted to extract various arterial indices for detection and diagnosis of CV disease [1–5]. Based on the pulse wave propagation theory, the arterial pulse signals measured at the carotid and femoral artery sites using dual tonometry are utilized to extract the carotid-femoral pulse wave velocity (PWV) as an assessment of the global arterial stiffness (or elasticity) [1,2]. According to force equilibrium of the arterial wall, the local arterial elasticity is extracted from a combination of the arterial radial displacement signal and the pulsatile pressure signal at an artery site, which are measured simultaneously by an imaging instrument and a tonometer, respectively [2]. These two interpretations utilize only the measured arterial pulse signals themselves but involve high cost and operation complexity of the medical instruments, rendering them unsuitable for routine and at-home use.

Due to its simplicity and low cost, photoplethysmography (PPG) sensors are widely used to acquire arterial pulse signals, commonly referred to as PPG signals [4–10]. A PPG signal essentially captures the blood volume change in an artery. Its first-order and second-order derivatives have been utilized to extract arterial indices [4–10]. Since the blood volume change in an artery is caused by the radial displacement of the arterial wall and this displacement is only a small percentage of the arterial radius, it is reasonable to treat the PPG signal as the radial displacement of the arterial wall (or arterial vasomotion) [5,6,8–10]. Since the radial displacement of the arterial wall facilitates the blood flow velocity (or rate) in an artery, the first-order derivative (velocity) of a PPG signal is intuitively considered to represent the blood flow velocity [6–12]. Then, the second-order derivative (acceleration) of a PPG signal is considered to represent the acceleration of blood flow [4–10]. Yet, a clear theoretical basis for such interpretation of the two derivatives is still missing [6–12]. Without relating to pulsatile parameters in blood flow, different ratios of peaks and valleys in the second-order derivative of a PPG signal were found to carry clinical values via statistical analysis [4,7], but physiological implications of these ratios are unclear. In recent years, great similarity in waveform between a PPG signal and a pulsatile pressure signal has been identified [9,10,13]. Owing to their unparalleled convenience, PPG sensors have been increasingly studied for continuous, cuffless blood pressure measurement. Without theoretical guidance, different features of a PPG signal and its derivatives are combined for estimation of blood pressure via statistical analysis and machine-learning techniques [9,14,15]. With the goal of developing an at-home solution to arterial health assessment, the author's group has developed a microfluidic-based tactile sensor for arterial pulse signal measurement, which features ease of use by a layperson and low cost [2,3]. The measured arterial pulse signal is treated as the radial displacement of the arterial wall. Consequently, its first-order and second-order derivatives represent the velocity and acceleration of the arterial wall, respectively. Due to its time-harmonic nature, the pulsatile radial motion of the arterial wall at an artery site is radial vibration. Then, the arterial wall is modeled as a unit-mass vibration system, and its system parameters are extracted from the measured pulse signal and its two derivatives. A scaling analysis is conducted to relate the system parameters to pulsatile parameters in blood flow for extraction of arterial indices. Although this vibration-model-based analysis of an arterial pulse signal has been validated by experimental data [3], similar to PPG signals, a clear theoretical basis for the relations derived from the scaling analysis is still lacking.

Contributed by the Applied Mechanics Division Technical Committee on Dynamics & Control of Structures & Systems (AMD-DCSS) of ASME for publication in the Journal of Engineering and Science in Medical Diagnostics and Therapy. Manuscript received June 29, 2022; final manuscript received August 20, 2022; published online October 3, 2022. Editor: Ahmed Al-Jumaily.

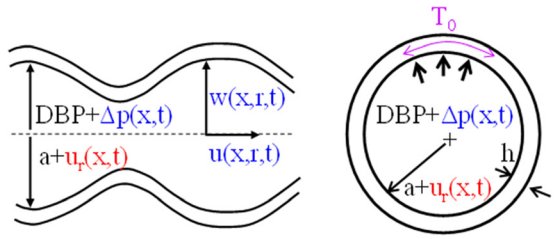

Fig. 1 Schematic of the arterial wall and blood flow in it and the associated geometries and pulsatile parameters

Given the above-mentioned utility of radial vibration (displacement, velocity, and acceleration) of the arterial wall for extraction of arterial indices, this paper presents a theoretical study on the relations of radial vibration of the arterial wall to pulsatile parameters in blood flow. Previously, the author developed a pulse wave propagation theory for obtaining theoretical expressions for various pulsatile parameters in terms of pulsatile pressure, without harmonics in a pulse signal and wave reflection [16]. Later on, harmonics in a pulse signal and wave reflection were included in the theory for calculating the waveforms of various pulsatile parameters [17]. In this paper, the developed theory is tailored to investigate the relations of the radial displacement and its two derivatives of the arterial wall to blood flow rate and pulsatile pressure. It is worth mentioning that axial displacement of the arterial wall [16,17] is omitted in this paper, due to its negligible influence on the pulsatile parameters considered in this paper. The pulse wave propagation theory is first presented for the wave velocity and the theoretical expressions for a radial displacement of the arterial wall and blood flow rate in terms of pulsatile pressure. With the harmonics of a pulse signal, theoretical relations of radial vibration of the arterial wall to pulsatile parameters in blood flow are derived under two conditions: without and with wave reflection. These derived theoretical relations identify the assumptions that are needed to simplify the relations of radial vibration of the arterial wall to pulsatile parameters in blood flow for clinical values [2,3,6]. With the arterial wall modeled as a unit-mass vibration system, these simplified relations are utilized to extract arterial indices from radial vibration of the arterial wall. Finally, some other applications of such relations for clinical values are discussed, and new insights into the interaction between the arterial wall and blood flow are revealed from the perspective of energy and one-dimensional (1D) wave equations.

