The Natural Frequencies of the Arterial System and Their Relation to the Heart Rate

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***Abstract*—We assume the major function of the arterial system is transporting energy via its transverse vibration to facilitate the blood flowing all the way down to the microcirculation. A highly efficient system is related to maintaining a large pressure pulse along the artery for a given ventricular power. The arterial system is described as a composition of many infinitesimal Windkessels. The strong tethering in the longitudinal direction connects all the Windkessels together and makes them vibrate in coupled modes. It was assumed that at rest condition, the arterial system is in a steady distributed oscillatory state, which is the superposition of many harmonic modes of the transverse vibration in the arterial wall and the adherent blood. Every vibration mode has its own characteristic frequency, which depends on the geometry, the mass density, the elasticity, and the tethering of the arterial system. If the heart rate is near the fundamental natural frequency, the system is in a good resonance condition, we call this "frequency matching." In this condition, the pulsatile pressure wave is maximized. A pressure wave equation derived previously was used to predict this fundamental frequency. The theoretical result gave that heart rate is proportional to the average high-frequency phase velocity of the pressure wave and the inverse of the animal body length dimension. The area compliance related to the efficiency of the circulatory system is also mentioned.**

***Index Terms*—Circulatory system, efficiency, heart rate, pulsatile pressure.**

I. Introduction

The arterial system has mostly been described as a transmission line [1] or lumped as a Windkessel or some Windkessels [2]. Otto Frank [3] proposed an arterial resonance model. Hamilton and Dow introduced the concept of standing wave [4]. Their followers assumed the intensive multiple reflections at various sites of the body [5]. McDonald criticized the concept of resonance as being physically impossible by arguing that the reflected wave is being damped considerably during its travel between reflection sites [6]. Recently, we have discussed that most of the energy transport in the arterial system is via the transverse vibration of the arterial wall and the dissipation of energy in the artery is significantly reduced [7]. This transverse vibration mechanism makes the resonance model plausible.

Li and Noordergraaf pointed out the fundamental pressure wavelength is several times that of linear dimension of the systemic circulation for mammals [8]. It implies that wavelengths of all the harmonic waves are neither much smaller (as assumed in Windkessel model) nor much greater (as assumed in the transmission line theory) than the total length of the artery. Thus, the arterial system would be more like a wave-guide of finite length.

Manuscript received May 9, 2002; revised May 11, 2003. *Asterisk indicates corresponding author.*

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Digital Object Identifier 10.1109/TBME.2003.820378

O'Rourke and Taylor [9] stated that there is an appropriate balance between heart rate and body length. Milnor [10] gave an opinion that pulse wavelength and arterial length are matched in a way that minimizes cardiac work. The theory of Womersley with rigid tube approximation and impedance minima criteria was mostly used to explain the matching.

According to the O'Rourke's result, the pulsatile power is but a small fraction of external left ventricular power [11]. The criteria to minimize the cardiac work cannot be based on the minima impedance. In order to make correct criteria, we need to find out the basic functions of the arterial system.

II. Theory

By maximizing the pulsatile pressure amplitude along the whole artery, the radial movement of the wall and the adherent blood will be enhanced. Furthermore, as most of the side branch arteries connecting the main artery and the organs are perpendicular to the main artery, maximal pulsatile pressure will drive maximal pulsatile blood flow to the side branch. This ensures the arterial system to provide not only a pressure gradient force but also a blood flow source for the microcirculation. From this point of view, the efficiency of the cardiovascular system is related to the pulsatile pressure amplitude of the artery for a given ventricular power.

From basic physics, any system of finite dimension has its own nonzero and discrete natural frequencies f_n. When it is under the action of an external harmonic force with frequency f for a long time, the system will be in a steady forced oscillating state with the same frequency f. The response depends greatly on the difference of f and the system's nature frequencies. If f coincides with one of the natural frequency f_i, the ith mode will be excited with maximal amplitude.

The force in the systemic circulation is generated by the burst of blood from the heart with beat frequency f_b. It is composed with harmonic forces of frequencies $f_b, 2f_b, 3f_b, \ldots$. The circulatory system is of finite dimension and will also have its own natural frequencies. If all the mf_b have one to one coincidences with system's natural frequencies, $f_1, f_2, f_3, \ldots$, the heart will reach its highest efficiency. That is, the pressures of all modes retain their maximum amplitudes for a given force source from the heart. We call this condition "perfect frequency matching."

In this paper, we propose that "frequency matching" might be the hidden rule for some observed facts in the circulatory system such as regular heart rate, similar organ structures, as well as the inverse correlation between the heart rate and the animal dimension.

