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## The ventricular-arterial coupling system can be analyzed by the eigenwave modes of the whole arterial system

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In response to harmonic forces generated by the heart, the arterial system executes strong coupled distributed oscillatory motions. These oscillations are described by a pressure-area wave equation, which is solvable subject to appropriate Sturm–Liouville boundary conditions. The response pressure can be represented as a sum of stationary waves which are the eigenmodes of the whole arterial system. Natural frequencies of the system are related to the eigenvalues and the phase velocity. Matching of these natural frequencies with heart rate or its harmonics is important in ventricular-arterial coupling. Transfer functions for the pressure can be constructed from the corresponding eigenfunctions. © 2008 American Institute of Physics. [DOI: 10.1063/1.2911746]

Arterial pulses are fundamental signs in clinical medicine. A proper solution of the pulse wave distribution in the ventricular-arterial coupling system is one of the most important and unsolved physical problem in hemodynamics. Most of the earlier researchers in pulse analysis of arterial systems adopted either the Windkessel model or the transmission line theory.<sup>1,2</sup> Efforts were later made to show that various models initiated the "natural frequency" and "standing waves" of the blood vessels by analogy with the behavior of manometers.<sup>3,4</sup> McDonald<sup>5</sup> criticized the concept as being physically impossible, arguing that the reflected waves are considerably damped during their travel between the reflection sites. Since then, many of the pulse wave analysis for arterial system treat measured pressure and flow waves as the sum of a single forward wave and a single backward wave.

Berger *et al.*<sup>6</sup> developed a more general wave reflection theory that allows repeated reflections of these waves. Khir and Parker<sup>7</sup> demonstrated *in vitro* that reflected waves can be rereflected. Nichols and O'Rourke<sup>2</sup> pointed out that it is impossible to explain the data on pressure waves without admitting wave reflection and conceding that a type of damped resonance does indeed occur.

Currently, most pulse wave analysis on the ventriculararterial coupling system is based on the concept that pressure gradient forces drive the axial flow Q, with the Navier– Stokes equation as the major governing equation.<sup>1</sup> These models took forces arising from compliant walls or any other nonlinear effects as perturbations.<sup>8–10</sup> We called them the PQ wave models.<sup>11</sup> We have pointed out that PQ wave models are not appropriate in describing systems of high pressure and low elastic modulus,<sup>11</sup> which is precisely the situation in most large arteries. We have also analyzed that the major wave modes in large arteries are the variation of the crosssectional area S of the artery driven by the pulsatile pressure force,<sup>11</sup> or the PS wave mode. Hence, the pressure-area wave equation<sup>12</sup> plays the role of the master equation for pulse waves. Energy dissipations during wave propagation in the PS wave model are much lower than in the prevailing PQ wave model, thus, making multireflections in large arteries plausible.<sup>11,13</sup>

In this letter, we will use Bernoulli's oscillatory method<sup>14</sup> to analyze pulse waves in the ventricular-arterial coupling system by solving the pressure-area wave equation. We will demonstrate that the response pressure is characterized by the natural frequencies of the whole arterial system. We will also derive the rule of "frequency matching"<sup>13</sup> in the coupled ventricular-arterial system by analyzing the amplitudes of the pressure eigenmodes.

Let us consider a blood vessel with cylindrical shape, and take the axis of symmetry as the z axis and the inner radius as r(z,t). In a previous study,<sup>12</sup> we have derived that the cross-sectional area  $S(z,t) = \pi r^2$  obeys the following wave equation:

$$\mu \frac{\partial^2 S}{\partial t^2} + R \frac{\partial S}{\partial t} + 2\pi \left[ \mathbf{E}_{\theta} h_w \frac{\Delta r}{r_0} - r(P_I - P_0) \right] = \tau \frac{\partial^2 S}{\partial z^2} + F_e.$$
(1)

Here we assume that the elastic vessel is of thickness  $h_w$ and density  $\rho_w$ , and there is a thin layer of blood with thickness  $h_b$  and density  $\rho_b$  transversely moving with the vessel. Other parameters appearing in Eq. (1) are  $\mu = \rho_w h_w + \rho_b h_b$ , resistance constant for transverse motion R, local internal pressure  $P_I(z,t)$ , local external pressure  $P_0(z)$ , and the inner radius of the artery  $r_0$  when  $P_I = P_0$ . Furthermore, we define  $\Delta r = r - r_0$  and  $\tau = E_{rz} h_w + T_w$ . Here,  $T_w$  is the longitudinal tension per unit circumferential length of the elastic vessel;  $E_{rz}$ is the shearing modulus of elasticity of the vessel. Also appearing is the external force per unit axial length  $F_e$  acting on the arterial system, and Young's modulus of elasticity  $E_\theta$  of the vessel in the circumferential direction.