2 Pulse Wave Propagation Theory

As shown in Fig. 1, the arterial wall is treated as a thin-walled tube with two geometrical parameters: a as the inner radius at diastolic blood pressure (DBP) and h as the thickness. It is assumed that both a and h do not vary in a pulse cycle. Due to DBP, the arterial wall contains an initial tension $T_0 = DBP \times a$ per unit length in the circumferential direction. With no axial displacement, the arterial wall undergoes solely the pulsatile radial displacement $u_r(x,t)$ to facilitate blood flow in the artery. Blood in the artery is assumed to be an incompressible, Newtonian fluid and contains three pulsatile parameters: radial velocity $w(x,r,t)$, axial (x-axis) velocity $u(x,r,t)$, and pulsatile pressure $\Delta p(x,t)$. Note that blood is known to be a non-Newtonian fluid, but it behaves as a Newtonian fluid in large arteries [18]. In small arteries (i.e., $a \leq 0.3$ mm), blood behaves as a non-Newtonian fluid and causes a noticeable reduction in wall shear stress [19]. Since all the pulse signals measured at the skin surface [1–15] are from large arteries, it is reasonable to treat blood as a Newtonian fluid. It is further assumed that (1) the four pulsatile parameters are axisymmetric and have small perturbations and (2) the arterial radius a is much smaller than the wavelength λ of the pulse wave ($a \ll \lambda$) [16,17,20,21].

Based on the above assumptions, the governing equations of blood flow in the artery include the continuity equation and the Navier–Stokes equations in the axial (x-axis) and radial (r-axis) directions [16,17,20,21]

$$\frac{\partial w}{\partial r} + \frac{w}{r} + \frac{\partial u}{\partial x} = 0 \tag{1a}$$

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

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

where ρ_b and μ denote the blood density and viscosity, respectively.

Womersley derived the solution to Eq. (1)—the wave expressions for w, u, and Δp [20] at the nth harmonic of the heart rate

$$w = \left[-\Delta p_0 \frac{\beta_0^2 r}{2\mu\alpha_0^2} + B\frac{\beta_0}{\alpha_0 J_0(\alpha_0)} J_1(\alpha_0 r/a)\right] \cdot e^{i(n\omega t - k_n x)} \tag{2a}$$

$$u = \left[-\Delta p_0 \frac{\beta_0 a}{\mu\alpha_0^2} + B\frac{J_0(\alpha_0 r/a)}{J_0(\alpha_0)}\right] \cdot e^{i(n\omega t - k_n x)} \tag{2b}$$

$$\Delta p = \Delta p_0 \cdot e^{i(n\omega t - k_n x)} \tag{2c}$$

where $\alpha_0^2 = i^3\alpha^2$ with $\alpha = a\sqrt{\rho_b n\omega/\mu}$ being the Womersley number and $\beta_0 = ian\omega/c = i\beta$ [20]; ω and $k_n = n\omega/c$ are the angular frequency of the heart rate and the nth wave number, respectively. Note that Δp_0 and B are two constant unknowns.

The governing equation of the arterial wall is given as

$$\rho h \frac{\partial^2 u_r}{\partial t^2} = \Delta p - [(E + \eta n\omega i)h - T_0] \cdot \frac{u_r}{a^2} \tag{3a}$$

where E and η denote the elasticity and viscosity, respectively, of the arterial wall in the circumferential direction. No-slip conditions at the blood-wall interface demand the blood flow velocities being equal to the arterial wall velocities in the radial and axial directions at the blood-wall interface

$$w_{r=a} = \frac{\partial u_r}{\partial t} \tag{3b}$$

$$u_{r=a} = 0 \tag{3c}$$

The wave expression for $u_r(x,t)$ at the nth harmonic of the heart rate can be written as

$$u_r = u_{r0} \cdot e^{i(n\omega t - k_n x)} \tag{4}$$

Substituting Eqs. (2) and (4) into Eq. (3) give rise to the following 3×3 matrix equation with a vector of the three constant unknowns of Δp_0, B, and u_{r0}:

$$\begin{bmatrix} -\frac{\beta_0 a}{\mu\alpha_0^2} & 1 & 0 \\ -\frac{\beta_0^2 a}{2\mu\alpha_0^2} & \frac{1}{2}\beta_0 F_{10} & -in\omega \\ 1 & 0 & [\tau_0 - (1 + \eta n\omega i/E)]\frac{Eh}{a^2} - \rho h(n\omega)^2 \end{bmatrix} \begin{Bmatrix} \Delta p_0 \\ B \\ u_{r0} \end{Bmatrix} = \begin{Bmatrix} 0 \\ 0 \\ 0 \end{Bmatrix} \tag{5}$$

where

$$\tau_0 = T_0/(Eh) \quad \text{and} \quad F_{10} = \frac{2J_1(\alpha_0)}{\alpha_0 J_0(\alpha_0)} \tag{6}$$

Note that F_{10} arises from fluid-loading and takes a complex value, and τ_0 is the normalized initial tension. A nonzero solution to the vector demands that the determinant of the 3×3 matrices in Eq. (5) be equal to zero, giving rise to the wave velocity

$$c = c_0\sqrt{(1-F_{10})\left\{[(1+\eta n\omega i/E)-\tau_0]-\frac{\rho a^2(n\omega)^2}{E}\right\}} \tag{7a}$$

where

$$c_0 = PWV = \sqrt{\frac{Eh}{2\rho_b a}} \tag{7b}$$

where c_0 is the same as the PWV commonly used in clinical studies [1–3]. Evidently, F_{10}, τ_0, and viscous and inertial terms of the arterial wall are all neglected in the estimation of PWV. The inertial term and viscous term in Eq. (7*a*) are both small quantities, as compared with the rest terms. Moreover, 1-F_{10} does not vary much with the harmonics (or n) of the heart rate. Thus, c remains the same for different harmonics in a pulse signal.

