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IMPROVED VIBRATION-MODEL-BASED ANALYSIS FOR ESTIMATION OF ARTERIAL PARAMETERS FROM NONINVASIVELY MEASURED ARTERIAL PULSE SIGNALS

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ABSTRACT

With the goal of achieving consistence in interpretation of an arterial pulse signal between its vibration model and its hemodynamic relations and improving its physiological implications in our previous study, this paper presents an improved vibration-model-based analysis for estimation of arterial parameters: elasticity (E), viscosity (η), and radius (r_0) at diastolic blood pressure (DBP) of the arterial wall, from a noninvasively measured arterial pulse signal. The arterial wall is modeled as a unit-mass vibration model, and its spring stiffness (K) and damping coefficient (D) are related to arterial parameters. Key features of a measured pulse signal and its first-order and second-order derivatives are utilized to estimate the values of K and D. These key features are then utilized in hemodynamic relations, where their interpretation is consistent with the vibration model, to estimate the value of r_0 from K and D. Consequently, E, η, and pulse wave velocity (PWV) are also estimated from K and D. The improved vibration-model-based analysis was conducted on pulse signals of a few healthy subjects measured under two conditions: at-rest and immediately post-exercise. With E, r_0, and PWV at-rest as baseline, their changes immediately post-exercise were found to be consistent with the related findings in the literature. Thus, this improved vibration-model-based analysis is validated and contributes to estimation of arterial parameters with better physiological implications, as compared with its previous counterpart.

Keywords: Arterial parameters, arterial pulse signals, tactile sensors, dynamic model, post-exercise, hemodynamic relations

NOMENCLATURE

K	spring stiffness
D	damping coefficient
E	elasticity of the arterial wall
η	viscosity of the arterial wall
r_0	radius of the arterial wall
PWV	pulse wave velocity
PVR	Peripheral vascular resistance
u_r	radial motion
Δp	pulsatile blood pressure
Δp_0	pulsatile pressure amplitude
σ	circumferential stress
h	arterial wall thickness
ε	circumferential strain
M	equivalent mass
ρ_w	arterial wall density
ρ_b	blood density
η_b	blood viscosity
v	velocity
a	acceleration
T	one pulse cycle time-period
Δt	time duration between a_{max} and a_{min}
Q	blood flow rate
HR	Heart Rate

1. INTRODUCTION

To assess the physiological and pathological condition of the arterial wall, arterial pulse signals are noninvasively measured by various instruments and their pulse waveforms are then processed to estimate arterial indices in clinical studies [1, 2]. To date, arterial stiffness is a well-established clinical index for arterial health assessment. Currently, the gold standard for arterial stiffness is carotid-femoral pulse wave velocity (cf-PWV), a global arterial stiffness [3]. This cf-PWV is aimed to

quantify arterial elasticity, E, but it is also affected by arterial radius r_0 at diastolic blood pressure (DBP), as will be seen later on. The value of cf-PWV is estimated from the two pulse waveforms simultaneously measured at the carotid artery and the femoral artery. In this estimation, arterial pulse signals are treated as a propagation wave from the left ventricle to periphery. To estimate local E and local viscosity, η, at an artery, both the pulsatile pressure signal and the radial motion signal of the artery need to be simultaneously measured and complex algorithms are then utilized to process both their amplitudes and waveforms to estimate the values of E and η at the artery [4]. In this estimation, the arterial wall at an artery is treated as a viscoelastic material with E and η in the circumferential direction. Note that inertia of the arterial wall in its radial motion is neglected.

Endotholium function is also a well-established clinical index for arterial health assessment, and is a measure of the response of arterial radius to blood flow change in it, and this response is dependent on both η and r_0 of the arterial wall [5, 6]. Meanwhile, peripheral vascular resistance (PVR) is also important to the cardiovascular (CV) system and is determined by r_0, as will be seen later on. Thus, an imaging instrument is needed to measure endothelium function and PVR [7]. Taken together, arterial stiffness, endothelium function, and PVR are about estimation of arterial parameters: elasticity (E), viscosity (η) and radius (r_0) of the arterial wall.

