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A hemodynamics model to study the collective behavior of the ventricular-arterial system

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Applying principles from complex systems to study the efficacy of integrative therapies has become a new interest in medical research. We aimed to construct a concise model for the ventricular-arterial (VA) system and to provide a systematic method for exploring its overall behavior. The transportation of blood from the heart to the peripheral arterioles via hydraulic pressure forces was described by a multi-rank model. Parts of the VA system that have strong mutual interactions were combined into a single sub system. Sub systems of four different ranks were characterized. We then applied the multi-rank model to analyze the aortic pressure wave generated by the periodic ventricular blood ejection, the renal pressure in response to the input from the VA system, and the blood flowing from the renal artery to its arterioles. Maintaining the pressure distribution along the main arteries and in all of the organs with the lowest possible ventricular input turned out to be the first principle for the operation of an efficient VA system. By this principle, we pointed out the benefit of some arterial structures in mammals, derived specific regulation rules and deduced some fundamental concepts for healing. The justification of the biomechanics in our model that differed greatly from those in the prevailing models was given. We concluded that the oscillatory motion and the pressure pulse of the arterial system can be analyzed as steady states with resonance behaviors and suggested utilizing this model to construct integrative therapies for diseases correlated with abnormality in blood circulation.
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I. INTRODUCTION

Recently, applying principles from complex systems to study the efficacy of integrative therapies becomes a new interest in medical research.[1] Understanding the collective behavior of the ventricular-arterial (VA) system will facilitate the development of integrated methods of diagnosis and treatment for cardiovascular diseases.

There are some basic problems still unsolved in the study of hemodynamics. The arterial resonance, referred to Otto[2] and his followers, was criticized by McDonald[3] as being physically impossible. The oscillatory motion of the arterial system has historically been depicted either as a steady state or as a transient phenomenon with traveling-wave behavior, and the associated debate about whether the pulse wave should be analyzed in the time or frequency domain is ongoing.[4] The present study aimed not only to answer long-existing questions about basic hemodynamics but also to provide a systematic method for exploring the overall behavior of the arterial system.

Stacy and Giles[5] related the input force to the peripheral pressure of an organ by modeling the organ as an oscillatory system with inertia and elasticity. The experiments on rats showed that some organs had resonant behavior[6,7] and the increase of pulsatile blood pressure enlarged the perfusion in the peripheral vascular beds.[8] We proposed that the arterial system has its natural frequency and suggested a matching rule in terms of frequencies.[9] We derived a transverse pressure-area wave equation;[10] and further analyzed that the radial oscillatory motion of the artery is the major wave mode in large arteries.[11] The wave equation was then solved in terms of the eigen functions of the arterial system.[12]

Multi-scale modeling was recently used to elucidate the structures and functions of living systems.[13,14] However, living systems operate in an integrated manner, with systems of higher ranks acting collectively on those of lower ranks.

In this study, based on the above findings, we proposed replacing the multi-scale modeling with the multi-rank (MR) modeling for the VA system and described the mechanism for blood transported from the left ventricle to the peripheral arterioles.

The interaction of the VA system with a kidney organ was analyzed to demonstrate how to utilize the MR modeling to study the collective behavior of the VA system.

The advantages of the MR modeling over other methods were revealed by comparing the major differences between them.

In recent years, researchers reported various models for studying the flow patterns in the left ventricle.[15] How to incorporate their results to this MR model was discussed.

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II. METHOD

A. Description of the multi-rank modeling for the VA system

The transportation of blood from the heart to the peripheral arterioles via hydraulic pressure forces can be described by a multi-rank model. We decomposed the entire VA system into several sub systems, parts that have strong mutual interactions are combined into a single system by examining the order of the various interacting forces. The operating mechanisms for sub systems of different ranks, (Figure 1), were described and analyzed below.

1. The left ventricle is a hydraulic pressure supplier of the first rank

The ejection of blood from the left ventricle into the arterial system greatly reduces its momentum, and this generates an impulsive force that is exerted on the arterial system. By Pascal's principle, the force is transmitted through the blood and acts on the elastic wall of the aorta, manifesting as an impulsive hydraulic pressure force in the radial outward direction. Since the ventricular output is periodic, it can be represented by harmonic forces with frequencies that are integer multiples of the heart rate; these sustain the pulsatile motion of the arterial system.