Based on Eq. (5), B and u_{r0} can be expressed in terms of Δp_0

$$B = \frac{-1}{c\rho_b}\Delta p_0 \tag{8a}$$

$$u_{r0} = \frac{(1-F_{10})a}{2c^2\rho_b}\Delta p_0 \tag{8b}$$

Based on Eq. (2*b*), the amplitude of axial blood velocity varies along the r-axis and is written as

$$u_0(r) = \frac{1}{c\rho_b}\left[1-\frac{J_0(\alpha_0 r/a)}{J_0(\alpha_0)}\right]\Delta p_0 \tag{8c}$$

Consequently, the blood flow rate amplitude Q_0 is expressed as

$$Q_0 = \int_0^a u_0(r)2\pi r dr = \frac{\pi a^2}{c\rho_b}(1-F_{10})\cdot\Delta p_0 = 2\pi a c u_{r0} \tag{9}$$

The relations of Q_0 and Δp_0 to u_{r0} remain the same for different harmonics in a pulse signal.

3 Relations of Radial Vibration of the Arterial Wall to Pulsatile Parameters in Blood Flow

An arterial pulse signal is a collection of the harmonics of the heart rate and experiences wave reflection in the CV system [22,23]. In this section, with the harmonics of a pulse signal considered, the relations of radial vibration of the arterial wall to pulsatile parameters in blood flow are derived under two conditions: without and with wave reflection.

3.1 Without Wave Reflection. Pulsatile pressure is the driving force for the pulse wave propagation in the artery [16,17,20,21]. When there is no wave reflection, the pulsatile pressure input at an artery site (or at a fixed-x position) is expressed as

$$\Delta p(t) = \sum_n A_n e^{i(n\omega t - k_n x)} \tag{10}$$

where A_n denotes the amplitude of the nth harmonic in a pulsatile pressure signal. According to Sec. 2, the radial displacement of the arterial wall and the blood flow rate are

$$u_r(t) = \frac{(1-F_{10})a}{2c^2\rho_b}\sum_n A_n e^{i(n\omega t - k_n x)} \tag{11}$$

$$Q(t) = \frac{1-F_{10}}{c\rho_b}\pi a^2\sum_n A_n e^{i(n\omega t - k_n x)} \tag{12}$$

As the time-derivative of the blood flow rate, the acceleration of blood flow $\partial Q(t)/\partial t$ is expressed as

$$\frac{\partial Q(t)}{\partial t} = \frac{1-F_{10}}{\rho_b}\pi a^2\sum_n ik_n A_n e^{i(n\omega t - k_n x)} \tag{13}$$

The pulsatile pressure gradient $\Delta p_x(t)$ is given by

$$\Delta p_x(t) = \sum_n -ik_n A_n e^{i(n\omega t - k_n x)} \tag{14}$$

Comparison of Eqs. (13) and (14) leads to

$$\frac{\partial Q(t)}{\partial t} = -\frac{1-F_{10}}{\rho_b}\pi a^2\Delta p_x(t) \tag{15}$$

Thus, the acceleration of blood flow can represent the pulsatile pressure gradient.

Now, we relate the above pulsatile parameters in blood flow to radial vibration of the arterial wall. The first-order derivative of the radial wall displacement is the velocity $v_r(t)$ of the arterial wall

$$v_r(t) = \frac{du_r}{dt} = \frac{(1-F_{10})a}{2c\rho_b}\sum_n ik_n A_n e^{i(n\omega t - k_n x)} \tag{16}$$

Comparison of Eqs. (12) and (16) gives rise to the following relation:

$$v_r(t) = \frac{du_r}{dt} = \frac{1}{2\pi a}\frac{\sum_n ik_n A_n e^{i(n\omega t - k_n x)}}{\sum_n A_n e^{i(n\omega t - k_n x)}}Q(t) \tag{17a}$$

By only keeping the first harmonic ($n = 1$) in the equation, the following relation is obtained:

$$v_{r1}(t) = \frac{ik_1}{2\pi a}Q_1(t) \tag{17b}$$

Thus, the first harmonic of the radial velocity can represent the first harmonic of the blood flow rate.

Based on Eq. (16), the second-order derivative of the radial displacement of the arterial wall is the acceleration of the arterial wall is

$$a_r(t) = \frac{d^2u_r}{dt^2} = -\frac{(1-F_{10})a}{2\rho_b}\sum_n k_n^2 A_n e^{i(n\omega t - k_n x)} \tag{18}$$

Then, a combination of Eqs. (18), (13), and (14) gives rise to

$$\begin{aligned} a_r(t) = \frac{d^2u_r}{dt^2} &= \frac{-1}{2\pi a}\frac{\sum_n k_n^2 A_n e^{i(n\omega t - k_n x)}}{\sum_n ik_n A_n e^{i(n\omega t - k_n x)}}\frac{\partial Q(t)}{\partial t} \\ &= \frac{(1-F_{10})a}{2\rho_b}\frac{\sum_n k_n^2 A_n e^{i(n\omega t - k_n x)}}{\sum_n ik_n A_n e^{i(n\omega t - k_n x)}}\Delta p_x(t) \end{aligned} \tag{19a}$$

