Effect of Length on the Fundamental Resonance Frequency of Arterial Models having Radial Dilatation

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***Abstract*—The pressure wave moving along an elastic artery filled with blood was examined as a moving Windkessel having a natural oscillation angular frequency ν_0 and a damping coefficient b. The radial directional motion for an element of the wall segment and the adherent fluid was considered. This equation was solved with conditions at both ends of an artery of length L. An external impulse force was applied at one end and a static pressure P_0 at the other. Analytic solution allowed only certain oscillation modes of resonance frequencies f_n, where $f_n^2 = a + c_n L^{-2}$ with**

$$a = \frac{\nu_0^2}{4\pi^2} - \frac{b^2}{16\pi^2},$$
$$c_n = \left(n + \frac{1}{2}\right)^2 \frac{V_\infty^2}{4}, \qquad n = 0, 1, 2, 3, \cdots$$

and V_∞ is the high frequency phase velocity.

The relationship between f_0 and L was examined experimentally for tubes constructed of latex, rubber, or dissected aorta. The effect of raising the static pressure P_0 or increasing the tension in the tube was consistent with the prediction. The hypertension that accompanies an augmentation in arterial wall and the association between the heart rate and the mean blood pressure were discussed.

***Index Terms*—Hypertension, moving Windkessel, transmission line model.**

I. Introduction

ARTERIAL-WALL elasticity influences pressure wave propagation; however, in various early studies, such as the Moens–Korteweg equation, the distensibility of the arterial wall was assumed to be small and the change in the tube cross section negligible.

Most models dealing with the equations of motion of fluid streaming through a tube concentrated on the longitudinal motion. These models linearized and simplified the Navier–Stokes equation. The wall was assumed static and the movement was then treated as a perturbation. As a result, no significant radial dilation was considered [1]–[4]. The kinetic energy of the flow was examined while the elastic energy stored in the wall was ignored.

Another approach, the transmission line model (which requires two lines) [4], [5], was used to simulate several Windkessels arranged in series. This model yields an electrical analog that can be applied when dealing with the relationship between pressure and flow. The "capacitance per length" represents the blood (or charge), as well as any energy other than the kinetic energy stored in the system. Milnor [6], in his derivation of a true propagation coefficient, used the transmission line model. Although this correction provides good results for high-frequency waves, for low-frequency waves, the experimental result for impedance is still much higher than the theoretical prediction. This analog is limited by defining the "length" in "per length" and the number of elements required for each artery. Moreover, it does not define how the different elements of "per length" are coupled with each other [4], [5]. We, therefore, propose a new coupling mechanism, resonance, to enlarge the application of the model [7]–[9]. This coupling mechanism explains partially the discrepancy between the experimental and theoretical results in Milnor's work [7]–[9]. For an *in situ* artery, over 90% of the energy is stored in the arterial wall and less than 10% is stored in the blood flow [3]. All of the models that start from the equation of motion of the flow in the longitudinal direction take into account only a small part of the energy. Therefore, any approximation made in the model will cause enormous deviation in the prediction of the entire system.

Our proposed model starts from the equation of motion in the radial direction for a cylindrical elastic tube with adherent fluid [10]. The wall squeezes the blood, and via the equation of continuity of the fluid, the pressure wave drives a blood flow wave.

The proposed pressure wave P is governed by the following equation:

$$\frac{\partial^2 P}{\partial t^2} + b\frac{\partial P}{\partial t} + \nu_0^2 P = V_\infty^2 \frac{\partial^2 P}{\partial z^2}. \tag{1}$$

The attenuation term b is related to the kinetic viscosity of the wall and the adherent fluid in the radial direction. The characteristic angular frequency ν_0 is related to the Young's modulus, arterial compliance, mass of the wall, the adherent fluid, as well as the radius of the tube. V_∞ is referred to as the high-frequency phase velocity related to the shear modulus of the wall.

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If the two analogous telegraph equations [4], [5] in the transmission line model are combined, a similar equation can be obtained

$$\frac{\partial^2 P}{\partial t^2} + \frac{LG + RC}{LC}\frac{\partial P}{\partial t} + \frac{RG}{LC}P = \frac{1}{LC}\frac{\partial^2 P}{\partial z^2}. \quad (2)$$

This equation also describes an attenuated propagating pressure wave with $\sqrt{1/LC}$ as its high-frequency phase velocity, where L is the inertia per length of the fluid and C is the ability to store blood per length of the artery. This occurs only when there are two lines. The oscillating system is the blood fluid between the two lines; the attenuation term is related to the leakage G between the two lines, and the resistance R in the axial direction. The characteristic frequency $\sqrt{RG/LC}$ becomes zero, when the leakage G or the longitudinal blood resistance R is small.