2. The main arterial system comprising all of the large arteries is a hydraulic pressure supplier of the second rank

The interaction between the blood and the arterial wall through the blood pressure force is high. Here, we treat the aorta comprising all of the large arteries as a single system. The pulsatile input force from the left ventricle induces a distributed pulsatile pressure along the system, and the entire aorta performs a forced distributed oscillatory motion that can be expressed in terms of the eigen wave mode solution[12] of the pressure-area wave equation[10] after reaching the steady state.

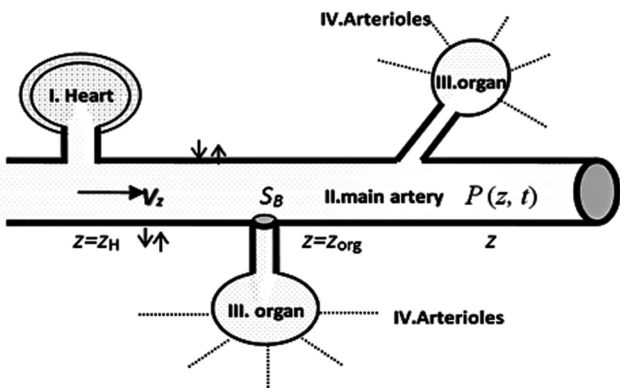

FIG. 1. Sketch of the VA system in the framework of a multi-rank hydraulic-pressure-supplier model: with the heart being the pressure supplier of the first rank, the main artery of the second rank, each organ of the third rank; and the peripheral arterioles as receivers of the hydraulic pressure forces. The axial velocity of the blood is V_Z. The arterial wall is elastic and executes a distributed pulsatile radial movement. $P(z, t)$ is the induced pressure at axial position z, and S_B is the cross-sectional area of the branch opening.

The main aorta system acts as a pressure and blood supplier of the second rank. The hydraulic pressure forces F_B exerted on the arterial branch unit connected at axial position $z = z_B$ along the aorta (Figure 1) is given by the product of the cross-sectional area S_B of the opening and the local pressure as

$$F_B = S_B P(z,t)|_{z=z_B}. \quad (1)$$

3. Every arterial branch unit is a hydraulic pressure supplier of the third rank

Each arterial branch unit attached to the aorta, such as artery of organs, artery of limbs, or artery of head, is treated as a single system. Every arterial branch unit also provides the pressure gradient force needed to deliver blood to its peripheral arterioles and plays the role of a third-rank pressure and blood supplier.

Since the dimensions of organs are much smaller than the primary wavelengths of the pulse pressure, each of them can be lumped as a resonator with a specific inertia, compliance, and resistance.[5] Each organ has its own natural frequency. The behavior of each organ or its pulsatile pressure can, therefore, be described as a damped forced oscillation in response to the pulsatile pressure force provided by the VA system.

4. Peripheral arterioles connected to the arterial branch units are receivers of the hydraulic pressure forces

Peripheral arterioles have high resistance and low distensibility. They are the primary sites for power consumption and the blood flow therein is driven mainly by the pressure gradient.

B. Utilizing the MR modeling to study the kidney system

In this section, we analyzed the collective interaction of the VA system with a kidney organ to demonstrate how the MR modeling maybe used to study the collective behavior of the VA system.

1. The force generated by the left ventricle

Assuming the left ventricle at axial position $z = z_H$ starts from $t = 0$ to eject blood periodically with heart rate f_H, this produces a force $F_H(t, z)$ that is exerted on the arterial system. The periodic force $F_H(t, z)$ can be decomposed into various harmonic components as

$$F_H(t,z) = \Gamma(t)\delta(z - z_H)\sum_{m=0}^{N} F_{Hm}\sin(\omega_{Hm}t + \phi_{Hm}). \quad (2)$$

Here, $\Gamma(t)$ is the unit step function.[16] The angular frequency $\omega_{Hm} = 2\pi m f_H$, the phase angle ϕ_{Hm}, and the amplitude of the m-th harmonic component F_{Hm}, are determined by the condition of the ventricular blood ejection such as the ejecting duration and the magnitude of the blood flux.

2. The pressure wave in the main arterial system

$$\frac{\partial^2 P}{\partial t^2} + 2b\frac{\partial P}{\partial t} = C^2\frac{\partial^2 P}{\partial z^2} + \frac{E_P}{R_0 M_L}F_H. \quad (3)$$

We use Eq. (3) as the pressure wave equation for the large arteries. It has been derived previously[10] from the radial momentum equation of the combined blood-wall system by assuming that the thin-walled artery is of rotational symmetry, the arterial vessel has thickness h_w and density ρ_w, and an adherent thin layer of blood with equivalent thickness h_b and density ρ_b is moving transversely together with the vessel. Here, P is the responded arterial pressure, b is the damping constant for transverse motion, C is the wave velocity correlated with the longitudinal tension, $E_P = R(\partial P/\partial R)$ is the Peterson's elastic modulus, R and R_0 are the inner radius and the static inner radius of the artery, respectively, and $M_L = 2\pi R(\rho_w h_w + \rho_b h_b)$.