Similarly, by only keeping the first harmonic in the equation, the above equation is simplified as

$$a_{r1}(t) = \frac{ik_1}{2\pi a}\frac{\partial Q_1}{\partial t} = -\frac{ik_1(1-F_{10})a}{2\rho_b}\Delta p_{x1}(t) \tag{19b}$$

where $\Delta p_{x1}(t)$ is the first harmonic of the pulsatile pressure gradient

$$\Delta p_{x1}(t) = -ik_1\Delta p_1(t) \tag{20}$$

Based on Eq. (19*b*), the first harmonic of the radial acceleration can represent the first harmonic of the acceleration of blood flow and the first harmonic of the pulsatile pressure gradient.

3.2 With Wave Reflection. When wave reflection is considered, the pulsatile pressure input at an artery site becomes

$$\Delta p(t) = \sum_n (A_n e^{-ik_n x} + A'_n e^{ik_n x}) e^{in\omega t} \tag{21}$$

where the first and the second terms denote the forward and backward waves of the pulsatile pressure, respectively. Then, the radial displacement of the arterial wall becomes

$$u_r(t) = \frac{(1-F_{10})a}{2c^2\rho_b}\sum_n \left(A_n e^{-ik_n x} + A'_n e^{ik_n x}\right) e^{in\omega t} \tag{22}$$

Since both $\Delta p(t)$ and $u_r(t)$ are in the radial direction, wave reflection augments them. Based on Eqs. (21) and (22), the waveform of $u_r(t)$ is the same as the waveform of $\Delta p(t)$.

The blood flow rate is written as

$$Q(t) = \frac{1-F_{10}}{c\rho_b}\pi a^2 \sum_n \left(A_n e^{-ik_n x} - A'_n e^{ik_n x}\right) e^{in\omega t} \tag{23}$$

Wave reflection reduces the blood flow rate, due to its axial direction. The acceleration of blood flow $\partial Q(t)/\partial t$ is expressed as

$$\frac{\partial Q(t)}{\partial t} = \frac{1-F_{10}}{\rho_b}\pi a^2 \sum_n ik_n \left(A_n e^{-ik_n x} - A'_n e^{-ik_n x}\right) e^{in\omega t} \tag{24}$$

The pulsatile pressure gradient $\Delta p_x(t)$ is given by

$$\Delta p_x(t) = \sum_n -ik_n (A_n e^{-ik_n x} - A'_n e^{ik_n x}) e^{in\omega t} \tag{25}$$

Comparison of Eqs. (24) and (25) leads to

$$\frac{\partial Q(t)}{\partial t} = -\frac{1-F_{10}}{\rho_b}\pi a^2 \Delta p_x(t) \tag{26}$$

Equations (15) and (26) imply that the waveform of $\partial Q(t)/\partial t$ can represent the waveform of $\Delta p_x(t)$, regardless of wave reflection.

The velocity of the arterial wall is expressed as

$$v_r(t) = \frac{(1-F_{10})a}{2c\rho_b}\sum_n ik_n \left(A_n e^{-ik_n x} + A'_n e^{ik_n x}\right) e^{in\omega t} \tag{27}$$

By keeping only the first harmonic in Eqs. (23) and (27), the following relations are obtained:

$$Q_1(t) = \frac{1-F_{10}}{c\rho_b}\pi a^2 \left(A_1 e^{-ik_1 x} - A'_1 e^{ik_1 x}\right) e^{i\omega t} = Q_{f1}(t) - Q_{b1}(t) \tag{28a}$$

$$v_{r1}(t) = \frac{(1-F_{10})a}{2c\rho_b} ik_1 \left(A_1 e^{-ik_1 x} + A'_1 e^{ik_1 x}\right) e^{i\omega t} \tag{28b}$$

where $Q_{f1}(t)$ and $Q_{b1}(t)$ are the first-harmonic forward and backward waves of the blood flow rate, respectively. A comparison of the above two equations shows

$$v_{r1}(t) = \frac{ik_1}{2\pi a}\left\{Q_{f1}(t) + Q_{b1}(t)\right\} \tag{29}$$

Due to the opposite effects of wave reflection on $v_r(t)$ and $Q(t)$, the waveform of $v_r(t)$ fails to represent the waveform of $Q(t)$, even when only their first harmonics are considered.

The acceleration of the arterial wall is expressed as

$$a_r(t) = -\frac{(1-F_{10})a}{2\rho_b}\sum_n k_n^2 \left(A_n e^{-ik_n x} + A'_n e^{ik_n x}\right) e^{in\omega t} \tag{30}$$