We assumed the arterial system is in a distributed transverse vibration governed by the pressure wave equation that has been derived in previous papers [12], [13]

$$\frac{\partial^2 P}{\partial t^2} + b\frac{\partial P}{\partial t} + \omega_0^2 P = V_\infty^2 \frac{\partial^2 P}{\partial z^2} + \frac{2\pi r_0}{L' C_A} F_{\text{ext}}. \tag{1}$$

It was assumed that the cross section of the artery at axial position z remains as circle during oscillation with variable radius r and area A. The wall is of thickness h_w and density ρ_w while the fluid is of density ρ and a layer of thickness h is adherent to the wall and oscillating transversely with the wall. For the transverse motion, we also assumed there is a damping force proportional to the radial velocity with damping constant η. P is defined as the difference of the internal blood pressure $P_i(z,t)$ and the static blood pressure, $P_0(z)$, $r_0(z)$ is the static radius and ω_0 is the residual angular frequency of the local Windkessel vessel which has area compliance C_A.

In view of the strong tethering of vessels *in vivo*, Bergel [14] assumed that the blood vessel length was kept constant by longitudinal force. Therefore, we may also describe the arterial vessel as a cylindrical bundle of elastic strings with large longitudinal tension. The circumferential stress holds the bundle of strings together. When the arterial system is subjected an external force per unit axial length $F_{\rm ext}$, the vessel and the adherent fluid with mass per unit axial length $L'(= 2\pi r_0(\rho h + \rho_w h_w))$ will conduct a transverse movement with damping constant $b(= 2\pi r_0 \eta / L')$. The restoring shearing stress and the radial component of the longitudinal tension arising from the local slope of the string, make the pressure wave traveling from one Windkessel to the adjacent Windkessel. Therefore, our high-frequency phase velocity V_∞ is related to the shearing modulus E_{rz}, cross-sectional area $S_w(= 2\pi r_0 h_w)$ of the wall as well as the longitudinal tension τ_L along the tube as

$$V_\infty = \sqrt{\frac{\tau_L + \mathrm{S_w} E_{rz}}{L'}}. \tag{2}$$

Most studies in hemodynamics were started from the equation of motion of the blood in the axial direction such as works done by Moens, Korteweg, and Womerseley [15]. Their concept is based on that the axial pressure gradient induces axial flow change, and by equation of fluid continuity, flow variation induces pressure change via the compliance of the wall. Thus, a pressure wave or flow wave of phase velocity related to the circumferential elastic modulus was obtained [16], [17]. Nevertheless, inside the blood vessel, the viscous force on the blood moving in the axial direction cannot be ignored. By their model, the pressure wave might not last without great attenuation even along the main artery. Those flow models led the misconception that multirereflection is impossible and McDonald [6] used that argument to criticize the resonance model [3] in arterial system.

Equation (1) describes the transverse vibration mechanism of the arterial system. Most of the energy is transported via this process. The 180 degrees bending at the arch of the aorta converts great amount of kinetic energy of the blood flow into other forms of energy. The low axial flow will reduce energy dissipation in viscosity and, therefore, allow the existence of the resonance model or standing wave for arterial system.

This wave equation also enables us to study the distributed pressure wave of any animal with any structure in a systematic way. The pressure becomes the central role in our model. So long as the vessels retain the amplitude in pressure, pulsatile blood will be squeezed out whenever there are openings.

If we apply the wave equation to a uniform tube of length l with the ending pressures maintain their static values, we may derive that the fundamental resonance frequency f_1 is about $V_\infty/2l$. It is quite similar to the fundamental frequency of a string of length l and phase velocity V.

For a real arterial system, by fulfilling suitable boundary conditions such as the continuity of the pressure at the reflection sites (places with discontinuity in phase velocity V_∞) and the equation of continuity of the fluid at bifurcations (such as the end of the aortic trunk), its allowed frequencies or its natural frequencies still could be analyzed. To simplify the problem, we may view the artery as to be composed with nonuniform tubes of various phase velocities, and the blood pressures maintain their respective static values at the ends. Thus, the fundamental natural frequency still could be approximated as $V_\infty/2l$, if we substitute V_∞ by the average value of the wave velocities on the whole system and l as the longest distance between the ends.

For a creature to meet the "frequency matching" condition, its heart rate f_b will be about $V_\infty/2l$. For human beings, the average high-frequency phase velocity V_∞ is near 500 cm/s [18]. Since the pressure pulse should be existing through the whole body, we may assume that until at the ends of the arterial system, the blood pressures maintain their steady values, thus, $l = 180$ cm from head to toe, the estimated heart rate is about 1.4 Hz. Many animals have the same pressure phase velocity as man [6], therefore, the "frequency matching" could be used to explain the well-known inverse correlation between heart rate and body size [19] in a quantitative way.

III. Conclusion

In this paper, we describe the arterial system as being composed of many infinitesimal Windkessels. The strong tethering in the longitudinal direction connects all the Windkessels together and makes them vibrate transversely in coupled modes. We propose a "frequency matching" rule and make some conclusions as follows.

