1 **Comparison of the Windkessel model and structured-tree model**
2 **applied to prescribe outflow boundary conditions for a**
3 **one-dimensional arterial tree model**

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Abstract

One-dimensional (1D) modeling is a widely adopted approach for studying wave propagation phenomena in the arterial system. Despite the frequent use of the Windkessel (WK) model to prescribe outflow boundary conditions for 1D arterial tree models, it remains unclear to what extent the inherent limitation of the WK model in describing wave propagation in distal vasculatures affect hemodynamic variables simulated at the arterial level. In the present study, a 1D model of the arterial tree was coupled respectively with a WK boundary model and a structured-tree (ST) boundary model, yielding two types of arterial tree models. The effective resistances, compliances and inductances of the WK and ST boundary models were matched to facilitate quantitative comparisons. Obtained results showed that pressure/flow waves simulated by the two models were comparable in the aorta, whereas, their discrepancies increased towards the periphery. Wave analysis revealed that the differences in reflected waves generated by the boundary models were the major sources of pressure wave discrepancies observed in large arteries. Additional simulations performed under aging conditions demonstrated that arterial stiffening with age enlarged the discrepancies, but with the effects being partly counteracted by physiological aortic dilatation with age. These findings suggest that the method adopted for modeling the outflow boundary conditions has considerable influence on the performance of a 1D arterial tree model, with the extent of influence varying with the properties of the arterial system.

Keywords: One-dimensional model, Arterial tree, Outflow boundary conditions, Windkessel model, Structured-tree model, Pressure wave

1. Introduction

One-dimensional (1D) models have been widely employed as a useful tool for studying wave propagation phenomena in the arterial system (Stergiopoulos et al., 1992; Sherwin et al., 2003; Liang et al., 2009a; Müller and Toro, 2014; Mynard et al., 2015). In most of the cases, 1D modeling is limited to large arteries, which requires the definition of outflow boundary conditions so that wave reflections at the distal ends of peripheral arteries can be captured. The Windkessel (WK) model has been frequently used to prescribe outflow boundary conditions for 1D arterial tree models (Alastruey et al., 2008; Stergiopoulos et al., 1992; Reymond et al., 2009). Physiologically, the WK model is defective because it overlooks the detailed anatomical and mechanical properties of the vascular system being modeled (Shi et al., 2011; Westerhof et al., 2009), thus compromising its capability for describing wave propagation phenomena. When the model is applied to represent the vascular systems distal to an arterial tree, the limitation may propagate upstream to compromise the fidelity of simulated pressure/flow waves in large arteries. By contrast, the structured-tree (ST) model provides a more sophisticated means to characterize the dynamic pressure-flow relationship in a distal vascular system with multiple generations of bifurcation (Olufsen et al., 1999, Olufsen et al., 2000; Cousins et al., 2013; Cousins et al., 2014). Advanced applications of ST-like models have been reported as well (Perdikaris et al., 2015). The ST model, given the inherent assumptions, is by far a simplified representation of the actual vascular anatomy which is usually organ/tissue-specific (Blanco et al., 2014, 2015), nevertheless, it provides a relatively reasonable approach to modeling smaller distal vessels whose anatomical parameters are not explicitly known, such as arterioles.

1 Theoretically, the differences of the WK and ST models can be assessed by comparing the
2 impedances resulting from the models. However, it remains unclear how the differences in
3 impedance affect the characteristics of pressure/flow waves simulated at the arterial level. In
4 the present study, a 1D model of the arterial tree was coupled respectively with a WK outflow
5 boundary model and a ST outflow boundary model to construct two types of arterial tree
6 model (namely, 1D-WK model and 1D-ST model). To permit quantitative comparisons, in
7 addition to uniform parameter assignment for the 1D model, parameters used in the WK and
8 ST models were tuned in such a way that the effective resistance, compliance and inductance
9 were equivalent for the two models. Moreover, the two arterial tree models were compared in
10 the context of aging to investigate whether the sensitivity of simulated pressure/flow waves to
11 outflow boundary models is affected by the properties of large arteries.

12

13 **2. Methods**

14 1D modeling was applied to the fifty-five largest arteries ([Liang et al., 2009a,b](#)). The
15 distal ends of the peripheral arteries were supported by a WK or a ST outflow boundary
16 model that represents the distal vascular systems ([see Fig.1](#)). The entrance of the ascending
17 aorta was connected to a lumped-parameter model of the left heart so that the resulting model
18 can account for ventricular-arterial coupling and spontaneously capture hemodynamic
19 changes resulting from the application of different outflow boundary models.