In the proposed model [10], the blood pressure wave is similar to a transverse string wave, while in the transmission line model, the blood pressure wave is like a longitudinal wave, which can only occur in a two-line electrical analog.

For an elastic tube of finite length L, there exist only certain resonance frequencies f_n. A generalized solution $f_n^2 = a + c_n L^{-2}$ is obtained which will fit tubes with different elasticities, densities, thicknesses, and lengths. *In vitro* experiments to test the relationship between f_0 and L of different tubes were performed. From this result, we also proposed a mechanism how the circulatory system adapts to match the resonance frequency of the aorta with the heart rate.

II. Theory

When a pressure wave moves along an elastic artery filled with blood, each segment of the artery alternately expands and shrinks in sequence in the radial direction. Viewed from the axial direction, the expanding segment is moving along the artery, squeezing the blood as it moves. This is similar to a moving Windkessel, a chamber having an elastic wall that moves along the artery.

If the tube is very long, we may assume $P(z, t) = P_0 e^{i(kz - \varpi t)}$ [10]. Then, at a fixed position located a distance of z_0 from any external force, (1) becomes

$$\frac{d^2 P}{dt^2} + b\frac{dP}{dt} + \nu_0^2 P = -k^2 P \quad (3)$$

where $-k^2 P$ is the force passing along the elastic tube to the point z_0.

This equation represents a forced harmonic oscillation equation for which the driving force originates at the heart.

If the restoring force and the damping force are ignored, that is, if the radius of the artery remains unchanged, then (1) becomes

$$\frac{\partial^2 P}{\partial t^2} = V_\infty^2 \frac{\partial^2 P}{\partial z^2}. \quad (4)$$

This is the general wave propagation equation for a wave having phase velocity V_∞ (i.e., the Moens–Korteweg equation).

Thus, (1) can be considered as simply the combination of a damped harmonic oscillation equation and a wave propagation equation that drives a radial simple harmonic oscillation along the artery.

The wave propagation equation can be derived [10] from the radial equation of motion of an element containing a small portion of the wall segment and the adherent fluid

$$\frac{dP_r}{dt} = F_P + F_N + F_S + F_f + F_{ext} \quad (5)$$

where

- P_r the momentum of the element in the radial direction;
- F_P the normal force acting on the wall by the fluid pressure;
- F_N restoring force due to the elasticity of the wall;
- F_S shear force;
- F_f frictional force in the radial direction;
- F_{ext} the external force.;

The cross section of the artery is assumed to remain circular with radius r, which is a function of the axial distance z and time t. The momentum P_r is proportional to the radial velocity of the wall, that is, dr/dt. We further assume that the wall material obeys Hook's law of elasticity. Then by the chain rule, the variable $r(z, t)$ is changed to the pressure variance $P(z, t)$ via the arterial compliance $C_A = (d(\pi r^2))/dP$. The pressure variance $P(z, t)$ is defined as the difference between the internal fluid pressure $P_i(z, t)$ and the static pressure $P_0(z)$. That is

$$P(z, t) = P_i(z, t) - P_0(z). \quad (6)$$

Thus, (5) becomes

$$\mu\frac{\partial^2 P}{\partial t^2} + R\frac{\partial P}{\partial t} + kP = \tau\frac{\partial^2 P}{\partial z^2} + \frac{F_{ext}}{C_A} \quad (7)$$

where

$$\mu = \rho_w h + \rho_f h_f, \quad k \cong \frac{E_n}{r_0} - \frac{2\pi r_0}{C_A}, \quad \tau = E_{rz} h + T$$

in which

- ρ_w density of the arterial wall;
- h thickness of the wall;
- ρ_f density of the fluid that adheres to the wall;
- h_f thickness of the fluid that adheres to the wall and moves radially together with the wall;
- R constant of viscosity for movement of the wall and the adherent fluid in the radial direction;
- $E_n = E_r + E_\theta$ the sum of the Young's modulus in the radial and that in the circumferential direction;
- E_{rz} the sum of the Young's modulus in the shear modulus of the wall;
- T tension along the wall per-unit circumferential length;
- r_0 static radius of the tube.