We call Eq. (1) the transverse PS wave equation. The blood acts more of a mediator to pass the pressure than a medium to transmit the ventricular energy output.

The local internal pressure starts from an initial value  $P_S(z)$  at t=0. We define the response pressure  $P(z,t) = P_I(z,t) - P_S(z)$  and assume  $P_S(z)$  has vanishing second derivative with respect to the axial position z. We also define

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area compliance as  $C_A = dS/dP_I$ . Then, Eq. (1) becomes

$$\mu \frac{\partial^2 P}{\partial t^2} + R \frac{\partial P}{\partial t} + \kappa = \tau \frac{\partial^2 P}{\partial z^2} + \frac{1}{C_A} F_e.$$
(2)

Here  $\kappa = 2\pi [(E_{\theta}h_w\Delta r/r) - r(P_I - P_0)]/C_A$ . Following Laplace's theory for thin-walled tubes, <sup>15</sup>  $E_{\theta}$  can be expressed as  $E_{\theta} = r_0^2 (P_I - P_0)/(h_w\Delta r)$ . Thus, as a first order of approximation, we take  $\kappa \approx 0$ , and Eq. (2) can be written as

$$\frac{\partial^2 P}{\partial t^2} + 2b\frac{\partial P}{\partial t} = C^2 \frac{\partial^2 P}{\partial z^2} + \frac{1}{\mu C_A} F_e,$$
(3)

with  $b=R/(2\mu)$ , and  $C=(\tau/\mu)^{1/2}$ . This expression for the wave velocity *C* plays the role in the PS model as the well-known Moens–Korteweg formula<sup>16</sup> in the PQ models.

The only driving force for the arterial system comes from the ventricular blood ejection. We assume that the position of the heart is at  $z=z_H$  and the ejection starts from t=0. The external force per unit length can be written as  $F_e(z,t)=F(t)\delta(z-z_H)$ .

In what follows, two different kinds of force will be considered. One is an impulsive force with  $F(t)=F_I=I_0\delta(t)$  since the impulse response method is often used to experimentally find out the natural frequencies of a system. The other is a harmonic force of input frequency  $\omega$  given by  $F(t)=F_{\omega}=F_{0\omega}\sin \omega t$ .

In this study, we only assume that general boundary conditions for the Sturm–Liouville problem<sup>17</sup> are satisfied. We will solve the PS wave equation by assuming that the area compliance, the resistance, and the wave velocity are constant along the artery.

As the external force is turned on at time t=0, the pressure starts to deviate from its initial value  $P_S(z)$ . Hence, the initial conditions for the response pressure can be written as P(z,0)=0, and  $\partial P(z,t)/\partial t|_{t=0}=0$ .

We first solve the initial value problems by Laplace transformation method. Taking the initial conditions into account, the transformed Eq. (3) can be written as

$$\frac{\partial^2 Y(z,s)}{\partial z^2} + \lambda_S Y(z,s) = -H(s)\,\delta(z-z_H)\,. \tag{4}$$

Here,  $\lambda_s = -(s^2 + 2bs)/C^2$ , Y(z,s) is the Laplace transform of P(z,t), and H(s) is the Laplace transform of  $F(t)/(C^2 \mu C_A)$ .

By the method of Green's functions,<sup>17</sup> the solution of Eq. (4) can be expressed in terms of the normalized eigenfunctions  $\varphi_n(z)$  of free oscillation of the arterial system as

$$Y(z,s) = \sum_{n=1}^{\infty} \varphi_n(z_H) \varphi_n(z) \frac{1}{\lambda_n - \lambda_S} H(s).$$
(5)

Each eigenfunction  $\varphi_n(z)$  with the corresponding eigenvalue  $\lambda_n$  satisfies both the required Sturm–Liouville boundary conditions<sup>17</sup> and the following equation:

$$\frac{d^2\varphi_n(z)}{dz^2} + \lambda_n\varphi_n(z) = 0, \quad n = 1, 2, 3, \cdots.$$
 (6)

The solution P(z,t) can then be obtained by taking the inverse Laplace transform of Eq. (5).