Given high-cost of an imaging instrument and operation complexity of measuring two pulse signals simultaneously at two artery sites for cf-PWV, we previously developed vibration-model-based analysis for estimating three arterial parameters and PWV from only one measured pulse signal with no need of calibration [8]. In the analysis, the arterial wall was treated as a unit-mass vibration model, and its spring stiffness and damping coefficient were related to arterial parameters. The values of spring stiffness and damping coefficient were extracted from the key features of a measured pulse signal and its first-order and second-order derivatives. However, to estimate the value of arterial radius from hemodynamic relations, the measured pulse signal and its two derivatives were interpreted with i) a lack of consistency with their usage in the vibration model and ii) a simplified physiological implications.

In order to improve the vibration-model-based analysis in our previous study [8], this work examines non-simplified physiological implications of a measured pulse signal and its two derivatives in hemodynamic relations and also removes the non-consistent interpretation of a measured pulse signal and its two derivatives between the vibration model and hemodynamic relations. Consequently, new estimations of arterial parameters and PWV in terms of spring stiffness and damping coefficient are obtained. Based on these new estimations, the measured pulse signals in the previous study [8] were processed again to estimate the values of arterial parameters and PWV, and were compared with the related findings in the literature for validation.

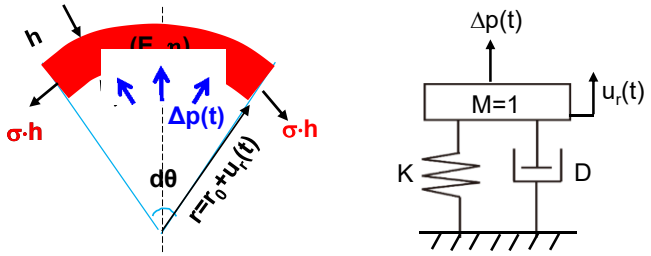
2. VIBRATION-MODEL-BASED ANALYSIS

2.1 Unit-mass vibration model of the arterial wall

At the start/end of a pulse cycle, the arterial wall is in equilibrium. As shown in Fig. 1(a), upon pulsatile blood pressure, Δp(t), the arterial wall undergoes radial motion, $u_r(t)$. The arterial wall in the circumferential direction is treated as a viscoelastic material with elasticity, E, and viscosity, η, to capture the relation of Δp(t) to $u_r(t)$. In a pulse cycle, radial motion causes circumferential stress, σ, in the arterial wall [8]:

$$\sigma = E\cdot\varepsilon + \eta\cdot\frac{d\varepsilon}{dt} \quad \text{with} \quad \varepsilon = \frac{u_r}{r_0} \tag{1}$$

where ε denotes the circumferential strain in the arterial wall.



(a) (b)

FIGURE 1: Schematics of the arterial wall (a) forces acting on a small element dθ of the arterial wall per unit length (b) unit-mass vibration model

The force acting on one side of the small element dθ of the arterial wall per unit length is: σ·h. The mass of this element is $\rho_w\cdot d\theta\cdot r_0\cdot h$, with ρ_w denoting arterial wall density. Equating inertial force of the element to all the forces acting on it leads to the following equation:

$$\rho_w h r_0 d\theta dx\cdot\frac{d^2u_r}{dt^2} = \Delta p\cdot r_0\cdot d\theta\cdot dx - (E\cdot\varepsilon\cdot h\cdot d\theta\cdot dx + \eta\cdot\frac{d\varepsilon}{dt}\cdot h\cdot d\theta\cdot dx) \tag{2}$$

Integrating both sides of Eq. (2) over 2π gives rise to the following equation:

$$\rho_w h r_0 2\pi\frac{d^2u_r}{dt^2} + \frac{h}{r_0}2\pi\eta\cdot\frac{du_r}{dt} + \frac{h}{r_0}2\pi E\cdot u_r = r_0 2\pi\Delta p \tag{3}$$

The above equation indicates that the arterial wall per unit length can be treated a whole-wall vibration model:

$$M_{wall}\cdot\frac{d^2u_r}{dt^2} + D_{wall}\cdot\frac{du_r}{dt} + K_{wall}\cdot u_r = r_0 2\pi\Delta p \tag{4}$$

where

$$M_{wall} = \rho_w h 2\pi r_0,\ K_{wall} = \frac{h}{r_0}2\pi E,\ D_{wall} = \frac{h}{r_0}2\pi\eta \tag{5}$$

Note that M_{wall}, K_{wall} and D_{wall} denote the equivalent mass, spring stiffness, and damping coefficient, respectively, of the arterial wall per unit length. By dividing both sides of Eq. (4) by M_{wall}, a unit-mass vibration model of the arterial wall is obtained:

$$M\frac{d^2u_r}{dt^2}+D\cdot\frac{du_r}{dt}+K\cdot u_r=\frac{\Delta p(t)}{\rho_w h} \tag{6}$$

where

$$\text{M=1},\ K=\frac{E}{\rho_w r_0^2},\ D=\frac{\eta}{\rho_w r_0^2} \tag{7}$$

Here, K and D denote the spring stiffness and damping coefficient, respectively, of the arterial wall per unit mass.