20 **2.1 Governing equations of the 1D model**

21 1D governing equations for blood flows in an artery can be obtained by integrating the
22 three-dimensional Navier-Stokes equations over the cross-section of the artery ([Formaggia et](#)
23 [al., 2006](#)). Obtained 1D hemodynamic equations are expressed as ([Liang et al., 2009b](#))

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial z} = 0, \quad (1)$$

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial z} \left(\alpha \frac{Q^2}{A} \right) + \frac{A}{\rho} \frac{\partial P}{\partial z} + F_r \frac{Q}{A} = 0, \quad (2)$$

where t is the time and z the axial coordinate; A , Q and P represent the cross-sectional area, volume flux and pressure, respectively; α is the momentum-flux correction coefficient and F_r the friction force per unit length. Herein, a Poiseuille cross-sectional velocity profile was assumed, based on which, α , F_r were calculated to be $4/3$, $-8\pi\mu$ (μ is the kinematic viscosity), respectively.

The system of Eqs.(1) and (2) was completed by a constitutive equation that accounts for the viscoelastic deformation of arterial wall (Bessems et al., 2008; Liang et al., 2013).

$$P + \tau_\sigma \frac{\partial P}{\partial t} = \phi(A) + \tau_\epsilon \frac{\partial \phi(A)}{\partial t}, \text{ with } \phi(A) = \frac{Eh}{r_0(1 - \sigma^2)} \left(\sqrt{\frac{A}{A_0}} - 1 \right) + P_0 \quad (3)$$

Here, τ_σ and τ_ϵ refer respectively to the relaxation times for constant stress and strain; P_0 is the reference pressure; E is the Young's modulus; h is the wall thickness; σ is the Poisson's ratio, here taken to be 0.5; A_0 and r_0 represent the cross-sectional area and radius of artery at the reference pressure, respectively.

The 1D hemodynamic equations of each artery were linked to those of the neighboring arteries by imposing continuity of mass flux and total pressure at the bifurcations so that blood flows can be solved for the entire arterial tree (Liang et al., 2009a,b; Mynard et al., 2008).

2.2 Modeling of the left heart

The classical elastance model was employed to account for the pumping function of the left heart (including the left atrium and ventricle) (see Fig.1). The mathematical

expressions followed from those used in our previous studies (Liang et al., 2014b). The mitral and aortic valves were modeled in such way that no reversed flows are allowed to occur. For more details on the heart model, please refer to our previous studies (Liang et al., 2014a,b).

2.3 Modeling of the outflow boundary conditions of the artery tree

Vasculatures distal to the peripheral arteries are featured by complex anatomical structures and heterogeneous mechanical properties. Anatomically, the distal vasculatures can be categorized into distal arteries, arterioles, capillaries, venules and veins, among which distal arteries and arterioles are the major determinant of the wave reflection conditions at the distal ends of peripheral arteries and were herein modeled using a WK model and a ST model, respectively.

2.3.1 Windkessel (WK) model

The WK model consists of four elements, namely, two resistors (R_1 , R_2), a capacitor (C) and an inductor (L) (see Fig. 1). These elements correspond to the lumped properties of the distal vasculatures, including the viscous resistance, the distensibility of vascular walls and the inertia of blood. The input impedance (Z) of the model can be expressed in the frequency domain as

$$Z(\omega) = R_1 + \frac{R_2 + i\omega L}{1 + iR_2\omega C - \omega^2 LC}, \quad (4)$$

where ω is the angular frequency, the sum of R_1 and R_2 ($R_T = R_1 + R_2$) represents the total resistance of the distal vasculatures.

2.3.2 Structured-tree (ST) model

The ST model was constructed based on a prescribed structured tree topology of the distal arteries and arterioles (Olufsen et al., 1999; Olufsen et al., 2000) (see Fig. 2). The input

1 impedance of a structured tree can be deduced through superimposing the separate
 2 impedances of all branch vessels along the tree. To facilitate mathematical operation, the
 3 impedance of each branch vessel was expressed in its reverse form using an admittance
 4 matrix (Qureshi et al., 2014)

$$5 \quad \mathbf{Y} = \begin{pmatrix} Y_{11} & Y_{12} \\ Y_{21} & Y_{22} \end{pmatrix}, \quad (5)$$

6 so that the pressures and flows at the proximal and distal ends of a vessel can be related by

$$7 \quad \begin{pmatrix} Q_p \\ Q_d \end{pmatrix} = \mathbf{Y} \begin{pmatrix} P_p \\ P_d \end{pmatrix}. \quad (6)$$

8 Here, Y_{ij} are functions of the geometrical and mechanical parameters of the vessel, a detailed
 9 definition for each Y_{ij} has been given in (Qureshi et al., 2014). Q_p , Q_d , P_p and P_d represent the
 10 flow rates and pressures at the proximal and distal ends of the vessel, respectively.