1) The main arterial system delivers energy to the whole circulatory system via the pulsatile pressure. The efficiency of the systemic system is related to the pressure amplitude response for a given ventricular power.
2) The arterial system is in a distributed oscillatory state; the pressure wave, the flow wave and the impedance are varied with positions.
3) At rest posture, all the arterial vessels are conducting a steady transverse vibration under a periodic force generated by the heart. The whole systemic circulatory system coupled together, their vibration modes are the over all effects of multireflections from all the boundaries [4].
4) In normal conditions, the heart rate is regular. The Fourier analysis of human pressure pulse shows the importance of the various harmonic modes [6]. Any model, no matter how simplified, should have a Fourier analysis as a first step. From the steady periodic pressure pulse at one position of the artery, the times of the systolic pressure and the diastolic pressure cannot give us the information about the location of reflection sites. It mainly gives us the pressure amplitude distribution of different harmonic modes at that position.
5) The arterial system has its natural frequencies that depend on its geometry, physical properties of the vessels and the blood, as well as the boundary conditions at junctions.
6) If the heart beats at a frequency near the fundamental natural frequency of the arterial system, it will have an optimal output for the pressure pulse and, therefore, highest flow transport. We call this condition "frequency matching." This is inconsistent with the conclusion made by Noordergraaf *et al.* who stated that heart rates at rest are at the lowest rate commensurate with minimal external work performed by the left ventricle [20].
7) The ultimate purpose of the circulatory system is to facilitate the whole arterial system maintaining a steady transverse vibration, that is, to keep a large area oscillation for all the blood vessels. The amplitude of the local area wave is proportional not only to the local pressure wave but also to the local area compliance. Therefore, a compliant arterial wall is more efficient than a stiff one under the same pressure pulse.

For healthy creatures, the perfect matching condition is fulfilled, therefore, the heart rate and the blood pressure are stable. Aging or diseases may lead a mismatch that will increase the load of the heart and affect its efficiency. We expect our matching theory would improve the design of circulatory-enhancing systems.

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Quantifying Ventricular Fibrillation: *In Silico* Research and Clinical Implications

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***Abstract*—Cardiovascular disease remains the leading cause of death in otherwise healthy humans. In particular, most cases of *sudden* cardiac death occur as a result of failure of the mechanical function of the heart which is triggered by a turbulent pattern of electrical excitation of the heart e.g., ventricular fibrillation (VF). Although the exact mechanisms of VF remain unknown, increasing evidence indicates that it is organized by multiple reentrant sources (wavelets).**

***Index Terms*—Cardiac tissue, heterogeneity, mathematical model, reentrant source, ventricular fibrillation, wavelets.**

I. Wavelet Mechanism

The wavelet mechanism of fibrillation was initially proposed by Moe *et al.* [1] as a mechanism of atrial fibrillation and was later extended to explain ventricular fibrillation (VF) as well. The original hypothesis explained the onset of the wavelets by the presence of heterogeneity of cardiac tissue with respect to the refractory period. Nowadays, the two most actively discussed mechanisms of wavelet onset are the "fibrillatory conduction" mechanism and the "restitution hypothesis." The former mechanism explains the maintenance of VF by the presence of a single source (or a small number of them), generating waves at high frequency [2], [3]. Such waves propagating through the ventricle interact with heterogeneities of the myocardium and induce a turbulent wave pattern. Alternatively, the "restitution hypothesis" considers the wavelets as active sources of excitation which occur from a single source as a result of additional wave breaks on functional heterogeneities of cardiac tissue. Such functional heterogeneities do not preexist in cardiac tissue but develop as a result of a dynamical instability. Such an instability can occur if the restitution curve of cardiac tissue (which relates the duration of cardiac pulses to the recovery time between the pulses) has a slope of more than one (for a review see [4]).

II. Therapeutic Issues Relevant for Bioengineering

What are the therapeutic consequences of both the "fibrillatory conduction" mechanism and the "restitution hypothesis"? If VF is driven by a small number of high-frequency sources (as in the case of the "*fibrillatory conduction*" mechanism) then an ablation or other specific treatments of the reentrant site can terminate the fatal cascade. Can a similar approach be applied in case of the "*restitution hypothesis*"? The answer to this question can be just in a simple arithmetic: in counting the number of active sources of excitation. The contribution by Clayton and Holden [5] published in this issue of the *Transactions* deals with this important question. The paper presents the results of numerical modeling of the process of spiral breakup in an anatomically accurate model of the canine ventricle. The anatomical data employed are developed by the Auckland group [6] and include a detailed description of fiber orientation, which allows description of the anisotropy of

Manuscript received August 4, 2003; revised September 26, 2003. *Asterisk indicates corresponding author.*

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Digital Object Identifier 10.1109/TBME.2003.820608