To obtain values of K and D from a measured pulse signal, the measured pulse signal is treated as radial motion, $u_r(t)$, of the arterial wall. Then, its first-order and second-order derivatives represent velocity, v(t), and acceleration, a(t), respectively, of the arterial wall. For the purpose of simplifying the measurement and avoiding extra measurement errors, the driving force, pulsatile pressure, is not measured. Then, the driving force amplitude is approximated by:

$$\frac{\Delta p_0}{\rho_w h}\propto(a_{\max}-a_{\min})\cdot\Delta t/T \tag{8}$$

where Δp_0, denotes pulsatile pressure amplitude; a_{max} and a_{min} denote the maximum and minimum, respectively, of the acceleration; Δt denotes the time duration between a_{max} and a_{min}; and T denotes the one pulse cycle time period. Note that inclusion of $\Delta t/T$ is aimed to factor in difference in heart rate between subjects and different conditions. The damping force amplitude and spring force amplitude of the arterial wall in a pulse cycle can then be approximated by $D\cdot v_{\max}$ and $K\cdot u_{r0}$, respectively. Consequently, K and D in the unit-mass vibration model of the arterial wall can be obtained from the following six key features: u_{r0}, v_{max}, a_{max}, a_{min}, Δt, and T, of the measured pulse signal and its two derivatives:

$$D=\frac{(a_{\max}-a_{\min})\cdot\frac{\Delta t}{T}}{v_{\max}},\quad K=\frac{(a_{\max}-a_{\min})\cdot\frac{\Delta t}{T}}{u_{r0}} \tag{9}$$

Related data-processing algorithms were written in matlab to estimate the key features of a measured pulse signal and its two derivatives. The details about these key features and the algorithms for their extraction can be found in the literature [6, 8].

2.2 Estimation of arterial radius from hemodynamic relations

In the previous work, blood flow rate was approximated by velocity of the arterial wall and blood pressure gradient was approximated by pulsatile pressure amplitude directly [8]. However, blood flow rate, Q, is also related to r_0. In fact, blood volume change in an artery, $2\pi r_0\cdot v(t)$, serves better as an indicator of blood flow rate [9]. Then, Q can be approximated by:

$$Q\propto 2\pi r_0\cdot v_{\max} \tag{10}$$

According to Eq. (8), the pulsatile pressure amplitude, Δp_0, is expressed by

$$\Delta p_0=\rho_w h(a_{\max}-a_{\min})\cdot\frac{\Delta t}{T} \tag{11}$$

Pressure gradient, Δp_x, is related to pulsatile pressure amplitude by [10]:

$$\Delta p_x=-\frac{1}{PWV}\cdot\Delta p_0 \tag{12}$$

PWV represents the propagation velocity of pulsatile pressure wave along the arterial tree and it is related to E and r_0:

$$PWV=\sqrt{\frac{Eh}{2r_0\rho_b}} \tag{13}$$

where ρ_b denotes blood density. A combination of Eq. (11) ~ (13) gives rise to an approximation of pressure gradient:

$$\Delta p_x\propto\frac{\rho_w h}{PWV}\cdot(a_{\max}-a_{\min})\cdot\frac{\Delta t}{T} \tag{14}$$

Peripheral vascular resistance, PVR, is given by [8, 10]:

$$PVR=\frac{\Delta p_x}{Q}=\frac{8\eta_b}{\pi r_0^4} \tag{15}$$

where η_b denotes blood viscosity. Substituting Eq. (10) and Eq. (14) into Eq. (15) gives rise to estimation of r_0 in terms of K and D:

$$r_0^{5/2}\propto\frac{\eta_b}{\sqrt{\rho_w\cdot\rho_b\cdot h}}\cdot\frac{\sqrt{K}}{D} \tag{16}$$