11 The admittances of neighboring vessels can be combined through solving the mass
 12 conservation and pressure continuity equations at the junction points (Qureshi et al., 2014).
 13 Taking the bifurcation structure highlighted in Fig.2 as an example, for the parallel vessels ‘a’
 14 and ‘b’ (their admittance matrices are denoted by $\mathbf{Y}^a$ and $\mathbf{Y}^b$, respectively), the combined
 15 admittance matrix ($\mathbf{Y}^P$) is calculated by

$$16 \quad \mathbf{Y}^P = \mathbf{Y}^a + \mathbf{Y}^b. \quad (7)$$

17 Since vessels ‘a’ and ‘b’ are again connected in series with vessel ‘c’ (admittance matrix
 18 denoted by $\mathbf{Y}^c$), the combined total admittance matrix ($\mathbf{Y}^T$) of the bifurcation structure can
 19 thus be obtained by

$$20 \quad \mathbf{Y}^T = \frac{1}{Y_{22}^P + Y_{11}^c} \begin{pmatrix} \det(\mathbf{Y}^P) + Y_{11}^P Y_{11}^c & -Y_{12}^P Y_{12}^c \\ -Y_{21}^P Y_{21}^c & \det(\mathbf{Y}^c) + Y_{22}^P Y_{22}^c \end{pmatrix}. \quad (8)$$

21 Following the admittance operation expressed in Eqs.(7) and (8), we were able to derive a

unitary admittance for the entire structured tree by recursively combining the admittances of branch vessels from the distal ends up to the root.

2.4 Numerical methods

Numerical methods used to solve the 1D model together with the WK boundary model and the heart model followed from those developed in our previous studies (Liang et al., 2009a). It is noted that viscoelasticity has been neglected at the boundaries and junctions of the arterial tree to simplify the numerical schemes, which is similar to the approaches employed in (Alastruey et al., 2011; Wang et al., 2015). In this study, we focused on developing numerical methods for coupling the 1D model and the ST boundary model. Herein, an iterative approach was adopted to ensure the conservation of mass flux and momentum across the model interfaces. Taking the interface located at the distal end of a peripheral artery as an example, the numerical procedure is illustrated in the right panel of Fig.1 (see Appendix A for a more detailed depiction).

For a ST model with its total admittance being denoted by $\mathbf{Y}^{\text{ST}}$, the flow rates and pressures at the entrance ($Q_{\text{in}}^{\text{ST}}, P_{\text{in}}^{\text{ST}}$) and distal end ($Q_{\text{ou}}^{\text{ST}}, P_{\text{ou}}^{\text{ST}}$) were related by

$$\begin{pmatrix} Q_{\text{in}}^{\text{ST}} \\ Q_{\text{ou}}^{\text{ST}} \end{pmatrix} = \mathbf{Y}^{\text{ST}} \begin{pmatrix} P_{\text{in}}^{\text{ST}} \\ P_{\text{ou}}^{\text{ST}} \end{pmatrix}. \quad (9)$$

Rewriting Eq.(9) in algebraic form, we obtained

$$\begin{cases} P_{\text{in}}^{\text{ST}} = A Q_{\text{in}}^{\text{ST}} + B P_{\text{ou}}^{\text{ST}} \\ Q_{\text{ou}}^{\text{ST}} = C P_{\text{in}}^{\text{ST}} + D P_{\text{ou}}^{\text{ST}} \end{cases}, \text{ with } A = \frac{1}{Y_{11}^{\text{ST}}}, B = -\frac{Y_{12}^{\text{ST}}}{Y_{11}^{\text{ST}}}, C = Y_{21}^{\text{ST}}, D = Y_{22}^{\text{ST}}. \quad (10)$$

Eq.(10) was subsequently transformed to the time domain using convolution so that hemodynamic variables calculated by the ST model can be used to perform time step-based 1D-ST model coupling at the interface.

$$\begin{cases} p_{\text{in}}^{\text{ST}}(t) = \int_0^T a(\tau) q_{\text{in}}^{\text{ST}}(t-\tau) d\tau + \int_0^T b(\tau) p_{\text{ou}}^{\text{ST}}(t-\tau) d\tau \\ q_{\text{ou}}^{\text{ST}}(t) = \int_0^T c(\tau) p_{\text{in}}^{\text{ST}}(t-\tau) d\tau + \int_0^T d(\tau) p_{\text{ou}}^{\text{ST}}(t-\tau) d\tau \end{cases} \quad (11)$$

From Eq.(11), assuming a constant pressure at the outlet of the structured tree is not pre-requisite and flow rate at the outlet can be solved simultaneously, which overcomes the limitations of the methods employed in previous studies (Olufsen et al., 2000; Cousins et al., 2013).

2.5 Parameter assignment

2.5.1 Geometrical and mechanical parameters of the 1D model

The parameters used in the 1D model were assigned based on the data reported in previous studies (Liang et al., 2009a,b). For τ_σ and τ_ϵ used in the viscoelastic model (Eq.(3)), since data corresponding to each artery are not derivable from the literature, they were estimated using a piecewise function of artery radius based on the experimental data reported in (Valdez-Jasso et al., 2009) (see Table 1).