The steady state solution of the Eq. (3) for a harmonic force of input angular frequency ω given by $F_\omega(t,z) = F_{0\omega}\sin\omega t\delta(z - z_H)$ and with the initial conditions for the response pressure to be $P_\omega(z,t)|_{t=0} = 0$ and $\frac{\partial P_\omega(z,t)}{\partial t}|_{t=0} = 0$, has been derived in previous study[12] as

$$P_\omega(z,t) = A_\omega\sum_{n=0}^{\infty}\varphi_n(z_H)\varphi_n(z)C_n\sin(\omega t + \phi_n).$$

Now let us consider the response pressure for the m-th harmonic component of the periodic force generated by the heart $F_{\omega_{Hm}}(t,z) = \Gamma(t)F_{0\omega_{Hm}}(\omega_{Hm}t + \phi_{Hm})\delta(z - z_H)$ with input angular frequency ω_{Hm}. Since the left ventricle starts to eject the blood at time $t = 0$, the induced pressure can reach the artery at position z only after a period of time $t_0(z) = |z - z_H|/C$, hence we modify the initial conditions for the pressure wave to

$$P_\omega(z,t)\Big|_{t=t_0(z)} = 0 \quad \text{and} \quad \frac{\partial P_\omega(z,t)}{\partial t}\Big|_{t=t_0(z)} = 0. \quad (4)$$

By defining a new time variable $\tilde{t} = t - t_0(z)$ and through a time shift Laplace transform,[16] the response pressure becomes as

$$\begin{aligned} P_{\omega_{Hm}}(z,t) &= A_m\sum_{n=0}^{\infty}\varphi_n(z_H)\varphi_n(z)C_{mn}\sin(\omega_{Hm}\tilde{t} + \phi_{Hm} + \phi_{mn}) \\ &= A_m\sum_{n=0}^{\infty}\varphi_n(z_H)\varphi_n(z)C_{mn} \\ &\quad\times\sin[\omega_{Hm}t + \phi_{Hm} + \phi_{mn} - \phi_{\omega_{Hm}}(z)]. \end{aligned} \quad (5)$$

Each $\varphi_n(z)$ satisfies the eigen value equation

$$\frac{d^2\phi_n(z)}{dz^2} + k_n^2\varphi_n(z) = 0 \quad n = 1,2,3,\ldots \quad (6)$$

where the eigen wave numbers k_n can be determined by the boundary conditions of the aortic terminal ends. The natural angular frequencies ω_n of the main aortic system are related to the eigen wave numbers k_n and the wave velocity C according to $\omega_n = k_n C$.

In Eq. (5),

$$C_{mn} = [(\omega_{Hm}^2 - \omega_n^2)^2 + 4b^2\omega_{Hm}^2]^{-1/2}, \quad (7)$$

$$\phi_{mn} = \tan^{-1}[2b\omega/(\omega_{Hm}^2 - \omega_n^2)], \quad (8)$$

and

$$\phi_{\omega_{Hm}}(z) = \omega_{Hm}t_0(z) = \omega_{Hm}|z - z_H|/C. \quad (9)$$

Besides the ordinary phase angle ϕ_{mn}, Eq. (9) shows that there is an additional phase angle $\phi_{\omega_{Hm}}(z)$ which is position dependent due to the finite value of the phase velocity. After the aortic system reaches the steady state, the phase difference at two sites along the main artery depends on the input angular frequency ω_{Hm}, the axial distance between them, and the phase velocity C.