Thus, the resistance coefficient b, the characteristic angular frequency ν_0, and the high-frequency phase velocity V_∞ in (1) are related to the physical property of the artery as

$$b = \frac{R}{\mu} \quad (8)$$

$$\nu_0 = \sqrt{\frac{k}{\mu}} = \sqrt{\frac{1}{\mu}\left(\frac{E_n}{r_0} - \frac{2\pi r_0}{C_A}\right)} \quad (9)$$

$$V_\infty = \sqrt{\frac{\tau}{\mu}} = \sqrt{\frac{E_{rz}h + T}{\mu}}. \tag{10}$$

In order to simulate the heart beat, an impulse external force, $\mathrm{Im(t)}$ with amplitude I, is applied at one end of the artery. At the other end of the artery $z = L$, the pressure P_i is maintained at the static pressure P_0. Thus, based on (6), the end condition for the pressure variance $P(z, t)$ in (7) is

$$P(z = L, t) = 0. \tag{11}$$

For a linear solution to fit this boundary condition, we let

$$P(z, t) = \sum_{n=0} \Phi_n(t) \cos \frac{\left(n + \frac{1}{2}\right)\pi z}{L}. \tag{12}$$

Substituting (12) into (7), we have

$$\mu \frac{d^2\Phi_n}{dt^2} + R\frac{d\Phi_n}{dt} + \left(k + \tau \frac{\left(n + \frac{1}{2}\right)\pi^2}{L^2}\right)\Phi_n = \mathrm{Im}(t)$$
$$n = 1, 2, 3, 4, \cdots$$

and the solution to this equation becomes

$$\Phi_n(t) = \frac{I}{\varpi_n} e^{-\beta t} \sin(\varpi_n t). \tag{13}$$

Substituting (13) into (12), we have

$$P(z, t) = \sum_{n=0} \frac{I}{\varpi_n} e^{-\beta t} \sin(\varpi_n t) \cos \frac{\left(n + \frac{1}{2}\right)\pi z}{L} \tag{14}$$

where

$$\varpi_n = \sqrt{\frac{\mu\left[k + \tau \frac{\left(n + \frac{1}{2}\right)^2 \pi^2}{L^2}\right] - \frac{1}{4}R^2}{\mu^2}}, \; n = 1, 2, 3, 4, \cdots \tag{15}$$

$$f_n^2 = \frac{\varpi_n^2}{4\pi^2}$$
$$= \frac{\mu k - \frac{R^2}{4}}{4\pi^2\mu^2} + \frac{\left(n + \frac{1}{2}\right)^2 \tau}{4\mu} L^{-2}$$
$$= a + c_n L^{-2} \tag{16}$$

in which

$$a = \frac{\mu k - \frac{R^2}{4}}{4\pi^2\mu^2} = \frac{\nu_0^2}{4\pi^2} - \frac{b^2}{16\pi^2} \tag{17}$$

$$c_n = \frac{\left(n + \frac{1}{2}\right)^2 \tau}{4\mu} = \frac{\left(n + \frac{1}{2}\right)^2}{4} V_\infty^2. \tag{18}$$

f_n $(n = 0, 1, 2, 3, 4, \cdots)$ are the allowed frequencies for the oscillation modes existing on the artery of length L. These frequencies are referred to here as the resonance frequencies of the tube. The amplitude of the mode decreases with the mode's number. For the fundamental mode, $n = 0$, the allowed frequency f_0 is given by

$$f_0^2 = a + c_0 L^{-2} \tag{19}$$

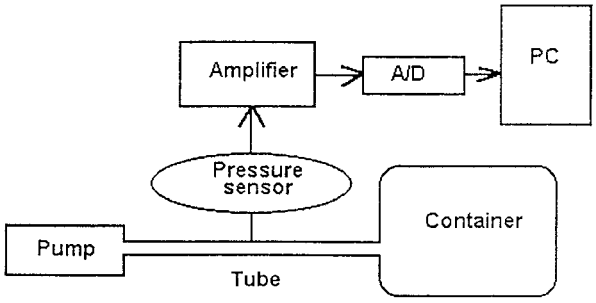

Fig. 1. Experimental setup for the measurement of the impulse response of the latex and the rubber tubes.

in which

$$c_0 = \frac{\tau}{16\mu} = \frac{1}{16} V_\infty^2. \tag{20}$$

III. Methods and Materials

The proposed theory was verified experimentally. A diagram of the system [7]–[10] is shown in Fig. 1. The tube was connected at one end to a stepping motor controlled pump, which generated an impulse. The other end of the tube was placed in a container filled with water, the water level was 25-cm H_2O above the level of the tube (with the exception of the tube to which an additional static pressure was applied to yield a total pressure of 35-cm H_2O). The response of the tube was measured at a position between 1/4 and 3/4 of the tube length using a DP103 differential pressure transducer (Validyne, Northridge, CA). The response in the time domain was digitized by ADC with a sampling rate of 460/s. Then, Fourier transform was used to obtain a response in the frequency domain. The fundamental resonance frequency f_0 was determined by the first peak of the amplitude in this domain.