2.5.2 Parameter matching for the WK model and the ST model

The effective resistances, compliances and inductances of the ST and WK models must be aligned to enable quantitative model comparisons. We modified the topological structure of each structured tree by tuning the scaling factor α so that the effective resistance of the structured tree is equal to that of the corresponding WK model used in our previous studies (Liang et al., 2009a,b). Similarly, the total effective compliance of each structured tree was tuned to fit the WK model by modifying the elastic parameters. Finally, the total inductance was calculated and used to directly assign the inductance parameter in the WK model. The resulting parameter values are summarized in Table 2.

2.5.3 Parameter adjustment for aging

Aging primarily affects large elastic arteries, resulting in stiffening of arterial wall and lumen dilatation (O'Rourke and Hashimoto, 2007). Relatively, the structural changes of peripheral muscular arteries/arterioles with age are mild (James et al., 2006), although there may appear impaired vasomotor function or endothelial dysfunction in advanced age, which would affect hemodynamic regulation under stress (e.g., exercise) rather than the hemodynamic profile in resting conditions (O'Rourke and Hashimoto, 2007). In addition, the mechanical function (e.g., elastic modulus, compliance) of small arteries and arterioles was found to be preserved during normal aging (Moreau et al., 1998). Therefore, we introduced the effects of aging by modifying the parameters of large elastic arteries (details have been given in (Liang et al., 2009b)), while keeping the parameters used in the WK and ST models unchanged.

2.6 Computation conditions

Numerical simulations were first performed under control conditions (for a 25 years old healthy adult). Further, we extended the physiological conditions of simulations to aging (i.e., middle age (55 years old) and advanced age (85 years)). Herein, two types of aging conditions were applied, one is characterized by isolated arterial stiffening with age, and the other characterized by arterial stiffening combined with aortic dilatation. In all the simulations, resting conditions were assumed, where the duration of a cardiac cycle was fixed at 0.8s, the blood pressure (P_{puv} in Fig. 1) at the inlet of the heart model was set to be 10.5 mmHg, and the outlets of the WK and ST models were both supported by a constant pressure that represents the precapillary pressure (In Fig. 1, $p_{ou}^{WK} = p_{ou}^{ST} = 30\text{mmHg}$ (Cousins et al., 2014)).

The computer programs of the models were written in FORTRAN language and run on a Dell Workstation (T3600). The CPU time taken by the 1D-ST program to complete a one cardiac cycle simulation was about 72 times longer than that taken by the 1D-WK program (3454s vs 47s). Both programs converged to periodic solutions after running for 15 cardiac cycles.

2.7 Data analysis

The wave separation technique is a classic approach to providing insights into wave interaction phenomena underlying an observed pressure or flow wave (Khir et al., 2005; Liang et al., 2012). In this study, the technique (see *Appendix B* for details) has been employed to identify the contribution of reflected waves originating from distal vasculatures to the observed changes in arterial pressure/flow waves when different outflow boundary models were applied.

To quantify the differences between two pressure or flow waves, the normalized root mean square (*NRMS*) of the pointwise differences was calculated.

$$NRMS = \sqrt{\frac{1}{N} \sum_{i=1}^N \left(\frac{y_1^i - y_2^i}{\bar{y}_1} \right)^2}, \quad (12)$$

where N is the total number of discrete data points computed during a cardiac cycle, y_1^i, y_2^i represent the pressure/flow values (computed by the 1D-WK and 1D-ST models, respectively) at an intermediate time step, $\bar{y}_1$ is the averaged value of y_1^i throughout a cardiac cycle, which is herein used to normalize the difference between y_1^i and y_2^i .

Wave reflection conditions in arteries of interest were quantified by the reflection coefficient (Γ) defined in the frequency domain (Westerhof et al., 1972)

$$\Gamma(f) = \frac{P_b(f)}{P_f(f)}, \quad (13)$$

where f is the frequency, P_b and P_f represent the reflected and incident pressure waves, respectively.

3. Results

3.1 Comparison of the input impedances of isolated WK and ST models

[Fig.3](#) shows the spectra of the input impedances of the WK and ST models connected to the distal end of the l. ant. tibial artery. Marked discrepancies were observed for both modulus and phase angle, indicating that the two models differ significantly in wave reflection properties despite the equivalent resistance, compliance and inductance. [Fig.3](#) also shows the results of sensitivity analysis aimed to investigate the sensitivity of impedance to model parameters. Herein, the effective resistances and compliances of the two models were respectively varied by $\pm 10\%$ relative to their default values. For the ST model, the resistance was varied through modifying the radii of branch vessels while keeping the topology unchanged, and the compliance was varied through modifying the elastic parameters. It was observed that the impedance spectra of the WK model were more sensitive to both resistance and compliance in the low frequency domain ($f < 100\text{Hz}$), in contrast, the impedance of the ST model exhibited stronger dependence on compliance only in the extremely high frequency domain ($f > 500\text{Hz}$).