By summing all the harmonic components of the force generated from the heart, the pressure distribution along the artery can be written as

$$\begin{aligned} P(z,t) = \sum_{m=0}^{N}A_m\sum_{n=0}^{\infty}\varphi_n(z_H)\varphi_n(z)C_{mn} \\ \times\sin[\omega_{Hm}t + \phi_{Hm} + \phi_{mn} - \phi_{\omega_{Hm}}(z)]. \end{aligned} \quad (10)$$

3. Determination of the natural frequencies of the main arterial system

We assume the main arterial system, or the aorta, extends from $z = 0$ to $z = L$ and focus on the eigen modes which have zero gradient at both boundaries

$$\frac{d\varphi_n(z)}{dz}\Big|_{Z=0} = 0, \quad \frac{d\varphi_n(z)}{dz}\Big|_{Z=L} = 0. \quad (11)$$

The boundary conditions yield the eigen functions as follows:

$$\begin{aligned} \varphi_n(z) &= (1/L)^{1/2} \quad \text{for } n = 0 \\ \varphi_n(z) &= (2/L)^{1/2}\cos(2\pi nz/\lambda_1) \quad \text{for } n = 1,2,3\ldots \end{aligned} \quad (12)$$

Here, $\lambda_n = L/(2n)$ and the corresponding eigen frequencies are given by

$$f_n = \omega_n/(2\pi) = nC/(2L) = nf_1. \quad (13)$$

From Eq. (7), for the eigen angular frequency ω_n near the input angular frequency ω_{Hm}, the pressure amplitude factor C_{mn} of that eigen mode is maximized. Since $\omega_{Hm} = 2\pi mf_H$ and $\omega_n = 2\pi nf_1$, to ensure highest pressure response, one possible choice of the heart rate will be

$$f_H = f_1 = C/(2L). \quad (14)$$

In this circumstance, the frequency spectrum of the force generated by the heart coincides with the natural

frequency spectrum of the main aorta system to satisfy the optimal resonance conditions between them.

There are some logic reasons to choose the eigen modes which fulfill the boundary conditions in Eq. (11). First, it ensures that at the arterial junctions to the head or to the upper extremities (at $z = 0$) or to the lower extremities (at $z = L$), are the anti-nodes for all the induced eigen modes, thus the main arterial system may provide maximum periodic hydraulic forces (Eq. (1)) to all of these branch openings. Since the heart is near one end of the aorta, $z_H \approx 0$, the factor $\varphi_n(z_H)$ in Eq. (10) will also have the optimal values for all n. Besides, zero gradient of the eigen modes of the pressure will induce zero axial acceleration of the blood and therefore reduces the axial oscillatory motion of the blood at the junction sites. Furthermore, Eq. (14) shows that the fundamental natural frequency of the main arterial system f_1 and heart rate f_H is inversely proportional to its aortic length L. It is consistent with the finding that there is no significant difference in the resting heart rate of people with traumatic amputations and of those with all limbs.[17]

Equation (14) can also be used to explain the physiological finding that the heart rate of mammals is inversely proportional to the mammal's size.[18]

4. Response pressure of the kidney organ

The Stacy and Giles's equation[5] for the pressure P_{org} of an organ in response to an input harmonic force of angular frequency ω can be written as

$$a' \frac{\partial^2 P_{org}}{\partial t^2} + b' \frac{\partial P_{org}}{\partial t} + c' P_{org} = F_{i0} \sin(\omega t).$$

We may define $\omega_{org} = \sqrt{c'/a'}$ as the natural angular frequency of the organ, then Stacy and Giles's equation becomes

$$\frac{\partial^2 P_{org}}{\partial t^2} + b \frac{\partial P_{org}}{\partial t} + {\omega_{org}}^2 P_{org} = \frac{1}{a'} F_{i0} \sin(\omega t).$$

This is a typical equation for forced oscillation; if the input frequency ω is near the natural frequency of the organ ω_{org}, a maximum amplitude for the response harmonic pressure P_{org} will occur.[16]

The hydraulic pressure force acting from the VA system on an organ attached at axial position $z = z_{org}$ through a branch opening with cross sectional area S_B (Figure 1) is $F_i = S_B P(z,t)|_{z=z_{org}}$. Thus, the Stacy and Giles's equation for the pressure of the renal artery P_{renal} can be written as

$$\frac{\partial^2 P_{renal}}{\partial t^2} + b \frac{\partial P_{renal}}{\partial t} + \omega_{kidney}^2 P_{renal} = \frac{1}{a'} S_B P(z,t)|_{z=z_{kidney}}. \quad (15)$$

Equation (12) shows that for the n-th eigenfunction $\varphi_n(z)$, the position $z = L/2$ is an anti-node with n even, and a node with n odd. Since the kidney is connected more near the midpoint of the main arterial system, $z_{kidney} \cong L/2$, the hydraulic pressure force (Eqs. (10) and (15)) acting on the renal artery from the VA system will be comprised dominantly of harmonic forces which have frequencies to be even multiples of the heart rate f_H. If the kidney has a fundamental natural frequency $f_{kidney} \cong 2f_H$, a maximum pressure response P_{renal} will occur, and thus enhances the amount of blood flowing from the renal artery to its arterioles.