Since ρ_w, ρ_b, η_b, and h are considered as constants in response to short-time external stimuli, arterial radius can be estimated by the following relation:

$$r_0\propto(K/D^2)^{1/5} \tag{17}$$

2.3 Estimation of arterial parameters and PWV

Substituting Eq. (17) into Eq. (7) gives rise to estimation of arterial elasticity and arterial viscosity:

$$E\propto K^{7/5}\cdot D^{-4/5},\quad \eta\propto K^{2/5}\cdot D^{1/5} \tag{18}$$

In clinical studies, the most commonly used arterial index for arterial health assessment is pulse wave velocity (PWV), and now it can also be estimated by K and D:

$$PWV = \sqrt{\frac{Eh}{2r_0\rho_b}} \propto K^{3/5} \cdot D^{-1/5} \quad (19)$$

Note that estimations of arterial parameters and PWV in Eqs. (17)~(19) are obtained via a scaling analysis, and thus they are all relative values and have no unit. Given that noninvasively measured pulse signals are affected by the instrument used and subject variations, absolute estimated values of arterial parameters and PWV bear their influence and might not be appropriate for comparison between subjects [8, 11]. To remove the influence of subject variations, we use changes in arterial parameters and PWV in response to a bout of moderate exercise to evaluate validity of this improved vibration-model-based analysis of measured pulse signals in the following section.

3. MEASURED RESULTS AND DISCUSSION

The measured pulse signals on a few healthy subjects were collected under approval by the Institutional Review Board (IRB) of Old Dominion University (ODU). The details about the related measurement protocols can be found in the literature [6, 8]. A brief description of the measured pulse signals is provided here for their use in validating the improved vibration-model-based analysis. Under one measurement protocol [8], pulse signals at the radial artery (RA) and the carotid artery (CA) of five healthy subjects (subjects: 1-5 in Table 1) were collected simultaneously using two microfluidic tactile sensors at-rest and immediately after each subject conducted approximately 5min moderate exercise. Under another measurement protocol [6], pulse signals at the RA of two healthy subjects (subjects: 6-7 in Table 1) were collected simultaneously using a microfluidic tactile sensor at-rest and immediately after each subject conducted approximately 5min moderate exercise. The details about the subjects' characteristics can be found in the literature [6, 8]. All the statistical analysis was conducted in IBM SPSS.

3.1 Post-exercise changes of arterial parameters and PWV, relative to at-rest

The estimated values of arterial parameters and the related parameters of the above-mentioned subjects at-rest at the RA and the CA are summarized in Table 1. Note that the values of these parameters vary randomly between subjects. As mentioned in Sec. 2, the difference in the estimated values between subjects does not differentiate their arterial health condition, due to the influence of individual variations on the estimated values. Note that the estimated values at the RA and the CA were combined for comparison of arterial parameters and the related parameters between at-rest and immediately post-exercise.

As shown in Fig. 2, there are no outliers in the estimated values of arterial parameters and the related parameters under at-rest and immediately post-exercise. Fig. 3 compares the estimated values of all the parameters between at-rest and immediately post-exercise. As compared with at-rest, an increase in E, η, and PWV and a decrease in r_0 were observed immediately post-exercise with statistical significance. The observed changes in E, r_0, and PWV are consistent with the related findings obtained using medical instruments in the literature [8].

TABLE 1 Estimated arterial parameters, K, D, PWV and heart rate (HR) at-rest (a) at the RA of seven subjects (b) at the CA of five subjects

(a)

Subject	K	D	E	η	r_0	PWV	HR
1	177.20	5.37	366.15	11.10	1.44	15.96	61.61
2	243.39	10.28	339.72	14.35	1.18	16.96	90.40
3	256.87	7.23	485.36	13.67	1.37	18.79	79.88
4	259.37	7.68	469.19	13.89	1.34	18.68	83.98
5	228.31	8.82	351.26	13.57	1.24	16.83	75.66
6	245.45	7.49	443.04	13.52	1.34	18.16	84.44
7	211.05	7.85	345.19	12.85	1.28	16.43	66.02

(b)

Subject	K	D	E	Eta	r0	PWV	HR
1	154.28	5.50	296.17	10.55	1.39	14.62	62.36
2	320.87	8.84	564.39	15.55	1.33	20.63	90.84
3	306.99	9.53	499.61	15.51	1.28	19.79	80.59
4	262.99	7.42	491.73	13.87	1.37	18.96	85.85
5	241.43	8.25	400.49	13.69	1.29	17.63	76.53

Table 2 compares changes in the parameters at the RA and the CA of subjects 1-5 immediately post-exercise, as compared with at-rest. Although the RA revealed a relatively large change in all the estimated parameters, as compared with the CA, these changes did not carry statistical significance, based on paired Student's t-test.