3.2 Comparisons of simulated aortic input impedances with in vivo data

[Figs.4\(a,b\)](#) show the aortic input impedances simulated by the 1D-WK and 1D-ST models under control conditions compared against the data measured in young subjects ([Murgo et al., 1981](#)). The results obtained by the two models showed considerable discrepancies in some frequency sections, but both were comparable to the in vivo data. [Figs.4\(c-f\)](#) show the changes in aortic input impedance when the total resistances and compliances of the WK and ST models were varied by $\pm 10\%$ relative to their default values. The changes were mild and the sensitivities of impedance to model parameters did not differ significantly between the two models. The attenuated sensitivities of aortic input impedance to terminal resistance and compliance (compared to those presented in [Fig.3](#)) are attributable to the predominant influence of large artery properties on the low-frequency spectra of aortic input impedance

(O'Rourke and Taylor, 1967).

3.3 Comparisons of pressure and flow waves simulated by the 1D-WK and 1D-ST models under control conditions

Fig.5 compares the pressure and flow waves simulated by the 1D-WK and 1D-ST models under control conditions. Here, six observing points (denoted by 'A' to 'F') were selected along the arterial tree. The simulated pressure and flow waves did not differ remarkably at the aortic level, whereas, the discrepancies grew larger from the heart towards the periphery. To better understand the origins of the discrepancies, the pressure waves in four arteries ('A', 'D', 'E' and 'F') were separated into the forward-traveling (incident) and backward-traveling (reflected) components (shown in Figs. 6(a, b)). It was evident that the discrepancies in reflected pressure waves were predominant. From the amplitude ratios of reflected to incident pressure waves and the moduli of wave reflection coefficients (see Figs.6(c, d)), less intensive wave reflections were predicted by the 1D-ST model, especially in the peripheral arteries.

3.4 Comparisons of the 1D-WK and 1D-ST models under aging conditions

The two models were first compared under the aging conditions characterized by isolated arterial stiffening. There was a trend toward increasing discrepancies between the two models due to arterial stiffening with age (see Fig.7(a) for comparisons of aortic pressure waves, and Fig.7(c) for normalized differences of pressure waves in three arteries). When aortic dilatation with age was incorporated, the enlargements of discrepancies with age became insignificant (see Figs.7(b, d)).

4. Discussion

Investigating factors that influence the characteristics of pressure/flow waves simulated by a 1D arterial tree model has become critical in the context that 1D arterial tree models are increasingly being applied not only to study physiological hemodynamic phenomena (Liang et al., 2009b; Johnson et al., 2011; Mynard et al., 2015; Willemet et al., 2015a) but also to assist in the validation or innovation of measurement methods (Trachet et al., 2010; Liang et al., 2013; Papaioannou et al., 2014; Willemet et al., 2015b). The majority of existing studies

1 placed emphasis on investigating factors at the arterial level, including numerical schemes
2 (Sherwin et al., 2003; Huang and Muller, 2015; Müller et al., 2015; Wang et al., 2015),
3 viscoelastic properties of arterial walls (Alastruey et al., 2011) and anatomy of the arterial tree
4 (Blanco et al., 2014; Blanco et al., 2015), while giving less attention to the influence of the
5 method adopted to model the outflow boundary conditions. In the present study, we compared
6 two sorts of outflow boundary models (i.e., WK model and ST model) with respect to their
7 influence on pressure/flow waves simulated at the arterial level. The main findings are
8 summarized as follows: 1) The WK model and ST model differ significantly in wave
9 reflection properties despite equivalent effective resistance, compliance and inductance; 2)
10 pressure/flow waves in peripheral arteries are more prone to the influence of outflow
11 boundary model; and 3) the influence of boundary model on pressure waves does not change
12 remarkably during normal aging but is enhanced by isolated arterial stiffening.

13 The higher sensitivity of pressure waves in peripheral arteries to terminal model is
14 attributable to the shorter distances to outflow boundaries. The different wave reflection
15 properties of the WK and ST boundary models result in marked discrepancies in reflected
16 pressure waves traveling through the peripheral arteries. As the reflected pressure waves
17 propagate towards the aorta, wave reflections at arterial bifurcations and wave dissipation due
18 to wall viscoelasticity will jointly reduce the discrepancies in reflected waves, leading to
19 smaller pressure wave discrepancies observed in the aorta. Interestingly, if the total
20 compliance of the arterial system was reduced as a result of arterial stiffening with age, the
21 discrepancies in pressure waves were enlarged (Figs. 7(a, c)). When the arterial
22 stiffening-induced reduction in arterial compliance was partly compensated by physiological
23 aortic dilatation with age, the enlarged discrepancies were considerably attenuated (Figs. 7(b,
24 d)). These phenomena indicate that arterial compliance plays a considerable role in damping
25 the influence of outflow boundary model.