III. RESULTS

In this MR model, maintaining the pressure distribution along the main arteries and in all of the organs with the lowest possible ventricular input is the first principle for the operation of an efficient VA system. This principle can be used to explain many physiological findings, deduce certain regulation rules and give fundamental concepts for healing; we summarize a few of them below.

A. Lowering the axial flow velocity in the main arteries is beneficial

To reduce the energy dissipation, the blood—being the mediator for the pressure—should have its axial flow velocity as small as possible in the main arteries. This rule reveals the physiological purpose of some of the anatomic structures of the mammalian arterial system.[19]

The 180° bend encountered by blood ejected from the left ventricle near the arch of the aorta results in a substantial mass of blood impinging on the elastic wall of the vessel, which greatly reduces its axial kinetic energy due to this collision being inelastic.

The ends of the ulnar artery and the radial artery join together as a loop at the palmar arch.[19] Similarly, the deep plantar artery unites with the termination of the lateral plantar artery to complete the plantar arch.[19] The head-on collision of blood flows approaching from opposite directions at these arches eliminates their axial momentum and helps to distribute blood more uniformly to all of the perpendicularly attached side branches. If the loop end were replaced by two unconnected open ends, most of the flow would go through those openings, which would greatly reduce the flow to all of the other perpendicular branches. If the loop were replaced by two closed ends, those ends would become reflection sites to stop the axial flow and thereby cause long-term damage.

B. The resonance model of the main arterial system is feasible

Skalak *et al.*[20] and Milnor[18] reported that most of the energy supplied by the left ventricle is dissipated in the friction of viscous flow and that almost all of the work done in distending the arteries is returned later in each cycle of the heart beat due to the relatively small viscosity of the vascular wall. Since the ventricular power input is mainly delivered via the radial pulsatile motion, there is little attenuation of the pressure wave. This implies that the high dissipation of the pulse wave as described by McDonald[3] does not actually occur in large arteries, we may thus conclude that the arterial resonance observed by Otto[2] and his followers is plausible. Therefore, the steady-state solution (Eq. (10)) of the pressure wave equation can be obtained based on the eigen wave

modes of the entire aortic system characterized by its natural frequencies.

C. The correct longitudinal tension is necessary

The reported[21,22] average relative stretching of the arterial length in vivo has ranged from 21% to 42%. The large longitudinal tension provides the tautness needed to support the propagation of a transverse wave—this is analogous to pulling an elastic string tightly so that it can support transverse vibrations. It is, therefore, beneficial to the blood circulation to adopt postures that maintain the correct longitudinal stretching in all parts of the large arteries.

D. Pulsatile input from the heart is essential for the operation of a low-dissipation system

If the heart were replaced by a continuous pump, once the arteries reached their equilibrium cross-sectional areas there would be no further pulsatile motion of the wall, and thus the benefit of low dissipation in power transmission associated with pulsatile motion of the arterial wall would be lost. A pulsatile input from the heart is not only essential for optimizing the energy efficiency but also has the advantage to adjust rapidly when facing different operating situations. The frequency matching between the heart rate and the natural frequency of the main arterial system (Eq. (7)) should be considered when studying changes in the heart rate during exercise and in the design of devices aimed for assisting the function of heart.

E. Resonance of an organ with the VA system

Each organ has its own natural frequency or frequencies. The presence of matching between the frequency spectrum of the local pulsatile pressure and the natural frequency spectrum of an organ may also produce resonance behaviors (Eq. (15)). This implies that adjusting the frequency-matching conditions could be an important concept for the treatment of cardiovascular diseases.

IV. DISCUSSION

The biomechanics in this MR model that differed greatly from those in the prevailing models was described below.

First, the pressure, a more stable parameter, is taken as the sole first-order parameter for the arterial waves in the MR model. The flow, being a vector quantity and more likely to vary with the posture change of the body, is considered to be a minor parameter. In main arteries, the attenuation of the axial flow wave is high;[18] the axial velocity of the blood is less than 0.6 m/s, and the blood pressure variation is about 40 mmHg or $5 \times 10^3\ \mathrm{N/m^2}$. The density of blood can be taken as $\rho = 1.06 \times 10^3\ \mathrm{kg/m^3}$. For these values, the energy density associated with the axial blood flow is less than 5% of that associated with the pressure wave. Furthermore, most side branches are connected perpendicularly with the main arteries.[19] These facts support that MR model is more realistic than the models that took the axial flow as the major focus and defined the vascular impedance in terms of pressure gradient and flow waves in arteries.[23]

Second, many models for circulatory system[14,24] considered the output of the blood from the left ventricle as a proximal boundary condition. We considered it as a blood and hydraulic pressure force supplier; the MR modeling provides a starting point to investigate how the heart regulates the blood circulation by changing the profile of blood outputs.