Table 3 compares the difference of the estimated values of the parameters at the RA and the CA of subjects 1-5 under two conditions: at-rest and immediately post-exercise. Although the estimated values of E, η, r_0, and PWV at the RA under both conditions were observed to be small, as compared with their counterparts at the CA, this difference did not reveal statistical significance, based on paired Student's t-test.

3.2 Discussion

As compared with our previous work, a measured pulse signal and its two derivatives are interpreted in hemodynamic relations with their same physical meanings in the dynamic model, giving rise to new estimations of arterial parameters and PWV in terms of K and D. Based on Eq. (7) and (17), arterial radius is related to both arterial elasticity and viscosity:

$$r_0 \propto E^{1/3} \cdot \eta^{-2/3} \quad (20)$$

Eq. (20) is consistent with CV physiology, in the sense that the three arterial parameters are simultaneously regulated by the CV system to accommodate the extra load raised by exercise. Furthermore, decrease in r_0 immediately post-exercise entails higher PVR, which is accompanied by increase in η for

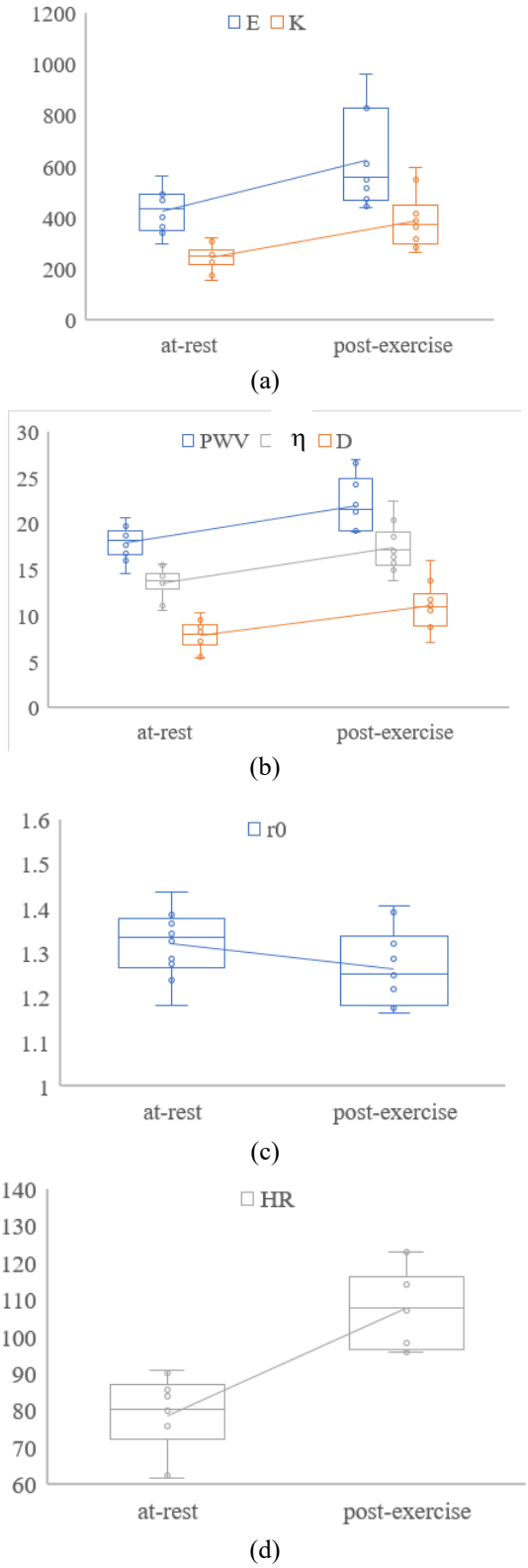


FIGURE 2: Boxplot of arterial parameters and the related parameters at-rest and immediately post-exercise (a) K, E (b) PWV, D, and η (c) r_0 (d) HR