26 Theoretically, the ST model may allow smoother wave propagation across the distal
27 vascular system owing to a detailed representation of the gradual variations in geometrical
28 and mechanical properties of branch vessels along the vascular system. As is evident from
29 Figs. 6(c, d), the intensities of wave reflections predicted by the 1D-ST model are
30 significantly lower (especially in the low frequency domain). Moreover, the results presented

1 in Fig. 3 indicate that the wave reflection properties of an isolated ST model are better
2 preserved upon mild variations in resistance or compliance. Overall, these results partly prove
3 the advantages of the 1D-ST model, but to confirm the conclusion, a more comprehensive
4 validation work should be done by, for example, comparing pressure/flow waves simulated by
5 a patient-specific model with in vivo measurements (Reymond et al., 2011), or comparing the
6 models with a more detailed model in which the organ/tissue-specific anatomical information
7 of smaller peripheral arteries is incorporated (Blanco et al., 2014, 2015). Despite the
8 limitations, the present study demonstrates the significant impact of the method adopted to
9 model the outflow boundary conditions on the output of a 1D arterial tree model. If the 1D-ST
10 model is superior, the model should be used when hemodynamics in peripheral arteries are
11 concerned. In contrast, for a study focusing solely on aortic hemodynamics, the WK boundary
12 model would be a practical choice in consideration of its low computational cost, unless the
13 arterial system is subject to severe arterial stiffening.

14 It is worthy to note that the method adopted to match the ST and WK models may affect
15 the comparisons between the models. In this study, the scaling factor α has been tuned for
16 each ST model to fit the resistance of the corresponding WK model. There existed other
17 alternative approaches. For example, the root radius of each ST and the length-to-radius ratios
18 of branch vessels have been varied to yield physiological flow divisions among different
19 organs (Steele et al., 2007). On the other hand, the distribution of resistance and compliance
20 in a WK model may be optimized to minimize the discrepancies in pulse waves simulated by
21 the WK model and a higher dimensional model. Efforts in this direction have been reported in
22 (Milišić and Quarteroni, 2004; Epstein et al., 2015). In addition, the WK and ST models were
23 not modified when simulating aging in view of the minor effects of aging on distal arterioles
24 (Moreau et al., 1998; James et al., 2006). However, for studies where hemodynamic
25 regulation is of major interest, the impairment in regulatory function of distal arterioles with
26 age should be carefully addressed. Finally, non-physical wave reflections may be generated at
27 the boundaries and junctions of the arterial tree where vascular viscoelasticity is neglected,
28 although their influence on systemic wave propagation would be minor compared to the
29 dominant role of wave reflections originating from the terminal models. Sophisticated
30 methods for solving the problem have been proposed in a recent study (Müller et al., 2016).

5. Conclusions

In the present study, the Windkessel (WK) model and the structured-tree (ST) model used to account for the outflow boundary conditions of a 1D arterial tree model were compared in terms of their influence on simulated arterial pressure/flow waves. Numerical experiments demonstrated that the wave reflection properties of outflow boundary model significantly affect the characteristics of pressure/flow waves simulated at the arterial level, especially in peripheral arteries, with the effects being further augmented by arterial stiffening. In particular, the ST model generated less intensive low-frequency wave reflections. Moreover, an isolated ST model was found able to better preserve the wave reflection properties upon mild variations in resistance or compliance. Despite the potential advantages, the ST model is computationally more expensive than the WK model. Taken all together, the study suggests that boundary models should be selected based on a comprehensive consideration of computational cost, the properties of the arterial system and the hemodynamic phenomena of concern.

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17 **Appendix A**

18 The numerical scheme starts from estimating the flow rate and extrapolating the
19 forward-traveling Riemann invariant (w_1) at the distal end of the peripheral artery. Blood
20 pressures and flow rates at the distal end of the peripheral artery and the root of the structured
21 tree are then coupled within a numerical time step through an iterative algorithm to reach
22 conservation of mass flux and momentum across the interface. The basic procedure is
23 described as follows.

- 24 a) Estimate the flow rate ($q_m^{1D}|_{m=0}$) and extrapolate w_1 at the distal end of the 1D
25 peripheral artery.
- 26 b) Impose the 1D flow rate (q_m^{1D}) to the root of the structured tree and solve the ST model

- 1 to obtain a new pressure (p_{in}^{ST*}).
- 2 c) Return p_{in}^{ST*} back to the 1D model and resolve flow rate (q_m^{1D*}) using the relationships
- 3 among p_{in}^{ST*} , w_1 and q_m^{1D*} (see Liang et al., 2009a for more details).
- 4 d) Judge convergence by comparing q_m^{1D} and q_m^{1D*} , if converged ($|q_m^{1D*} - q_m^{1D}| < \varepsilon$), go
- 5 to the next time step, if not, update q_m^{1D} using the weighted sum method
- 6 ($q_{m+1}^{1D} = \alpha q_m^{1D} + (1 - \alpha) q_m^{1D*}$, where α is the underrelaxation factor, herein taken to be
- 7 0.5), $m=m+1$, and go to step ‘b’.

8

9 **Appendix B**

10 A pressure wave can be separated into the forward-traveling and backward-traveling
 11 components to quantify the magnitudes of the incident and reflected waves. Wave separation
 12 can be performed either in the time domain or in the frequency domain. Herein, the
 13 time-domain approach was adopted. By definition, a pressure wave consists of three
 14 components

$$15 \quad p(t) = p_0 + p_f(t) + p_b(t), \quad (A1)$$

16 where p_0 is the blood pressure at the beginning of a cardiac cycle ($t = 0$), p_f and p_b represent
 17 respectively the forward-traveling and backward-traveling pressures.