By keeping only the first harmonics of the pulsatile parameters in Eqs. (24), (25), and (30), the following relations are obtained:

$$\frac{\partial Q_1(t)}{\partial t} = \frac{1-F_{10}}{\rho_b}\pi a^2 ik_1 \left(A_1 e^{-ik_1 x} - A'_1 e^{ik_1 x}\right) e^{i\omega t} = \frac{\partial Q_{f1}(t)}{\partial t} - \frac{\partial Q_{b1}(t)}{\partial t} \tag{31a}$$

$$\Delta p_{x1}(t) = -ik_1 (A_1 e^{-ik_1 x} - A'_1 e^{ik_1 x}) e^{i\omega t} = \Delta p_{xf1}(t) + \Delta p_{xb1}(t) \tag{31b}$$

$$a_{r1}(t) = -\frac{(1-F_{10})\pi a}{2c\rho_b} k_1^2 \left(A_1 e^{-ik_1 x} + A'_1 e^{ik_1 x}\right) e^{i\omega t} \tag{31c}$$

where Δp_{xf1} and Δp_{xb1} denote the first-harmonic forward and backward waves of $\Delta p_x(t)$, respectively. A comparison of the above three equations gives rise to

$$a_{r1}(t) = \frac{ik_1}{2\pi a}\left\{\frac{\partial Q_{f1}(t)}{\partial t} + \frac{\partial Q_{b1}(t)}{\partial t}\right\} = -\frac{ik_1 a}{2\rho_b}(1-F_{10})\left\{\Delta p_{xf1}(t) - \Delta p_{xb1}(t)\right\} \tag{32}$$

Due to wave reflection, the waveform of $a_r(t)$ neither represents the waveform of $\partial Q(t)/\partial t$ nor the waveform of $\Delta p_x(t)$, even when only their first harmonics are considered. Note that $\Delta p_{x1}(t)$ is related to $\Delta p_1(t)$ by

$$\Delta p_{x1}(t) = \frac{-ik_1 (A_1 e^{-ik_1 x} - A'_1 e^{ik_1 x})}{(A_1 e^{-ik_1 x} + A'_1 e^{ik_1 x})}\Delta p_1(t) \tag{33}$$

4 Extraction of Arterial Indices With the Arterial Wall as a Unit-Mass Vibration System

4.1 Relation of Radial Vibration of the Arterial Wall to Its Mechanical Properties and Geometries. At an artery site, the pulsatile radial motion of the arterial wall is essentially radial vibration. As such, the arterial wall at an artery site can be modeled as a unit-mass vibration system with pulsatile pressure as the driving force [2,3]

$$M\frac{d^2 u_r}{dt^2} + D\frac{du_r}{dt} + Ku_r = \frac{\Delta p(t)}{\rho h} \tag{34}$$

where the damping coefficient D and the spring stiffness K are related to the elasticity and viscosity of the arterial wall by

$$D = \frac{\eta}{\rho a^2}, \quad K = \frac{E(1-\tau_0)}{\rho a^2} \tag{35}$$

To simplify the measurement method for extraction of arterial indices, the following theory is developed for extracting arterial indices solely from one single measured arterial radial displacement signal with no calibration [2,3]. Figure 2 shows a measured

arterial radial displacement signal and its first-order and second-order derivatives. The key features labeled in the figure can be easily obtained by using built-in functions in MATLAB.

The pulsatile pressure amplitude Δp_0 can be approximated by the difference between the maximum and minimum acceleration of the arterial wall

$$\frac{\Delta p_0}{\rho h} \propto (a_{r\_\max} - a_{r\_\min}) \cdot \Delta t/T \tag{36}$$

Note that $\Delta t/T$ is utilized to factor in difference in heart rate between different conditions (e.g., before versus postexercise) and subjects. Consequently, K and D can be estimated by

$$K \propto \frac{a_{r\_\max} - a_{r\_\min}}{u_{r0}} \cdot \frac{\Delta t}{T}, \quad D \propto \frac{a_{r\_\max} - a_{r\_\min}}{v_{r0}} \cdot \frac{\Delta t}{T} \tag{37}$$

where u_{r0} and v_{r0} are the radial displacement and velocity amplitudes of the arterial wall, respectively.

4.2 Extraction of Arterial Indices. As analyzed in Sec. 2, when wave reflection is neglected and only the first harmonics of the pulsatile parameters are considered, $v_r(t)$ can represent $Q(t)$ and $\Delta p(t)$ can represent $\Delta p_x(t)$. The following analysis for extraction of arterial indices from the key features in Fig. 2 is based on these two assumptions. Note that subscript 1 for the first harmonic is dropped for simplicity here, and subscript 0 denotes the amplitude of a pulsatile parameter. According to Eq. (17*b*), the blood flow rate amplitude Q_0 can be approximated by the velocity amplitude v_{r0} of the arterial wall

$$Q_0 = \frac{2\pi a \cdot v_{r0}}{ik} \tag{38}$$

Based on Eq. (33), the pulsatile pressure gradient amplitude Δp_{x0} is associated with the pulsatile pressure amplitude Δp_0 by

$$\Delta p_{x0} = (-ik)\Delta p_0 \tag{39}$$

The peripheral vascular resistance (PVR) at an artery site is given by [2,3,23]

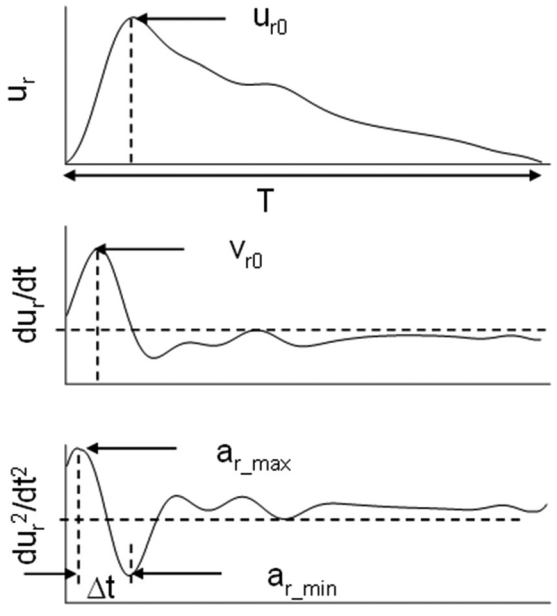

Fig. 2 The radial displacement, velocity, and acceleration of the arterial wall in one pulse cycle, labeled with the associated key features (adapted from Ref. [3])