The hog aorta was dissected from the animal immediately after it was sacrificed. The outside fat and the connective tissue were cleaned, and only the cylindrical shaped tube was kept. Although the intima and probably a large portion of the media were retained, most of the adventitia was removed. In order to isolate a cylindrical tube, the cutting points on the branches were very close to the aorta tube and the holes generated by the cut branches were sealed by heat. The entire aorta was immersed in Ringer's solution at room temperature and gas (CO_2 5% O_2 95%) was bubbled constantly to the solution to maintain the pH $= 7.4$ and the oxygen content [11], [12] immediately after the aorta was dissected. A diagram of the system for the hog aorta experiments is shown in Fig. 2. All hog aorta experiments were performed within 24 h after the death of the animals. An ultrasound flowmeter was also used in this setup to monitor the flow wave in the hog aorta. If the aorta is healthy, both the pressure wave and the flow wave will be steady, the flow wave is more sensitive to the deterioration of the aorta. The flow wave was, therefore, used as a control for determining the health of the specimen.

Tubes made of latex, rubber and hog aorta (composed of thoracic aorta and abdominal aorta) were tested. The dimensions of these tubes are listed in Table I.

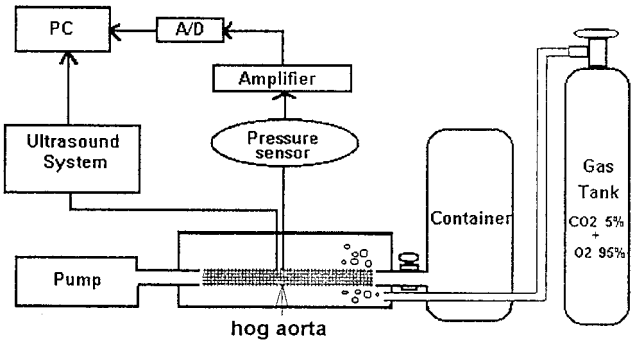

Fig. 2. Experimental setup for the measurement of the impulse response of the dissected hog aorta.

TABLE I

Tube material	D_0	h	Density
Latex	1.11	0.16	0.90
Rubber	0.7	0.02	0.50
Hog aorta (measured after the experiment)	1.65	0.17	1.04

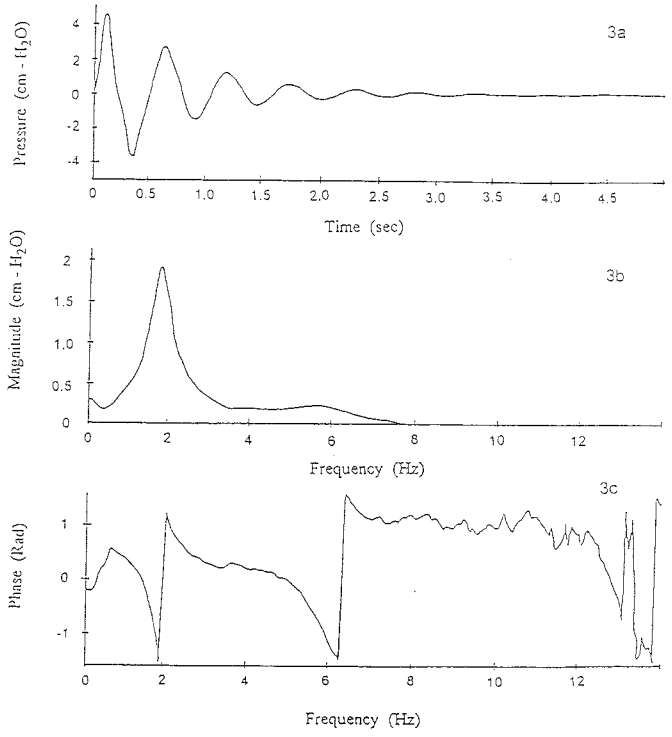

Fig. 3. (a) Impulse response in the time domain, (b) impulse response (amplitude) in the frequency domain, and (c) impulse response (phase) in the frequency domain.