18 p_f and p_b at a transient moment (t) can be obtained by summing up the incremental
 19 changes of the forward-traveling and backward-traveling pressures (Δp_f , Δp_b).

$$20 \quad p_{f,b}(t) = \sum_{\tau=0}^t \Delta p_{f,b}(\tau). \quad (A2)$$

21 Δp_f and Δp_b are calculated based on the changes of blood pressure (Δp) and flow velocity
 22 (Δu) over a short sampling time ($\Delta \tau$),

23

$$24 \quad \Delta p_{f,b} = \frac{1}{2}(\Delta p \pm \rho c \Delta u), \quad (A3)$$

25 where ρ is the density of blood and c the local pulse wave velocity.

Captions

Table 1 Values of τ_ε and τ_σ assigned according to the radius (r) of artery based on experimental data (Valdez-Jasso et al. 2009).

Table 2 Parameters used in the WK and ST models. Notations of parameters: r_{root} , the radius at the distal end of peripheral artery; r_{min} , the terminal radius of the structured tree; α and β , the scaling factors used to generate branch vessels; R , resistance; C , compliance; L , inductance. Superscripts: ST, structured tree model; WK, Windkessel model. Since the effective R , C , and L of each WK model are equal to (with the errors being less than 0.1%) those of the corresponding ST model, only the values of R_1 and R_2 in the WK models are listed.

Fig.1 Schematic description of the arterial tree model (left) and the flowchart of the numerical procedure for coupling the 1D and ST models (right). The entrance of the artery tree is coupled with a lumped model of the left heart, while the distal ends of the tree are coupled with WK or ST boundary models. Notations of parameters used in the heart model: E , elastance; L , inductance; R , viscous resistance; C , compliance; B , Bernoulli's resistance; S , viscoelasticity coefficient of myocardial wall; P_{pc} , pericardium pressure; p_{puv} , pulmonary venous pressure. Subscripts: la, left atrium; lv, left ventricle; mv, mitral valve; av, aortic valve. For notations of parameters used in the flowchart, see the text for details.

Fig.2 Topology of a structured tree. The structured tree originates from the distal end of a peripheral artery (with its radius being the root radius (r_{root}) of the tree) and bifurcates downward according to the scaling factors (α and β). The bifurcating operation will terminate whenever the radius of a branch artery is reduced smaller than the prescribed terminal radius ($r_{\text{min}}=30\mu\text{m}$). The total admittance of the structured tree is obtained by recursively combining the admittances of branch vessels from the distal ends up to the root according to the topology of the tree. The right panel illustrates an example for combining admittances (see the text for details).

Fig.3 Comparison of the input impedances of isolated WK and ST models in terms of impedance modulus and phase angle. Herein, the WK and ST models are connected to the distal end of the l. ant. tibial artery. The thick black lines represent the results calculated under the baseline conditions ($R=R_0$ (default value of effective resistance), $C=C_0$

(default value of effective compliance)), the thin red and blue lines represent the calculated results when R and C are respectively varied by $\pm 10\%$ relative to R_0 and C_0 . Broken points are created between 100Hz and 500Hz and between 600Hz and 900Hz for visualization purpose.

Fig.4 Aortic input impedances (Z_{in}) simulated by the 1D-WK and 1D-ST models: (a, b) comparisons with in vivo data (Murgo et al., 1981); (c-f) changes in Z_{in} as the total terminal resistance (R_T) and compliance (C_T) are respectively varied by $\pm 10\%$ relative to their default values ($R_{T,0}$, $C_{T,0}$). Note that the impedance moduli have been normalized by the characteristic impedance (Z_c) of the aorta to facilitate comparisons between simulations and in vivo data.

Fig.5 Comparisons of pressure and flow waves simulated by the 1D-WK and 1D-ST models under control conditions. Herein, six observing points are selected along the arterial tree: ‘A’, ascending aorta; ‘B’, l. branchial artery; ‘C’, abdominal aorta; ‘D’, l. common carotid artery; ‘E’, r. femoral artery; ‘F’, l. ant. tibial artery.

Fig.6 Forward-traveling (incident) and backward-traveling (reflected) components of pressure waves in arteries ‘A’ (ascending aorta), ‘D’ (l. common carotid artery), ‘E’ (r. femoral artery) and ‘F’ (l. ant. tibial artery) (a, b), amplitude ratios ($A(p_b)/A(p_f)$) of reflected to incident waves (c) and moduli of wave reflection coefficients (Γ) (d).