$$\text{PVR} = \frac{-\Delta p_{x0}}{Q_0} = \frac{8\mu}{\pi a^4} \tag{40}$$

By substituting Eqs. (38) and (39) into Eq. (40), the following relation is obtained:

$$-\frac{k^2 \Delta p_0}{2\pi a \cdot v_{r0}} = \frac{8\mu}{\pi a^4} \tag{41}$$

Note that the wave number k is related to the wavelength λ by

$$k = \frac{\omega}{c} = \frac{2\pi}{\lambda}, \quad \lambda = \frac{c \cdot 60}{\text{HR}} = c \cdot T \tag{42}$$

where HR denotes the heart rate. It is assumed that λ and μ do not vary between subjects and different conditions, substituting Eqs. (36) and (37) into Eq. (41) leads to the following relation:

$$a \propto \left(\frac{v_{r0}}{\Delta p_0}\right)^{1/3} \propto D^{-1/3} \tag{43a}$$

Consequently, based on Eq. (35), the elasticity and viscosity of the arterial wall and *PWV* can be estimated by

$$\eta \propto D^{1/3}, \; E(1-\tau_0) \propto K \cdot D^{-2/3} \tag{43b}$$

$$PWV \cdot \sqrt{1-\tau_0} \propto \sqrt{K \cdot D^{-1/3}} \tag{43c}$$

5 Discussion

With harmonics and wave reflection, the theoretical expressions for radial wall vibration and pulsatile parameters in blood flow better represent the pulsatile parameters measured in clinical studies, and the derived theoretical relations between radial wall vibration and pulsatile parameters in blood flow better manifest the intrinsic relations between them in an artery. Measurement complexity and high-cost prevent from measuring all the pulsatile parameters in an artery for best assessing the cardiovascular system. As such, theoretically solid relations between different pulsatile parameters become more important for interpreting only one measured pulse signal for clinical values with clearly defined physiological implications and assumptions.

5.1 Applicability of the Assumptions in Interpretation of the Radial Vibration of the Arterial Wall for Clinical Values. As shown in Sec. 4, to extract the arterial radius a from radial vibration of the arterial wall, two assumptions are necessary: (1) wave reflection is neglected; and (2) only the first harmonics of the pulsatile parameters are considered. Given that the early time duration of a pulse cycle is considered to be free of wave reflection and the first harmonics of the pulsatile parameters are dominant [22,23]; and v_{r0} and Δp_0 are extracted from the key features in the early time duration of a pulse cycle, these two assumptions are reasonably realistic. Additionally, v_{r0} and Δp_0 are calculated from Eqs. (36) and (37) include both wave reflection and all the harmonics, with wave reflection having the same augmenting effect in v_{r0} and Δp_0. Then, according to Eq. (43*a*), the inclusion of wave reflection and all the harmonics in both v_{r0} and Δp_0 might alleviate the effect of wave reflection and harmonics on the extraction of a. The estimation of arterial indices based on Eq. (43) has been experimentally validated by the measured difference of arterial indices on healthy subjects between at-rest and immediately after postexercise with statistical significance [3].

For measurement simplicity, PPG signals have been utilized to represent other pulsatile parameters. As pointed out earlier, A PPG signal essentially represents the radial displacement of the arterial wall [5,6,8–10]. The velocity of the PPG signal (or

velocity of PPG) is considered to represent the blood flow rate in an artery; the acceleration of the PPG signal is considered to represent the acceleration of blood flow in an artery and consequently the pulsatile pressure gradient in the literature [4–10]. As derived in Sec. 3, interpretation of the velocity and acceleration of a PPG signal as blood flow rate and acceleration of blood flow, respectively, is only valid, when wave reflection is not considered and only the first harmonics of the pulsatile parameters are considered. This may explain the algorithms for adjusting the velocity of a PPG signal to match the waveform of the blood flow rate measured using an imaging instrument in the literature [11,12]. Due to the technical difficulty in measuring Δp_x, the first-order derivative of measured blood flow rate $Q(t)$ was utilized to represent the waveform of $\Delta p_x(t)$ [24]. It is interesting to note that only this representation is valid, regardless of wave reflection and harmonics. Taken together, wave reflection and harmonics modify the relations of different pulsatile parameters and their time-derivatives that are obtained under the assumptions of no wave reflection and one harmonic term [25].

Without relating to pulsatile parameters in blood flow, a PPG signal and its derivatives are also utilized for clinical values via statistical analysis [4,7–10]. Without relating to forward and backward waves of a pulse signal, peaks and valleys in the second-order derivative of a PPG signal were arbitrarily labeled as a, b, c, d, e waves, and different ratios of these peaks and valleys were explored for their clinical values [4,7–10]. Among them, b/a ratio ($|a_{r\_\min}/a_{r\_\max}|$ ratio in Fig. 2) was most studied. It was positively correlated with age and arterial stiffness but was negatively correlated with arterial distensibility [4]. When the second-order derivative of a PPG signal is interpreted as radial wall acceleration, a large b/a ratio implies reduced radial wall displacement and then increased arterial elasticity. With aging, arterial elasticity is increased and arterial distensibility is reduced. As such, the physiological implication of the b/a ratio becomes clear, when it is interpreted in terms of the radial wall acceleration. Similarly, without physiological implications, different features (i.e., peaks and valleys and their time intervals) and their combinations of a PPG signal and its two derivatives have been utilized for estimating blood pressure [9,14,15] in recent years. Given the complexity of the combination of different features [15], the physiological implication of such a combination is not discussed here.