The pressure response of an impulsive force  $F_I$  can be derived as

$$P_I(z,t) = A_I \sum_{n=1}^{\infty} \varphi_n(z_H) \varphi_n(z) \frac{e^{-bt}}{\omega_n^*} \sin(\omega_n^* t).$$
(7)

Here,  $\omega_n^* = (\omega_n^2 - b^2)^{1/2}$ ,  $A_I = I_0 / (\mu C_A)$ , and  $\omega_n = \lambda_n^{1/2} / C$ . Equation (7) shows that our model of the arterial system

Equation (7) shows that our model of the arterial system is characterized by a set of natural frequencies which are related to the eigenvalues  $\lambda_n$  and the wave velocity C. We may then describe the arterial system as a special damped drum with the elastic arterial wall as its drumhead. The exact values of these  $\omega_n$  depend on the physiological structure of the arterial system.

Similarly, in the steady state, the pulsatile part of the pressure response to an input harmonic force  $F_{\omega}$  of angular frequency  $\omega$  can be obtained as

$$P_{\omega}(z,t) = A_{\omega} \sum_{n=1}^{\infty} \varphi_n(z_H) \varphi_n(z) C_n \sin(\omega t + \phi_n), \qquad (8)$$

where  $A_{\omega} = F_{0\omega}/(\mu C_A)$ ,  $\phi_n = \tan^{-1}[2b\omega/(\omega^2 - \omega_n^2)]$ , and  $C_n = [(\omega^2 - \omega_n^2)^2 + 4b^2\omega^2]^{-1/2}$ .

As shown above, the amplitudes of different eigenmodes of the pressure response at position z are proportional to  $C_n$ . From the expression of  $C_n$ , we see that the eigenmode with natural frequency near the input frequency  $\omega$  is the mode that will be greatly enhanced so that the pressure amplitude of that eigenmode is maximized. We may call this the frequency matching condition.

Were the pressure waves in arterial system primarily propagating via the PQ wave mode, the high dissipation through the flow viscosity would have resulted in significant attenuation and prevented multireflections. In real arterial systems, since the energy associated with the axial flow is low,<sup>18</sup> the dissipation in flow viscosity is also small. With the PS wave mode as the major energy transport mechanism, an arterial system behaves as a forced radial vibration system with damping. In the PQ wave model, reflections occur at sites with mismatch in impedance (P/Q).<sup>1,2</sup> In this study, we point out that major reflections only happen at locations with discontinuities in the wave velocity.

We may assume that after a transient time, the ventricular-arterial system will reach a steady state and the whole arterial system will execute distributed stationary vibrations. Hence, the primary power consumption in delivering the blood from the left ventricle to the veins is only the dissipation in flow viscosity and in vibration resistance.

When the blood periodically bursts from the left ventricle at a frequency near the principal natural frequency of the arterial system, it is said to be in the first frequency matching condition. By this coupling, the efficiency of the principal harmonic component of the ventricular output is optimized since the amplitude of the pressure wave influences the amount of blood flowing into the arterioles.

With further physiological investigations of the reflection sites and the conditions at the ends, the values of the natural angular frequencies  $\omega_n$  of the arterial system can be computed.

In studying the matching between organs and the supplied force from the large arteries,<sup>19</sup> this frequency matching rule for the PS wave model may also be utilized and be compared with the result of the prevailing "impedance matching"<sup>20</sup> for the PQ wave model. The input force from the heart is comprised of many harmonic components so that it generates many different harmonic pressure responses. Equation (8) also shows that the contributions from the different eigenmodes to the pressure response have different position dependencies through the eigenfunctions  $\varphi_n(z)$ . This provides a method to explain the change of the pressure shape along the artery,<sup>1,2</sup> which also allows one to construct a transfer function<sup>21</sup> for the pressure waves at two different sites of the arterial system. All these will need further physiological and physical investigations.

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