Fig.7 Comparisons of aortic pressure waves simulated by the 1D-WK and 1D-ST models under different aging conditions: (a) isolated arterial stiffening with age, (b) combined arterial stiffening and aortic dilatation with age, where ‘ p ’ represents the simulated aortic pressure wave, with its forward-traveling and backward-traveling components being denoted by ‘ p_f ’ and ‘ p_b ’, respectively, and ‘ p_0 ’ refers to the blood pressure at the end of diastole. Panels (c) and (d) show the *NRMS* of pressure waves simulated by the 1D-WK and 1D-ST models in the ascending aorta, abdominal aorta and l. ant. tibial artery.

Tables

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Table 1

Radius	$r>10.0\text{mm}$	$9.0\text{mm}<r<10.0\text{mm}$	$8.50\text{mm}<r<9.0\text{mm}$	$6.0\text{mm}<r<8.5\text{mm}$	$r<6.0\text{mm}$
τ_{ε} (s)	0.0883	0.0626	0.0545	0.0734	0.0382
τ_{σ} (s)	0.0576	0.0245	0.018	0.0306	0.009

Table 2

		ST model						WK model		
		r_{root}	r_{min}	α	β	R_{T}^{ST}	C^{ST}	L^{ST}	R_1^{WK}	R_2^{WK}
No. Artery		(cm)	(cm)			(mmHg·s/ml)	(ml/mmHg)	(mmHg·s ² /ml)	(mmHg·s/ml)	(mmHg·s/ml)
6	R.Vertebreal	0.200	0.003	0.84	0.6	34.09	1.04E-3	4.40E-3	6.10	27.99
8	R.Radial	0.140	0.003	0.88	0.6	31.63	2.80E-4	5.94E-3	11.89	19.74
14	Intercoastals	0.300	0.003	0.87	0.6	8.02	5.80E-3	2.74E-3	2.00	6.01
16	L.Vertebreal	0.200	0.003	0.84	0.6	34.09	1.04E-3	4.40E-3	6.10	27.99
19	L.Radial	0.140	0.003	0.89	0.6	31.63	2.80E-4	5.94E-3	11.01	20.63
22	Hepatic	0.250	0.003	0.84	0.6	20.39	2.55E-3	3.48E-3	2.80	17.59
23	Splenic	0.150	0.003	0.88	0.6	31.77	3.63E-4	5.62E-3	8.59	23.18
24	Gastric	0.200	0.003	0.88	0.6	13.64	1.33E-3	4.06E-3	4.05	9.59
26	Sup.mensenteric	0.350	0.003	0.87	0.6	5.36	1.01E-2	2.34E-3	1.20	4.17
28	R.renal	0.275	0.003	0.88	0.6	6.63	4.65E-3	2.93E-3	2.02	4.60
30	L.renal	0.275	0.003	0.88	0.6	6.63	4.65E-3	2.93E-3	2.02	4.60
32	Inf.mensenteric	0.200	0.003	0.84	0.6	38.37	1.02E-3	4.43E-3	3.83	34.54
36	R.int.iliac	0.200	0.003	0.85	0.6	31.09	1.06E-3	4.37E-3	4.95	26.13
37	R.deep femoral	0.200	0.003	0.87	0.6	19.24	1.21E-3	4.18E-3	4.99	14.25
39	R.ext.carotid	0.200	0.003	0.83	0.6	42.01	1.00E-3	4.49E-3	5.23	36.78
40	L.int.carotid	0.300	0.003	0.81	0.6	27.76	4.37E-3	3.08E-3	2.10	25.66
41	R.post.tibial	0.175	0.003	0.85	0.6	41.66	6.09E-4	5.02E-3	7.67	33.99
42	R.ant.tibial	0.250	0.003	0.85	0.6	18.90	2.65E-3	3.44E-3	3.32	15.59
43	R.interosseous	0.100	0.003	0.90	0.6	53.07	7.21E-5	8.31E-3	26.45	26.62
44	R.ulnar II	0.180	0.003	0.86	0.6	30.64	7.28E-4	4.77E-3	7.15	23.49
45	L.ulnar II	0.180	0.003	0.86	0.6	30.64	7.28E-4	4.77E-3	7.15	23.49
46	L.interosseous	0.100	0.003	0.90	0.6	53.07	7.21E-5	8.31E-3	26.45	26.62
47	R.int.cartiod	0.350	0.003	0.80	0.6	27.82	7.09E-3	2.72E-3	1.53	26.29
48	L.ext.carotid	0.200	0.003	0.83	0.6	42.01	1.00E-3	4.49E-3	5.23	36.78
51	L.int.iliac	0.200	0.003	0.85	0.6	31.09	1.06E-3	4.37E-3	4.95	26.13
52	L.deep femoral	0.200	0.003	0.87	0.6	19.24	1.21E-3	4.18E-3	4.99	14.25
54	L.post.tibial	0.175	0.003	0.85	0.6	41.66	6.09E-4	5.02E-3	7.67	33.99
55	L.ant.tibial	0.250	0.003	0.85	0.6	19.15	2.64E-3	3.45E-3	3.96	15.18

Figures

Fig.1

Fig. 2

Fig.3

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