5.2 Characteristic Impedance. Characteristic impedance is an important clinical marker for the CV system and is defined as [23]

$$Z_c = \frac{\Delta p}{Q}\pi a^2 = \frac{\rho_b c}{1 - F_{10}} \tag{44}$$

where Δp and Q denote the forward waves of the two parameters, and according to the above equation, Z_c manifests the wave velocity c and is also affected by F_{10}. The majority of the studies on Z_c neglect F_{10} [22,23,26]. To extract the value of Z_c from the measured Q and Δp, the following equation is utilized:

$$\rho_b \frac{\partial Q}{\partial t} = -\pi a^2 \frac{\partial \Delta p}{\partial x} - 2\tau_w \pi a \tag{45}$$

where τ_w is wall shear stress (wss). When wave reflection is neglected, the pulsatile pressure gradient and wall shear stress [23] can be represented by

$$-\frac{\partial \Delta p}{\partial x} = -\frac{1}{c}\cdot\frac{\partial \Delta p}{\partial t} \tag{46a}$$

$$\tau_w(x,t) = -\frac{aF_{10}}{2c}\cdot\frac{\partial \Delta p}{\partial t} \tag{46b}$$

Substituting Eq. (46) into Eq. (45) gives rise to

$$Z_c = \frac{\rho_b c}{(1 - F_{10})} = -\pi a^2 \frac{\partial \Delta p}{\partial t} \Big/ \frac{\partial Q}{\partial t} \tag{47}$$

Consequently, the value of Z_c can be extracted from the time derivatives of the measured Q and Δp. It is worth emphasizing that the extracted value of Z_c is based on Eq. (47) factors in τ_w and harmonics. In the study of Lucas et al. [23], the authors did not include the influence of F_{10} on Z_c and thus failed to recognize the inclusion of τ_w in the extracted value of Z_c from Eq. (47). Table 1 lists the calculated values of Z_c for the first ten harmonics at the carotid artery, based on the related mechanical properties and geometries [27]. The calculated values for the two parameters without F_{10}: $c_0 = 8.29$ m/s, $\rho_b c_0 = 8741$ Pa·s/m, are independent of harmonics. Evidently, F_{10} (or τ_w) reduces wave velocity but introduces wave attenuation. Meanwhile, the influence of F_{10} on Z_c is overall negligible.

5.3 Evidence of Harmonics in the Measured Acceleration Signal of the Arterial Wall. Due to their small size and low cost, accelerometers have also been utilized to measure the acceleration signal caused by radial vibration of the arterial wall [28]. The measured acceleration signal is then integrated once to obtain the velocity and twice to obtain the displacement of the arterial wall. It was found [28] that the velocity amplitude of the arterial wall is well above $\omega \times u_{r0}$, and the maximum acceleration of the arterial wall is well above $\omega \times v_{r0}$, manifesting the existence of the harmonics in a pulse signal, given the same effect of wave reflection on them. Accordingly, the inclusion of the harmonics in both v_{r0} and Δp_0 in Eq. (43*a*) might alleviate the effect of harmonics on the extraction of a.

5.4 New Insights on the Interaction Between the Arterial Wall and Blood Flow

5.4.1 Energy Associated With the Arterial Wall and Blood Flow. As analyzed in Sec. 2, the pulse wave propagation in an artery is a coordinated response of blood flow and the arterial wall. The maximum potential elastic energy and the maximum kinetic energy in the arterial wall per unit length (along the x-axis) are given by

$$PE_{\text{wall}} = \frac{1}{2}E\left(\frac{u_{r0}}{a}\right)^2 2\pi a h, \quad KE_{\text{wall}} = \frac{1}{2}\rho 2\pi a h v_{r0}^2 \tag{48}$$

Due to a high E and a small a, $KE_{\text{wall}} \ll PE_{\text{wall}}$. The maximum kinetic energy of blood flow per unit length is given by

$$KE_{\text{blood}} = \frac{1}{2}\rho_b \frac{Q_0}{\pi a^2}^2 \tag{49}$$

It is interesting to note that equating PE_{wall} to KE_{blood} gives rise to c_0 in Eq. (7*b*).

Now, it is tempted to conclude that elastic energy of the arterial wall and kinetic energy of blood flow keep exchanging to facilitate blood flow. Yet, according to Eq. (9), since the imaginary part of the wave velocity is much smaller than its real part, $Q(t)$ and $u_r(t)$ reach their maximum values at very similar times in a pulse cycle, suggesting that the blood flow and the arterial wall reach their peak values at similar times. This is opposite to a mass-spring vibration system, in which the kinetic energy of the mass is out of phase with the elasticity energy of the spring. As such, it is unreasonable to treat the blood as the mass and the arterial wall as the spring for an equivalent vibration model, which would further lead to an extremely high angular frequency (or heart rate). Then, the unit-mass vibration model of the arterial wall with the pulsatile pressure from blood flow as the driving force is more realistic to relate the pulsatile radial motion of the arterial wall to pulsatile parameters in blood flow for extraction of arterial indices. Note

Table 1 The calculated values of wave velocity and characteristic impedance for the first ten harmonics at the carotid artery ($a = 3.3$ mm, $h = 0.62$ mm, heart rate = 75 bpm, and $E = 771$ kPa [27])

	c (m/s)		$Z_c = \frac{\rho_b c}{1-F_{10}}$	
n	Real	Imaginary	Value (Pa·s/m)	Phase (deg)
1	6.68	0.33	9607.8	–9.9
2	7.03	0.48	9209.9	–6.7
3	7.18	0.55	9035.0	–5.4
4	7.27	0.60	8932.3	–4.6
5	7.34	0.64	8862.7	–4.1
6	7.38	0.67	8811.7	–3.7
7	7.42	0.69	8772.2	–3.4
8	7.45	0.71	8740.5	–3.2
9	7.47	0.72	8714.4	–3.0
10	7.49	0.73	8692.3	–2.8

that the discussion on the energy involved in an artery here is focused on large healthy arteries with the assumptions in Sec. 2. Whether CV disease affects the energy relation depends on how CV disease affects the relation between radial vibration of the arterial and blood flow rate.