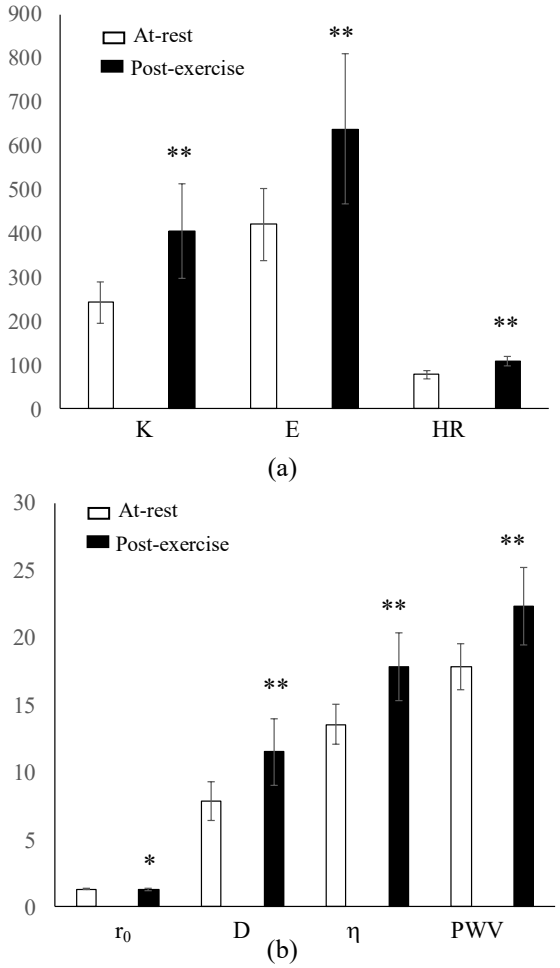


FIGURE 3: Comparison in (a) estimated values of K, E, and HR (b) estimated values of r_0, D, η, and PWV between at-rest and immediately post-exercise using paired Student's t-test (mean ± standard deviation, * $p<0.05$, ** $p<0.001$)

TABLE 2 Changes in arterial parameters and the related parameters immediately post-exercise, as compared with at-test at the RA and the CA

Parameter change	Artery	Mean	Standard Deviation (std)
ΔK	RA	147.68	81.65
	CA	140.39	110.79
ΔD	RA	3.03	1.39
	CA	3.32	3.32
ΔE	RA	208.13	167.19
	CA	185.70	133.96
Δη	RA	3.95	1.54
	CA	3.74	2.80
Δr_0	RA	-0.0574	0.0853
	CA	-0.0549	0.1017
ΔPWV	RA	4.32	2.31
	CA	3.87	2.47
ΔHR	RA	29.04	9.94
	CA	28.75	9.51

TABLE 3 Comparison of the estimated values of arterial parameters and the related parameters between the RA and the CA (a) at-rest (b) immediately post-exercise

(a)

Parameter	Artery	Mean	Standard Deviation (std)
K	RA	233.03	33.57
	CA	257.31	65.97
D	RA	7.88	1.83
	CA	7.91	1.56
E	RA	402.33	69.28
	CA	450.48	104.16
Eta	RA	13.32	1.27
	CA	13.84	2.04
r0	RA	1.316	0.103
	CA	1.329	0.048
PWV	RA	17.44	1.24
	CA	18.33	2.35
HR	RA	78.31	10.80
	CA	79.23	10.87

(b)

Parameters	Artery	Mean	Standard Deviation (std)
K	RA	380.70	102.75
	CA	397.70	129.75
D	RA	10.91	1.14
	CA	11.23	3.62
E	RA	610.46	207.81
	CA	636.18	184.18
Eta	RA	17.26	2.02
	CA	17.58	3.27
r0	RA	1.258	0.054
	CA	1.274	0.116
PWV	RA	21.77	3.16
	CA	22.20	3.24
HR	RA	107.35	11.60
	CA	107.99	11.16

heat dissipation. E increases and r_0 decreases simultaneously, in order to increase PWV for facilitating increased blood flow. Overall, changes in three arterial parameters might be coordinated by the CV system to accommodate external stimuli.

4. CONCLUSION

In this paper, an improved vibration-model-based analysis of measured pulse signals for estimation of arterial parameters has been presented. As compared with the previous study [8], interpretation of a measured pulse signal and its two derivatives in hemodynamic relations is consistent with their interpretation in the vibration model; their physiological implications in hemodynamic relations are also improved. Therefore, new estimations of arterial parameters and PWV with improved physiological implications are obtained. These new estimations are further validated by the expected changes in arterial parameters and PWV immediately post-exercise, as compared with at-rest.

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