Third, in the transmission line model or the multi-scale modeling, the main arterial tree (including the large arteries and the attached side-branch organs) is typically decomposed into many arterial tree units, and both the flow and the pressure parameters need to fit certain basic rules of fluid mechanics[14,24] at all the junction sites between two adjacent arterial tree units. Actually, the main large arteries are connected by a strong longitudinal tension,[21,22] and each side branch is connected to the main arteries by a stiffer vessel with a much smaller radius.[19] Hence, to a first-order approximation we treated the main arteries together as a single system and each attached organ as a separated system, which we considered to better reflect known physiological structures and physical mechanisms.

Fourth, Euler-based theories of hemodynamics have often been expressed in the form of axial flow Q through blood vessels by taking the vessel walls as the boundary of the enclosed blood.[1] Most of the basic wave equations (PQ wave) were related to the hydraulic Navier-Stokes equation in the longitudinal direction. Observing the phase difference of the pulse wave between two different sites of the arterial system, many researchers utilized the high-dissipation PQ wave mode, treated the pulse wave as a travelling wave with transient phenomenon and used the phase lag to calculate the pressure wave velocity. Here, we treated the blood and the wall together as a single system and the pressure wave distribution in the large arterial system is governed by a transverse pressure wave equation as in Eq. (3). Due to the low-dissipation character of the radial oscillation[18,20] for the transverse wave mode, we deduced that a continuous periodic ventricular input will result in the large arteries reaching a steady state and undergoing collective distributed radial damped forced oscillations. Equation (9) shows that the phase lag still exists even after the aortic system reached a steady state. In resting conditions, the arterial systems are in steady states; thus, there is no longer a need for the heart to initiate the distributed oscillatory motion or the associated pressure wave. Instead, during each beat the heart needs only to refill the amount of blood flowing out from the aorta system to the branches; and only the amount of power dissipated during the delivery process needs to be supplied. This MR modeling can explain why only a few watts of input power are required from the left ventricle in humans.[18]

Fifth, in this model, each organ artery also undergoes a radial damped forced oscillation in response to the input pressure force from its nearby supplying aorta. It provides a basis to diagnose or treat a specific organ disease by studying the natural frequency of the organ, the correlated harmonic component of the arterial pressure, and the matching conditions between the harmonic frequency and the natural frequency of the organ.

Finally, the steady periodic pressure wave resulting from the overall behavior of the coupled VA system constitutes a major characteristic parameter. Moreover, analysis of

the pressure wave in the frequency domain[25] may represent one useful method for examining the integrated conditions of the circulatory system.

In this study, we applied the MR model to analyze the aortic pressure wave generated by the periodic blood ejection from the heart, the renal pressure caused by the input from the VA system to a kidney organ, and the blood flowing into its arterioles. The quantitative results provided the biomechanical basis to explain the animal experiments in literature such that the heart rate of mammals is inversely proportional to the mammal's size,[18] the abdominal aortic blood pressure determined the renal cortex flux,[8] and the conditions of the kidney were associated with the second harmonic component of the arterial pressure wave.[6,7,26]

We suggest utilizing this model to analyze the collective effect of the VA system on other organs and to construct integrative therapies for diseases correlated with abnormality in blood circulation.

The periodic force source to the arterial system is generated by the momentum change of the ventricular blood output as it encounters the arch of the aorta. Recently, flow patterns in the left ventricle have been greatly studied.[15,27] Extending the studies to investigate how the flow pattern correlates with the harmonic components of the force $F_H(t,z)$ (Eq. (2)), may make the mechanism of the MR modeling more complete.

In formulating the MR model of the VA system, we considered (to a first-order approximation) that systems with higher ranks provide the blood source and the hydraulic pressure force to the systems with lower ranks. Feedback from the lower-rank to the higher-rank systems may be taken into account as secondary effects. For example, as the main aorta gives a force on an attached organ, there is also a reaction force acting on the main aorta by the organ, and it can be counted by adding a secondary force source term into the right hand side of Eq. (3) for the pressure equation of the aorta. However, further physical and physiological studies are needed to fully assess the applicability of these approaches.

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