5.4.2 One-Dimensional Wave Equation for the Interaction Between the Arterial Wall and Blood Flow. The pulse wave propagation in an artery results from the interaction between the arterial wall and blood flow, but Eqs. (1) and (3) are totally different from the 1D wave equations. Here, we attempt to derive a 1D wave equation that manifests the interaction between the arterial wall and blood flow. To this end, average axial blood velocity $\bar{u}(x,t)$ is chosen as the pulsatile parameter for deriving a 1D wave equation, given that this parameter directly represents blood circulation. As shown in Fig. 3, for a segment of dx in an artery, its mass m is from the blood flow

$$m = \rho_b \pi a^2 dx \tag{50}$$

The 1D wave equation for $\bar{u}(x,t)$ is written as

$$\frac{\partial^2 \bar{u}}{\partial t^2} = c_0^2 \frac{\partial^2 \bar{u}}{\partial x^2} \tag{51}$$

Note that $\bar{u}(x,t)$ propagates with the same wave velocity as pulsatile pressure and radial displacement of the arterial wall. By substituting Eq. (7*b*) into Eq. (51) and multiplying m on both sides of Eq. (51), the following equation is obtained:

$$\rho_b \pi a^2 \frac{\partial^2 \bar{u}}{\partial t^2} = E_{x-\text{axis}} \cdot \pi a^2 \frac{\partial^2 \bar{u}}{\partial x^2} \quad \text{with} \quad E_{x-\text{axis}} = \frac{Eh}{2a} \tag{52}$$

where $E_{x\text{-axis}}$ arises from the arterial wall.

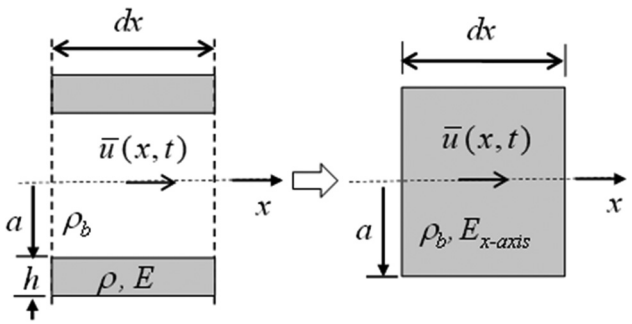

Fig. 3 The interaction between the arterial wall and the blood flow is equivalent to longitudinal wave propagation with average axial blood flow velocity $\bar{u}(x,t)$ as the pulsatile parameter in an elastic rod with elasticity $E_{x\text{-axis}}$, density ρ_b, and cross section area πa^2

The wave equation for an elastic rod is

$$\rho A \frac{\partial^2 u}{\partial t^2} = \text{EA} \frac{\partial^2 u}{\partial x^2} \tag{53}$$

where E, ρ, and A denote the elasticity, density, and cross section area of the elastic rod, respectively, and the pulsatile parameter $u(x,t)$ is the axial displacement of the elastic rod. In an elastic rod, the same medium provides mass and the elasticity. In contrast, in an artery, the arterial wall provides the elasticity $E_{x\text{-axis}}$ and the blood provides the mass m. Comparison of Eqs. (52) and (53) reveals that the interaction between the arterial wall and blood flow is equivalent to longitudinal wave propagation with $\bar{u}(x,t)$as the pulsatile parameter in an elastic rod with elasticity $E_{x\text{-axis}}$, density ρ_b, and cross section area πa^2.

6 Conclusion

With two physiological realities: harmonics and wave reflection, being considered, the theoretical expressions for radial vibration of the arterial wall and pulsatile parameters in blood flow are first derived. Afterward, the theoretical relations between them are derived, revealing the effect of harmonics and wave reflection on these relations. The derived theoretical relations identify the assumptions that are needed in the utility of radial vibration of the arterial wall for extraction of arterial indices. With the arterial wall modeled as unit-mass vibration system, the obtained relations under the assumptions are utilized to relate radial wall vibration to arterial indices, providing a solid theoretical basis for the previous related experimental work [3]. These theoretical relations further identify some misconceptions in the interpretation of a PPG signal and its two derivatives for clinical values. New insights on the interaction between the arterial wall and blood flow are further revealed from the perspective of energy and 1D wave equations. Given the ever-increasing need for measurement simplicity for routine and at-home monitoring of the cardiovascular system, interpretation of only a PPG signal or a pulse signal measured by a tactile sensor for clinical values becomes more important. The derived theoretical expressions for all the pulsatile parameters and their relations in this paper provide theoretical guidance for improving their interpretation of clinical values with clearly defined physiological implications and assumptions.

Funding Data

- National Science Foundation (NSF) (Grant No. 1936005; Funder ID: 10.13039/100000146).

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