IV. Results

The pressure impulse responses for latex tubes, rubber tubes, or dissected hog aortas were similar. Typical data is shown in Fig. 3. The impulse response in time domain for a hog aorta of length $L = 43$ cm measured at $3L/4$ from the pump is shown in Fig. 3(a). The first peaks of the amplitude and the phase in the frequency domain observed in Fig. 3(b) and (c), respectively, gave the fundamental frequency $f_0 = 1.8$ Hz; the second peak gave the second resonance frequency of approximately 6 Hz. The position of measurement did not affect the resonance frequencies of the various modes, it affected only the relative amplitude between different modes. Each experiment was repeated four times. For all of the experiments, the CV (coefficient of variance) = standard deviation/mean $\leq$1%. (The standard derivations are too small to be shown in the figure.)

The fundamental resonance frequencies f_0 for various lengths L of different tube materials and conditions are summarized in Fig. 4. The solid theoretical line is determined by (19) using the least squares method to determine a and c_0. All five data fit the theoretical predictions. From Fig. 3, we estimated that $b \cong 2\pi \times 0.4$ Hz at 25-cm H_2O. $V_\infty \cong 0.2 \times 10^3$ cm/s is the measured phase velocity obtained by passing a

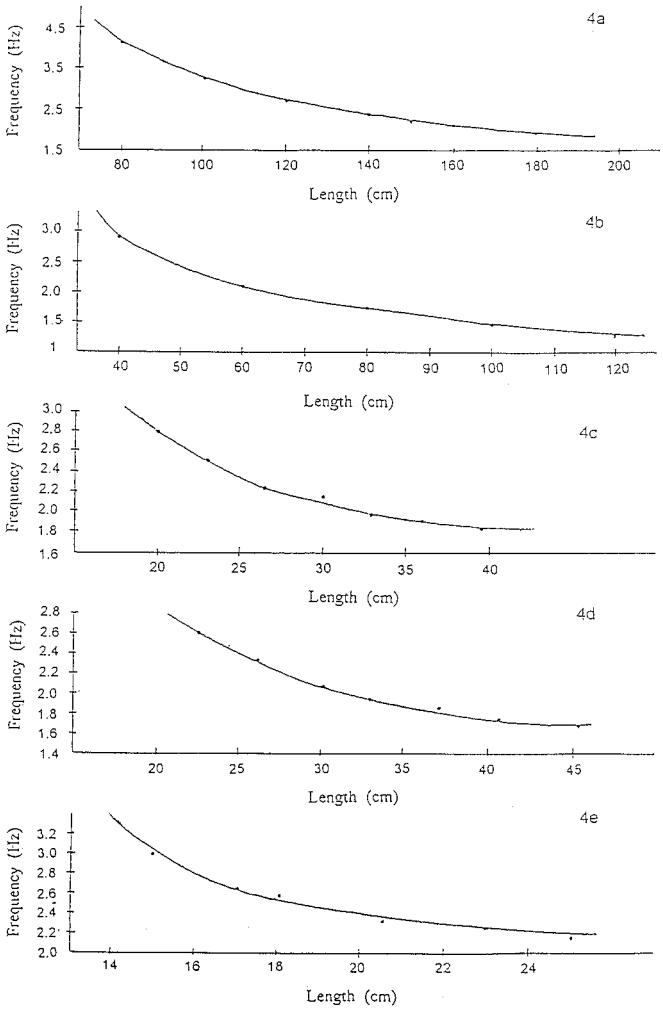

Fig. 4. Plot of f_0 versus length of tube ($\cdot$) for various tubes. The solid theoretical line is determined by least squares fitting. (a) Latex tube. The solid line is $f_0 = \sqrt{0.27 + 10.7 \times 10^4 L^{-2}}$. (b) Rubber tube. The solid line is $f_0 = \sqrt{0.77 + 1.30 \times 10^4 L^{-2}}$. (c) Dissected hog aorta. The solid line is $f_0 = \sqrt{1.85 + 0.241 \times 10^4 L^{-2}}$. (d) Same dissected hog aorta as (c) with additional tension (stretched to *in situ* length). The solid line is $f_0 = \sqrt{1.16 + 0.282 \times 10^4 L^{-2}}$. (e) Similar dissected hog aorta as (c) with larger static pressure (35-cm H_2O rather than 25-cm H_2O). The solid line is $f_0 = \sqrt{2.83 + 0.119 \times 10^4 L^{-2}}$.

high-frequency pressure wave [approximately 4 Hz, which is well above the $f_0 = 1.8$ Hz].

From Fig. 4, $a \cong 1.85$ is for a hog aorta at a static pressure of 25-cm H_2O and $a \cong 2.83$ is for a hog aorta at a static pressure of 35-cm H_2O. Substituting into (17), we have $\nu_0^2 \cong 76$ 1/s^2. The static radius r_0 is 0.9 cm at a static pressure of 25-cm H_2O, and r_0 is 1.0 cm at a static pressure of 35-cm H_2O, thus, the arterial compliance C_A is

$$\frac{\Delta A}{\Delta P} = \frac{\pi(1\text{ cm})^2 - \pi(0.9\text{ cm})^2}{(35-25)\text{cm} - \text{H}_2\text{O}} = \frac{0.6\text{ cm}^2}{10 \times 980\text{ dyne/cm}^2} = 6 \times 10^{-5}\text{ cm}^4/\text{dyne}.$$

Assuming $\mu \cong \rho r_0$, it implies that all of the water inside the tube is squeezed forwardly by the wall movement whereas no water adheres at the outside of the wall and by (9), we have $E_n \cong 10^5$ dyne/cm^2 at a static pressure of approximately 30-cm H_2O.

E_n is similar to E_{inc} (incremental elastic modulus) and increases rapidly as the rise in static pressure [2]. Due to the difficulty involved in keeping the prepared aorta from leaking through the dozens of cut branches, elevating the static pressure further was not possible. Therefore, E_n is estimated to be approximately 10^6 dyne/cm^2 for a striped aorta at a static pressure of 105-cm H_2O.

From $C_0 = V_\infty^2/16$, we have $V_\infty \cong 2 \times 10^2$ cm/s for aorta and $V_\infty \cong 10^3$ cm/s for latex.

V. Discussion

The aortas used in the present study were approximately cylindrical. All outside attachments were stripped, and all of the branches were cut very near to the branch point. The results shown in Fig. 4(c)–(e) indicate some scattering from the theoretical results. The aorta is not a good cylindrical tube as compared to latex or rubber tubes.

The experimental results confirm that the fundamental frequency f_0 of an arterial model of length L is related as $f_0^2 = a + c_0 L^{-2}$. If the length L is long enough such that $a \gg c_0 L^{-2}$, then f_0 is independent of the length. However, if $c_0 L^{-2} \gg a$, then f_0 is inversely proportional to the length, which is similar to the standing wave result in which the resonance wave length is determined mainly by the length of the tube. In the present experiment, both terms are contributed.

At first glance, these f_n are not the harmonics of the heartbeat, and appear not to be consistent with the physiology. Actually, in the hog aorta, the wavelength of the resonance frequency λ_{f_0} is about 10 m, which is very long compared to the body length. Therefore, all other arteries that extend from the aorta (illiac, femoral) are integrated and behave as an extension of the aorta. In addition those organs (kidney, spleen) that are coupled with the aorta [7]–[9] will be integrated. The individual resonance frequencies for the organs such as the kidney are concealed when these organs are in the body, all the arteries and organs are integrated into one unit and all of the resonance frequencies in the entire circulatory system should then be the harmonics of the heartbeat. This may be the reason why the spectrum of the pressure pulse is harmonics of the heartbeat no matter where the measurement is performed.

For a 50-cm-long aorta, the curves or equations in Fig. 4(c)–(e) are used: $f_0 = 1.68$ Hz for the dissected aorta, $f_0 = 1.51$ Hz for the aorta with tension, and $f_0 = 1.82$ Hz for the aorta at elevated static pressure P_0. All resonance frequencies were in agreement with (17). The rise in static pressure causes an increase in the Young's modulus E_n, which in turn increases a, and subsequently f_0. This result is consistent with physiology: when only the heart beat is temporarily increased, the blood pressure is usually elevated, so that the heart and the aorta may match each other in order to maintain the resonance. Equation (17) may also explain the results of Fung *et al.* [13], [14]. They found that when the mean pressure in an artery was raised by constriction, the arterial wall was quickly remodeled. By (17), the increase in wall thickness causes μ to become larger and decreases a and c, thus lowering f_0. The arterial wall appears to thicken in order to counterpoise the effect of the pressure rise, so that f_0 of the artery will remain matching the heart rate. This may be a general phenomenon for all hypertension cases.

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